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# Diagnostic, prognostic, and immunological roles of NCAPG in pan-cancer

## A bioinformatics analysis

Min Fang, Msc[a,b], Zhu Wu, Msc[c], Zhi Xia, MD[d], Jian Xiao, MD[e,f,*]

**Abstract**

Growing studies have shown that non-SMC condensin I complex subunit G (NCAPG) was highly expressed in a variety of tumors and was involved in the progression of multitumors, but the role of NCAPG in tumorigenesis is not fully understood. Our study purposed to systematically investigate the role of NCAPG across cancer types. Interacting molecules with NCAPG were analyzed using searching bioinformatics websites including Search Tool for the Retrieval of Interacting Genes/Proteins, GeneMANIA, and Global Positioning System-Prot. NCAPG-related diseases were acquired using the Open Targets Platform. The interaction of NCAPG and 14 cancer functional states was achieved using the CancerSEA website. The databases including the University of California Santa Cruz Xena, Genotype-Tissue Expression, The Cancer Genome Atlas Program, Human Protein Atlas, and XIANTAO Academic were used to interpret the expression of NCAPG. Correlations between NCAPG expression and immune infiltration and immune-related molecules were analyzed by using Tumor Immune Estimation Resource Version 2 and Tumor and Immune System Interaction Database databases. NCAPG expression was significantly upregulated in most cancer types. NCAPG was identified as a marker of diagnostic value and prognostic significance in most cancer types. NCAPG expression was related to immune cell infiltration and immune-related molecules across various cancers, especially kidney renal clear cell carcinoma and thyroid carcinoma. Furthermore, NCAPG expression could affect the enrichment and decrease immune cell infiltration to influence prognosis in kidney renal clear cell carcinoma but was devoid of evidence in thyroid carcinoma. NCAPG was a prospective marker for the diagnosis and prognosis of pan-cancer. Our results suggested that NCAPG was a potential cancer biomarker for the diagnosis and prognosis of pan-cancer. NCAPG might affect the immune microenvironment, which could be applied in the development of new-targeted drugs for immunotherapy.

**Abbreviations:** ACC = adrenocortical carcinoma, Act_CD4 = activated CD4 T cells, AUC = area under the curve, BC = breast cancer, BLCA = bladder urothelial carcinoma, BRCA = breast invasive carcinoma, CESC = cervical squamous cell carcinoma and endocervical adenocarcinoma, CHOL = cholangiocarcinoma, COAD = colon adenocarcinoma, CRC = colorectal cancer, DLBC = lymphoid neoplasm diffuse large B-cell lymphoma, DSS = disease-specific survival, ESCA = esophageal carcinoma, GBM = glioblastoma multiforme, GC = gastric cancer, GPS = Global Positioning System, GTEx = Genotype-Tissue Expression, HNSC = head and neck squamous cell carcinoma, HPA = Human Protein Atlas, IHC = immunohistochemistry, KICH = kidney chromophobe, KIRC = kidney renal clear cell carcinoma, KIRP = kidney renal papillary cell carcinoma, LAML = acute myeloid leukemia, LGG = brain lower grade glioma, LIHC = liver hepatocellular carcinoma, LUAD = lung adenocarcinoma, LUSC = lung squamous cell carcinoma, MEL = melanoma, MESO = mesothelioma, MHC = major histocompatibility complex, NCAPG = non-SMC condensin I complex subunit G, NK = natural killer, OS = overall survival, OV = ovarian serous cystadenocarcinoma, PAAD = pancreatic adenocarcinoma, PC = prostate cancer, PCPG = pheochromocytoma and paraganglioma, PFI = progression-free interval, PRAD = prostate adenocarcinoma, READ = rectum adenocarcinoma esophageal carcinoma, SARC = sarcoma, SKCM = skin cutaneous melanoma, STAD = stomach adenocarcinoma, STRING = Search Tool for the Retrieval of Interacting Genes/Proteins, TCGA = The Cancer Genome Atlas Program, TGCT = testicular germ cell tumor, Th2 = helper T-cell 2, THCA = thyroid carcinoma, THYM = thymoma, TIMER2 = Tumor Immune Estimation Resource Version 2, TISIDB = Tumor and Immune System Interaction Database, UCEC = uterine corpus endometrial carcinoma, UCS = uterine carcinosarcoma, UCSC = University of California Santa Cruz, UVM = uveal melanoma.

**Keywords:** diagnostic value, immune, NCAPG, pan-cancer, prognosis

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## 1. Introduction

A malignant tumor is a major public health problem currently that seriously endangers human life and health, depriving countless patients of their lives every year.[1,2] The global cancer-related data released in 2024 showed that there will be 20 million new cancer cases and 9.7 million cancer deaths worldwide in 2020.[3] The pathogenesis of cancers is extremely complex. Massive studies have reported that the abnormal expression of genes played key roles in the occurrence and development of tumors.[4,5] Therefore, identifying key tumor-related genes is important for understanding the onset, maintenance, and progression of cancers and for seeking precisely targeted therapies.[6] In the field of tumor research, data in various large public databases have been shared, so pan-cancer analysis can be conducted by exploring some important tumor markers and other genes with prognostic value, which helps to further screen potential target genes for clinical application to a certain extent.

Non-SMC condensin I complex subunit G (NCAPG), which is located on human chromosome 4p15.32 and could encode a subunit of the condensing complex, regulates chromosome cohesion and stability during mitosis and meiosis.[7] At present, some evidence has demonstrated NCAPG was implicated in various cancers including gastric cancer (GC), breast cancer (BC), colorectal cancer (CRC), lung adenocarcinoma (LUAD), and so on.[8–11] For instance, abnormally higher NCAPG expression was observed in CRC cells and its knockdown resulted in inhibiting proliferation and impeding the cell cycle but inducing apoptosis in CRC cells.[9] Similarly, NCAPG expression was greatly enhanced in triple-negative breast cancer, and its silencing exerted suppressing influences on malignant behaviors of triple-negative breast cancer cells through mediating epidermal growth factor receptor/Janus kinase/signal transducer and activator of transcription 3 pathway.[8] In addition, NCAPG, positively mediated by galectin-1, was highly expressed in GC and NCAPG knockdown decreased migrative and invasive capabilities of GC cells.[11] The abovementioned evidence indicated that NCAPG served as a promoter in diverse cancers. Furthermore, a review, published in 2022, summarized the abnormally higher expression and promoting role of NCAPG in 9 types of cancers.[7] Several studies suggested that NCAPG was a valuably prognostic biomarker for numerous cancers.[12–16] Although there is a lot of experimental evidence that NCAPG has a tumor-promoting effect in a variety of tumors, there is a lack of comprehensive studies on NCAPG in pan-cancer. Therefore, our study aimed to fully and systematically understand the role of NCAPG in pan-cancer.

Considering that the research data on NCAPG in pan-cancer were few and the analysis was not comprehensive enough, we performed the systemic analysis to illustrate the underlying role of NCAPG in pan-cancer. We retrieved resources including The Cancer Genome Atlas Program (TCGA), Genotype-Tissue Expression (GTEx), Human Protein Atlas (HPA), and University of California Santa Cruz (UCSC) Xena to investigate the abundance of NCAPG across cancer types and normal tissues. In addition, we analyzed the interaction of NCAPG and the potential molecules, associations of NCAPG and diseases and phenotypes, diagnostic value, prognosis of NCAPG, and influences of NCAPG on immune cell infiltration in pan-cancer. We believe that our study will contribute to a better understanding of the role of NCAPG in the development and progression of various malignancies.

## 2. Methods

### 2.1. The analysis of interacting molecules with NCAPG

Three databases including Search Tool for the Retrieval of Interacting Genes/Proteins (STRING; https://string-db.org/), GeneMANIA (http://genemania.org/), and Global Positioning System (GPS)-Prot (http://gpsprot.org/) were used for searching interacting molecules with NCAPG. Among these 3 databases, we set the max number of interactors to show 15 for STRING and set Min Conf score = 0 and Zoom = 1 for GPS-Prot, while all of the other parameters were default.

### 2.2.The analysis of NCAPG-related diseases and the interaction of NCAPG and 14 cancer functional states

The Open Targets Platform (https://platform.opentargets.org/) was employed for analyzing NCAPG-related diseases. In addition, the correlation between NCAPG and 14 cancer functional states including angiogenesis, apoptosis, cell cycle, differentiation, DNA damage, DNA repair, epithelial-mesenchymal transition, hypoxia, inflammation, invasion, metastasis, proliferation, quiescence, and stemness was analyzed using the CancerSEA website (http://biocc.hrbmu.edu.cn/CancerSEA/home.jsp). Among these 2 databases, we set minimum score = 0.05 for the Open Targets Platform, while all of the other parameters were default.

### 2.3. Gene expression analysis

The expression difference of NCAPG between various tumor types and corresponding normal tissues of TCGA (https://www.cancer.gov/ccg/research/genome-sequencing/tcga), GTEx (https://www.gtexportal.org/home/), and UCSC Xena (https://xenabrowser.net/) databases was analyzed using XIANTAO Academic (https://www.xiantao.love/products), which collected the cleaned and filtered RNA-seq and miRNA-seq data in multicancer transcripts per million and fragments per kilobase of exon per million mapped reads formats.[17,18] NCAPG expressions in human tissues, single cells, and cell lines were obtained from the HPA database (https://www.proteinatlas.org/). The paired comparison of NCAPG expression of the pan-cancer with case-matched adjacent normal tissues from the TCGA database was analyzed using the XIANTAO Academic. We conducted the analyses with default parameters by using both XIANTAO Academic and HPA.

### 2.4. Immunohistochemistry staining

Immunohistochemistry (IHC) images of NCAPG protein expression in different normal tissues/organs and corresponding cancer tissues were obtained from the HPA database to probe protein expression differences.

### 2.5. Analysis of diagnostic value and prognosis of NCAPG

Variations within NCAPG expression for diagnostic value and prognosis of cancers using RNA sequencing data were acquired from TCGA, and XIANTAO Academic analyzed these data statistically. XIANTAO Academic was used to the receiver operating characteristic (ROC) curve and area



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under the ROC curve, which was applied for evaluating the diagnostic value of NCAPG in pan-cancer. Diagnostic value: high accuracy (area under the curve [AUC] > 0.9), certain accuracy (AUC, 0.7–0.9), and low accuracy (AUC, 0.5–0.7). XIANTAO Academic was used for analyzing overall survival (OS) and disease-free survival, progression-free interval (PFI),

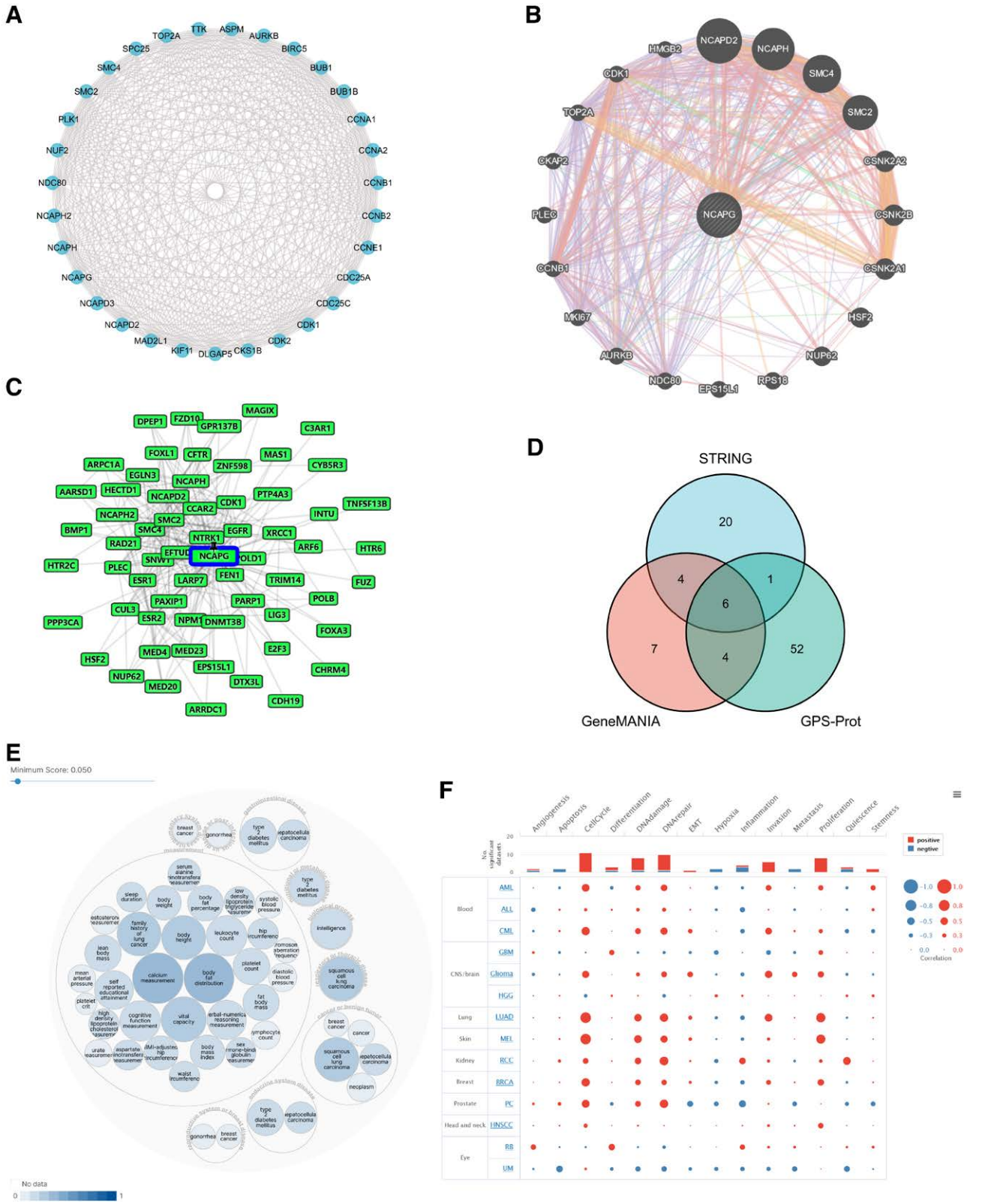


**Figure 1.** Analysis of molecules interacting with non-SMC condensin I complex subunit G (NCAPG) and diseases and phenotypes associated with NCAPG. (A–C) The interactions of NCAPG and molecules were predicted using Search Tool for the Retrieval of Interacting Genes/Proteins (STRING), GeneMANIA, and Global Positioning System (GPS)-Prot, respectively. (D) Molecules overlapped with NCAPG interactions in 3 databases. (E) NCAPG participation in diseases was predicted using the Open Targets Platform. (F) NCAPG relating to 14 cancer functional states was predicted using CancerSEA.

and disease-specific survival (DSS) to explore the prognostic value of NCAPG in cancers across all TCGA tumors. We conducted all these analyses with default parameters by using XIANTAO Academic.

### *2.6. The analysis of immune infiltration and immune-related molecules*

The Tumor Immune Estimation Resource Version 2 (TIMER2) website (http://timer.cistrome.org/) was used to analyze the

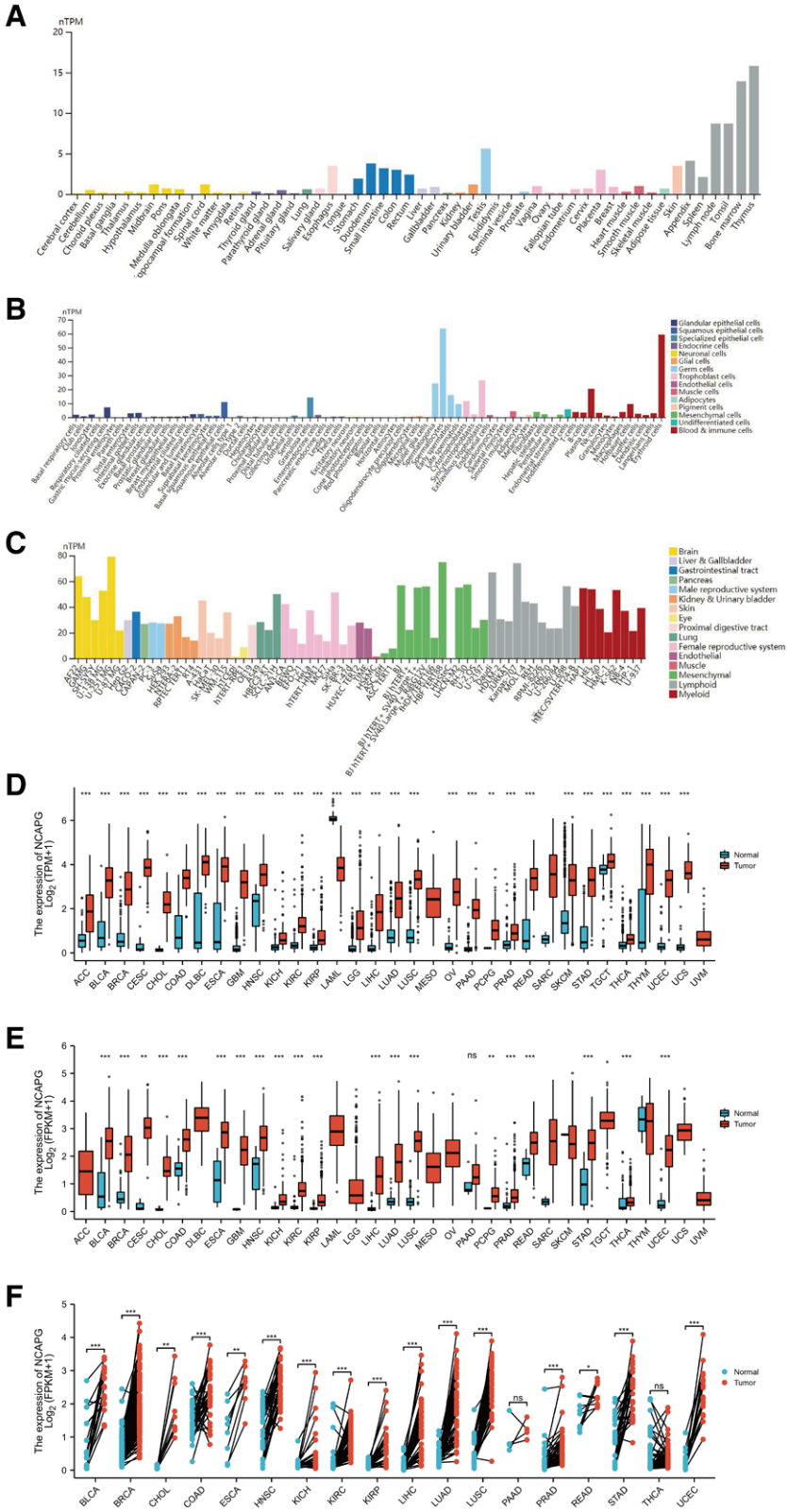


**Figure 2.** Expression profile of non-SMC condensin I complex subunit G (NCAPG) in pan-cancer. (A) Expression profile of NCAPG in human tissues. (B) Expression profile of NCAPG in single cells. (C) Expression profile of NCAPG in cell lines including cancer cell lines. (D and E) Differential expression of NCAPG in tumor tissues and normal tissues. (F) Differential expression of NCAPG in tumor tissues and paired normal tissues.

correlation between the expression of NCAPG and different immune cell infiltrations in numerous tumor types. The scores of immune infiltrating cells of 33 tumors were achieved from the TIMER2 database. The Tumor and Immune System Interaction Database (TISIDB; http://cis.hku.hk/TISIDB/) was employed for analyzing the correlation between NCAPG and tumor-infiltrating lymphocytes/immune inhibitors/immune stimulators/major histocompatibility complex (MHC) molecule/chemokines/chemokines receptors. The Kaplan-Meier plotter (https://kmplot.com/analysis/) was used for exploring the effect of different immune cell infiltration on kidney renal clear cell carcinoma (KIRC) and thyroid carcinoma (THCA) OS prognosis.[19] We set the split patients by the median for the Kaplan-Meier plotter, while all of the other parameters were default for these 3 databases.

## 3. Results

### 3.1. Analysis of molecules interacting with NCAPG and diseases and phenotypes associated with NCAPG

First, we analyzed the close molecules interacting with NCAPG by searching databases, including STRING, GeneMANIA, and GPS-Prot. Figure 1A exhibits the network of NCAPG interacting with the latent molecules, which was obtained from searching the STRING database. Similarly, through hunting GeneMANIA, the intercorrelations of the relative genes and NCAPG are presented in Figure 1B. GPS-Prot also predicted that various molecules were possibly bound with NCAPG (Fig. 1C). Of note, by making Venn diagrams, it was found that there were 6 overlapping genes (including NCAPG itself) in the 3 databases, namely, cyclin-dependent kinase 1, non-SMC condensin I complex subunit D2, non-SMC condensin I complex subunit H, structural maintenance of chromosome 2, and structural maintenance of chromosome 4, NCAPG (Fig. 1D), suggesting these 5 molecules had intimate relationships with NCAPG. Subsequently, we employed the Open Targets Platform to predict which diseases NCAPG may participate in. The results displayed that 132 diseases or phenotypes were in connection with NCAPG (Fig. 1E). Intriguingly, we observed that the most intimate diseases with NCAPG were tumors such as BC, hepatocellular carcinoma, and squamous cell lung carcinoma. Afterward, CancerSEA was used to analyze the correlations between NCAPG and 14 cancer functional states including angiogenesis, apoptosis, cell cycle, differentiation, DNA damage, DNA repair, epithelial-mesenchymal transition, hypoxia, inflammation, invasion, metastasis, proliferation, quiescence, and stemness. As presented in Figure 1F, there was a significant positive correlation between NCAPG and cell cycle in LUAD, melanoma (MEL), and breast invasive carcinoma (BRCA). Besides, in MEL, LUAD, and renal cell carcinoma, NCAPG was significantly positively correlated with DNA damage. In addition, NCAPG was positively correlated with DNA repair in renal cell carcinoma, prostate cancer (PC), and LUAD. Moreover, in LUAD, glioma, and chronic myelogenous leukemia, NCAPG was observably positively correlated with invasion. Finally, there was a positive relationship between NCAPG and proliferation in LUAD, MEL, and BRCA. Of note, due to the large amount of data, as a functional representation, we only described 5 cancer functional states (cell cycle, DNA damage, DNA repair, invasion, and proliferation) that were significantly positively correlated with NCAPG and only described the 3 tumors in the top 3 (the correlation was in descending order). NCAPG was positively associated with cell cycle, DNA repair and damage, proliferation, and invasion of most tumors.

### 3.2. Expression profile of NCAPG in pan-cancer

The abundance of NCAPG in different normal tissues from humans was achieved through the HPA database. Among various tissue types, bone marrow and lymphoid tissue had the highest

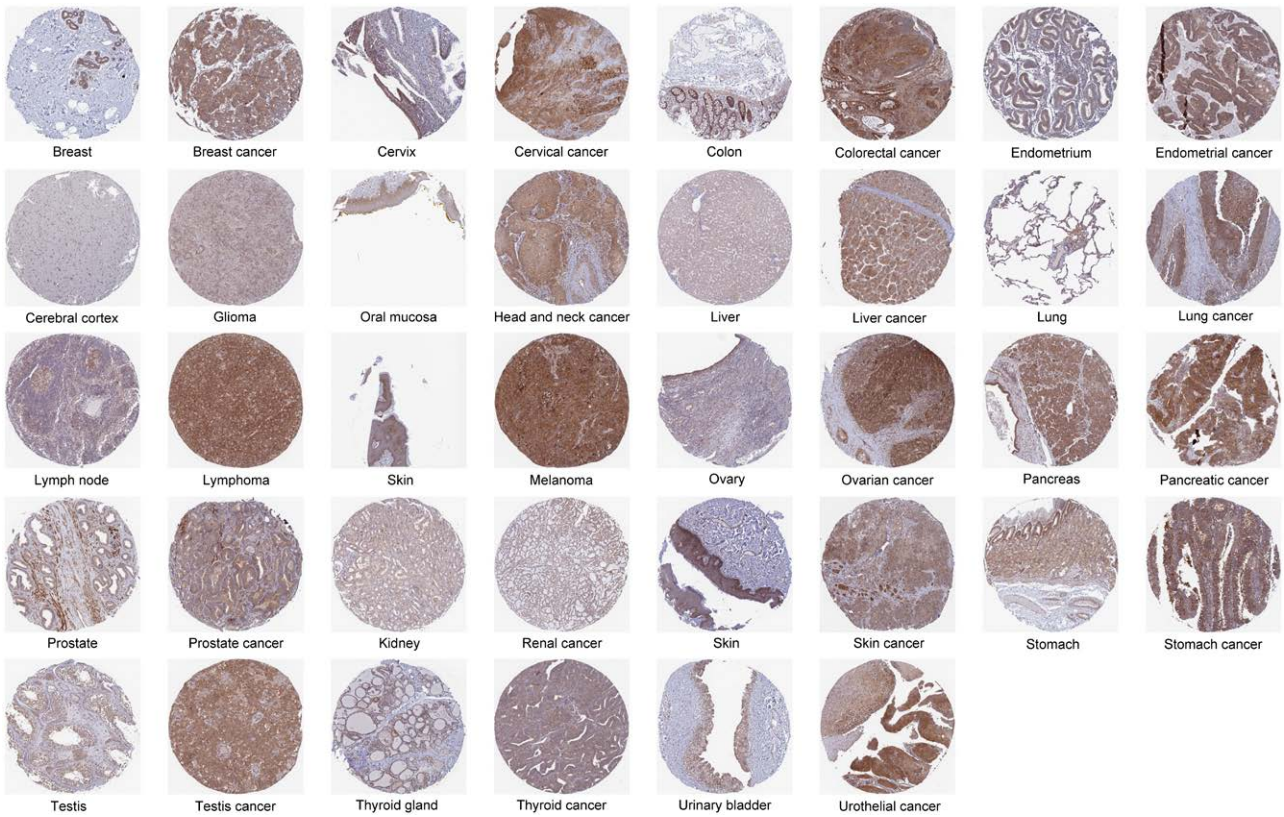


**Figure 3.** Differential expression of non-SMC condensin I complex subunit G (NCAPG) protein in different normal tissues/organs and corresponding cancer tissues.

expression of NCAPG (Fig. 2A). We also investigated the expression profile of NCAPG in multiple single cells. The results exhibited that NCAPG in spermatocytes, erythroid cells, extravillous trophoblasts, spermatogonia, and plasma cells had relatively higher expression than in other single cells (Fig. 2B). As presented in Figure 2C, NCAPG in different cell lines including cancer cells and noncancer cells was expressed, and data were achieved from the HPA database. In addition, we pooled and analyzed NCAPG expression in a total of 15,776 cancer sample numbers (unpaired samples) from UCSC Xena, TCGA, and GTEx databases and a total of 11,093 cancer sample numbers (unpaired samples) from the TCGA database merely. The results indicated that NCAPG expression in acute myeloid leukemia (LAML) was significantly lower than that in the normal group, while it was significantly higher in other cancer types including adrenocortical carcinoma (ACC), bladder urothelial carcinoma (BLCA),

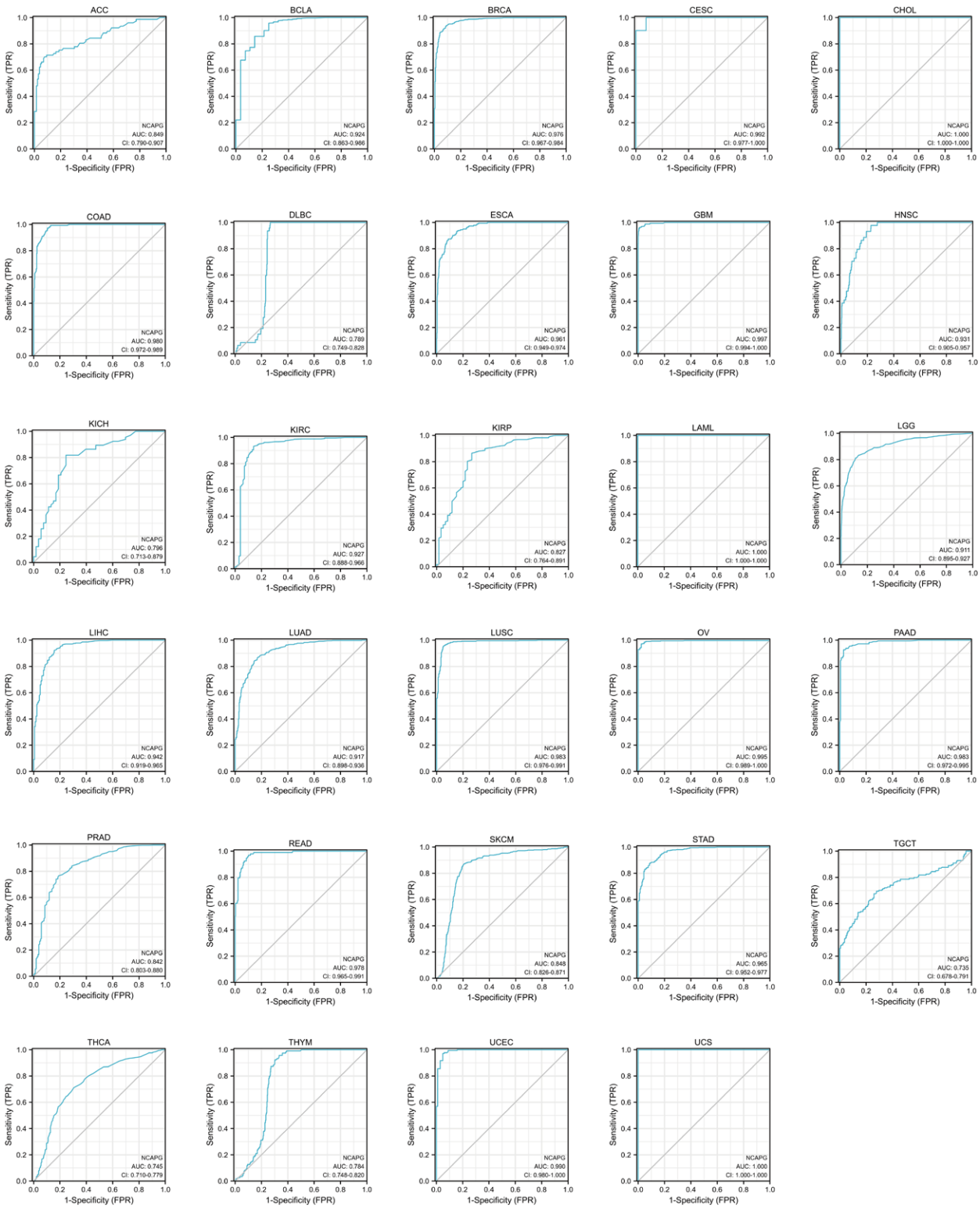


**Figure 4.** Receiver operating characteristic (ROC) curves of non-SMC condensin I complex subunit G (NCAPG) in different cancer species.

BRCA, cervical squamous cell carcinoma and endocervical adenocarcinoma (CESC), cholangiocarcinoma (CHOL), colon adenocarcinoma (COAD), lymphoid neoplasm diffuse large B-cell lymphoma (DLBC), esophageal carcinoma (ESCA), glioblastoma multiforme (GBM), head and neck squamous cell carcinoma (HNSC), kidney chromophobe (KICH), KIRC, kidney renal

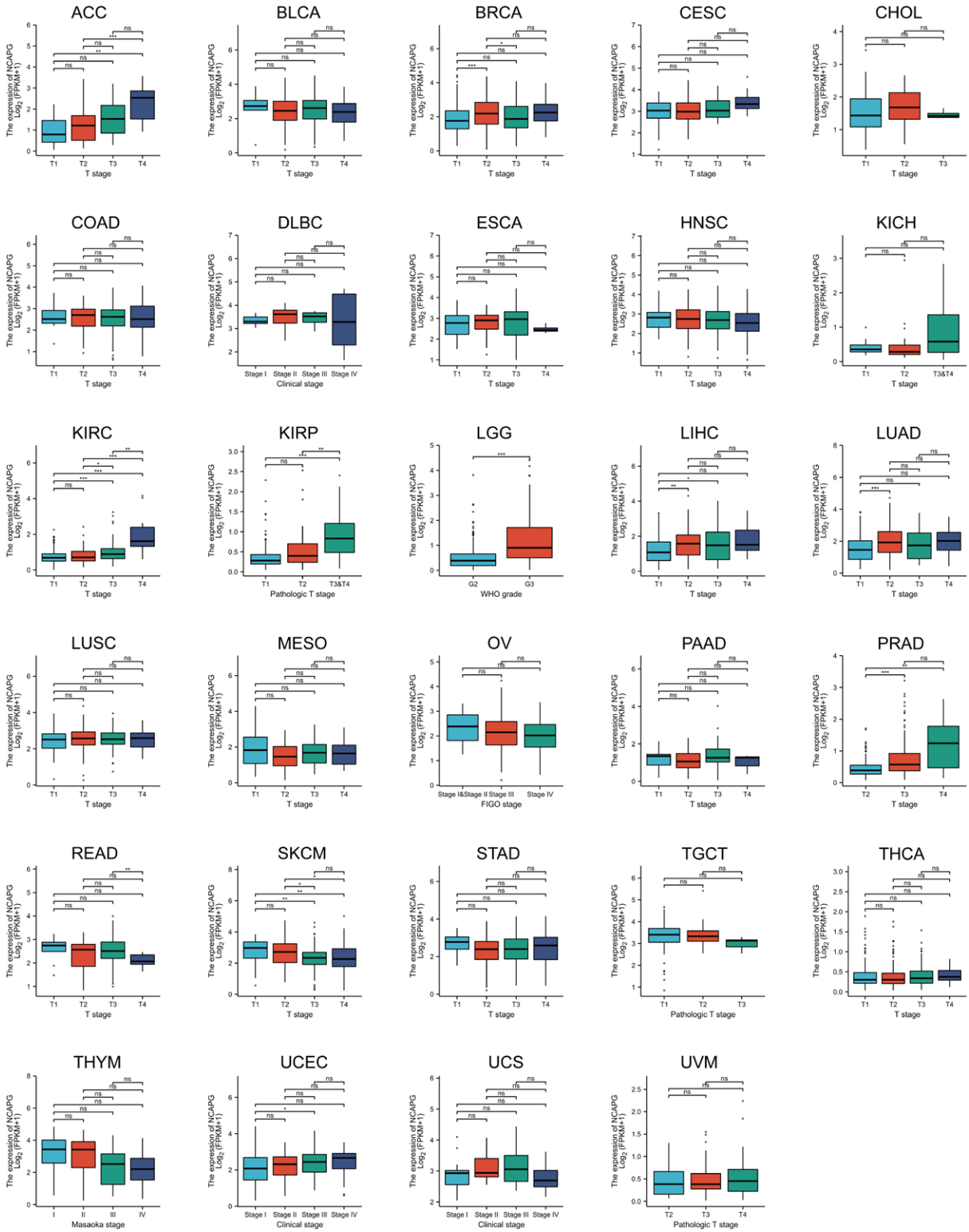


**Figure 5.** The differential expression of non-SMC condensin I complex subunit G (NCAPG) in different cancer species and different stages. ACC = adrenocortical carcinoma, BLCA = bladder urothelial carcinoma, BRCA = breast invasive carcinoma, CESC = cervical squamous cell carcinoma and endocervical adenocarcinoma, CHOL = cholangiocarcinoma, COAD = colon adenocarcinoma, DLBC = lymphoid neoplasm diffuse large B-cell lymphoma, ESCA = esophageal carcinoma, HNSC = head and neck squamous cell carcinoma, KICH = kidney chromophobe, KIRC = kidney renal clear cell carcinoma, KIRP = kidney renal papillary cell carcinoma, LGG = brain lower grade glioma, LIHC = liver hepatocellular carcinoma, LUAD = lung adenocarcinoma, LUSC = lung squamous cell carcinoma, MESO = mesothelioma, OV = ovarian serous cystadenocarcinoma, PAAD = pancreatic adenocarcinoma, PRAD = prostate adenocarcinoma, READ = rectum adenocarcinoma esophageal carcinoma, SKCM = skin cutaneous melanoma, STAD = stomach adenocarcinoma, TGCT = testicular germ cell tumor, THCA = thyroid carcinoma, THYM = thymoma, UCEC = uterine corpus endometrial carcinoma, UCS = uterine carcinosarcoma, UVM = uveal melanoma.

papillary cell carcinoma (KIRP), brain lower grade glioma (LGG), liver hepatocellular carcinoma (LIHC), LUAD, lung squamous cell carcinoma (LUSC), mesothelioma (MESO), ovarian serous cystadenocarcinoma (OV), pancreatic adenocarcinoma (PAAD), pheochromocytoma and paraganglioma (PCPG), prostate adenocarcinoma (PRAD), rectum adenocarcinoma esophageal carcinoma (READ), sarcoma (SARC), skin cutaneous melanoma (SKCM), stomach adenocarcinoma (STAD), testicular germ cell tumors (TGCTs), THCA, thymoma (THYM), uterine corpus endometrial carcinoma (UCEC), uterine carcinosarcoma (UCS), and uveal melanoma (UVM; Fig. 2D and 2E). We retrieved the TCGA database and obtained the paired samples of various cancers. NCAPG mRNA expression was increased in BLCA, BRCA, CHOL, COAD, ESCA, HNSC, KICH, KIRC, KIRP, LIHC, LUAD, LUSC, PRAD, STAD, READ, and UCEC while no statistic difference in PAAD and THCA (Fig. 2F).

Then, we focused on NCAPG protein expression in normal tissues and cancer tissues. Figure S1A, Supplemental Digital Content, http://links.lww.com/MD/O466, presented the protein expression profile of NCAPG in organs of human, and NCAPG was widely expressed in different organs. Further data suggested that NCAPG protein levels were higher in tissues including cerebellum, nasopharynx, esophagus, stomach, rectum, placenta, smooth muscle, lymph node, and tonsil, whereas there was almost no expression in tissues including parathyroid gland, liver, seminal vesicle, skeletal muscle, and adipose tissue (Figure S1B, Supplemental Digital Content, http://links.lww.com/MD/O466). Moreover, according to IHC results from the HPA database, NCAPG protein levels in cancer tissues of various cancers including BC, cervical cancer, CRC, endometrial cancer, glioma, head and neck cancer, live cancer, lung cancer, lymphoma, MEL, ovarian cancer (OV), pancreatic cancer, PC, renal cancer, skin cancer, stomach cancer, testis cancer, thyroid cancer, and urothelial cancer were significantly increased compared with the corresponding normal tissues (Fig. 3).

### 3.3. Diagnostic value of NCAPG in pan-cancer

The ROC curve was applied to analyze the diagnostic value of NCAPG in various cancers. As shown in Figure 4, NCAPG might serve as an excellent diagnostic marker in ACC (AUC = 0.849), BCLA (AUC = 0.924), BRCA (AUC = 0.976), CESC (AUC = 0.992), CHOL (AUC = 1.000), COAD (AUC = 0.980), DLBC (AUC = 0.789), ESCA (AUC = 0.961), GBM (AUC = 0.997), HNSC (AUC = 0.931), KICH (AUC = 0.796), KIRC (AUC = 0.927), KIRP (AUC = 0.827), LAML (AUC = 1.000), LGG (AUC = 0.911), LIHC (AUC = 0.942), LUAD (AUC = 0.917), LUSC (AUC = 0.983), OV

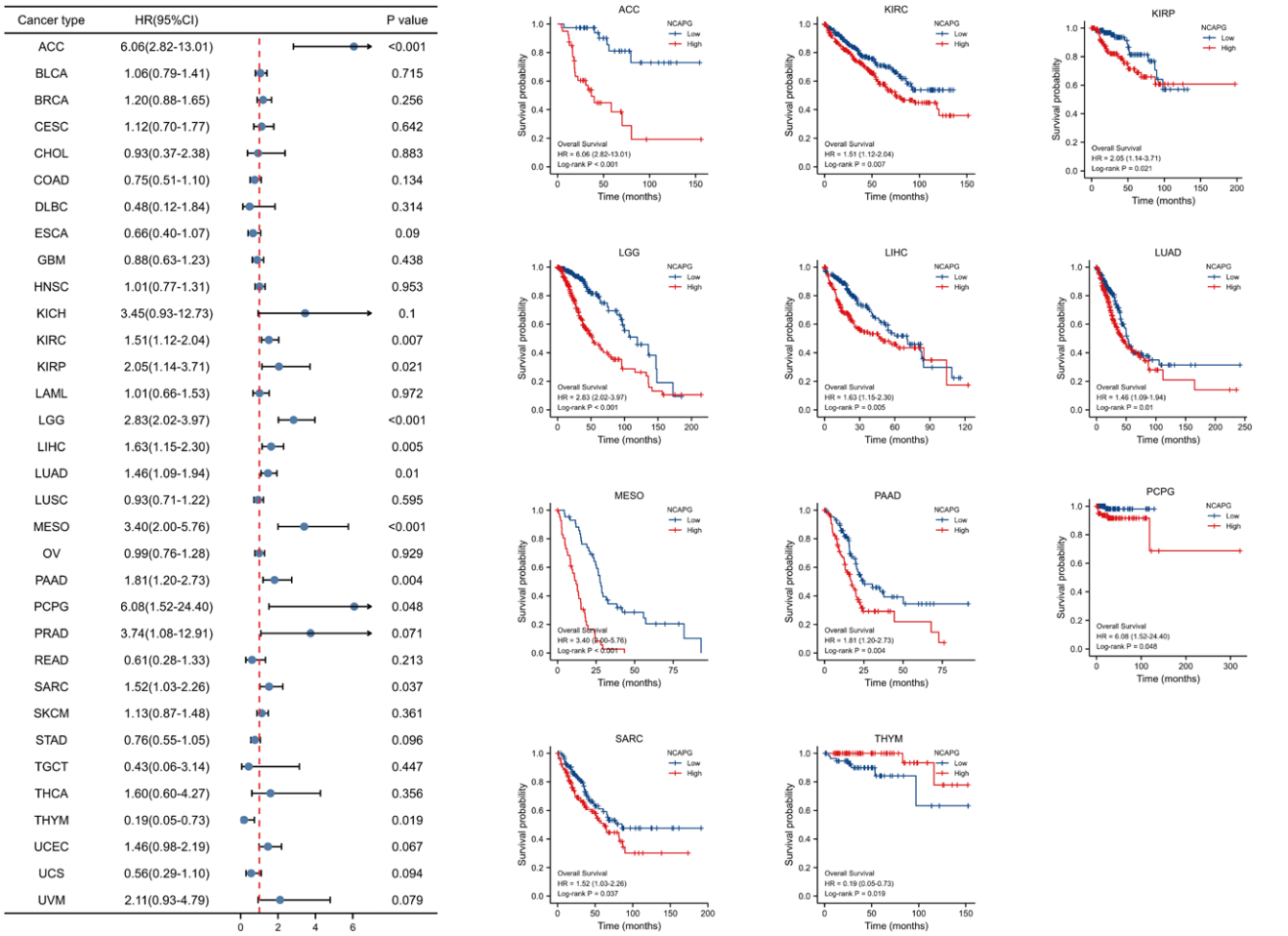


**Figure 6.** The relationship between non-SMC condensin I complex subunit G (NCAPG) expression and overall survival (OS). ACC = adrenocortical carcinoma, BLCA = bladder urothelial carcinoma, BRCA = breast invasive carcinoma, CESC = cervical squamous cell carcinoma and endocervical adenocarcinoma, CHOL = cholangiocarcinoma, COAD = colon adenocarcinoma, DLBC = lymphoid neoplasm diffuse large B-cell lymphoma, ESCA = esophageal carcinoma, HNSC = head and neck squamous cell carcinoma, KICH = kidney chromophobe, KIRC = kidney renal clear cell carcinoma, KIRP = kidney renal papillary cell carcinoma, LGG = brain lower grade glioma, LIHC = liver hepatocellular carcinoma, LUAD = lung adenocarcinoma, LUSC = lung squamous cell carcinoma, MESO = mesothelioma, OV = ovarian serous cystadenocarcinoma, PAAD = pancreatic adenocarcinoma, PRAD = prostate adenocarcinoma, READ = rectum adenocarcinoma esophageal carcinoma, SKCM = skin cutaneous melanoma, STAD = stomach adenocarcinoma, TGCT = testicular germ cell tumor, THCA = thyroid carcinoma, THYM = thymoma, UCEC = uterine corpus endometrial carcinoma, UCS = uterine carcinosarcoma, UVM = uveal melanoma.

(AUC = 0.995), PAAD (AUC = 0.983), PRAD (AUC = 0.842), READ (AUC = 0.978), SKCM (AUC = 0.848), STAD (AUC = 0.965), TGCT (AUC = 0.735), THCA (AUC = 0.745), THYM (AUC = 0.784), UCEC (AUC = 0.990), and UCS (AUC = 1.000).

For tumor stages or grades of NCAPG expression, we observed that NCAPG expression significantly increased in the advanced stages of ACC, BRCA, KIRC, KIRP, LGG, LIHC, PRAD, READ, and SKCM. However, there was no significant difference in BLCA, CESC, CHOL, COAD, DLBC, ESCA, HNSC, KICH, LUSC, MESO, OV, PAAD, STAD, TGCT, and THCA (Fig. 5). Meanwhile, the alteration of NCAPG expression with gender is presented in Figure S2, Supplemental Digital Content, http://links.lww.com/MD/O466. The higher expression in males than in females was found in KIRC, LAML, and LUAD but had the opposite effects in KIRP, SARC, and STAD. There was no significant difference between NCAPG and gender in the remaining cancer types in the picture.

### 3.4. Prognostic significance of NCAPG in pan-cancer

To further explore the correlation between NCAPG expression and prognosis in pan-cancer, we analyzed the effects of relatively high or low NCAPG expression on OS, DSS, and PFI in various tumors. The Cox proportional hazards model was applied to analyze and found that the expression levels of NCAPG were connected with OS in ACC ($P < .001$), KIRC ($P = .007$), KIRP ($P = .021$), LGG ($P < .001$), LIHC ($P = .005$), LUAD ($P = .01$), MESO ($P < .001$), PAAD ($P = .004$), PCPG ($P = .048$), SARC ($P = .037$), and THYM ($P = .019$). Subsequently, the Kaplan-Meier survival analysis presented that lower NCAPG expression had a favorable OS in patients with ACC, KIRC, KIRP, LGG, LIHC, LUAD, MESO, PAAD, PCPG, and SARC, while it had an undesirable OS in patients with THYM (Fig. 6).

Furthermore, DSS data analysis was also conducted. The results displayed the relationships between higher NCAPG expression and dissatisfied prognosis in patients with ACC ($P < .001$), KIRC ($P < .001$), KIRP ($P < .001$), LGG ($P < .001$), LIHC ($P = .003$), LUAD ($P = .025$), MESO ($P < .001$), PAAD ($P = .009$), PCPG ($P = .021$), PRAD ($P = .035$), SARC ($P = .035$), and UCEC ($P = .029$; Fig. 7).

In terms of associations between NCAPG expression and PFI in various cancers, higher NCAPG expression was obtained poor PFI in ACC ($P < .001$), KIRC ($P = .001$), KIRP ($P < .001$), LGG ($P < .001$), LIHC ($P = .001$), LUAD ($P = .023$), MESO ($P < .001$), PAAD ($P < .001$), PCPG ($P = .027$), PRAD ($P < .001$),

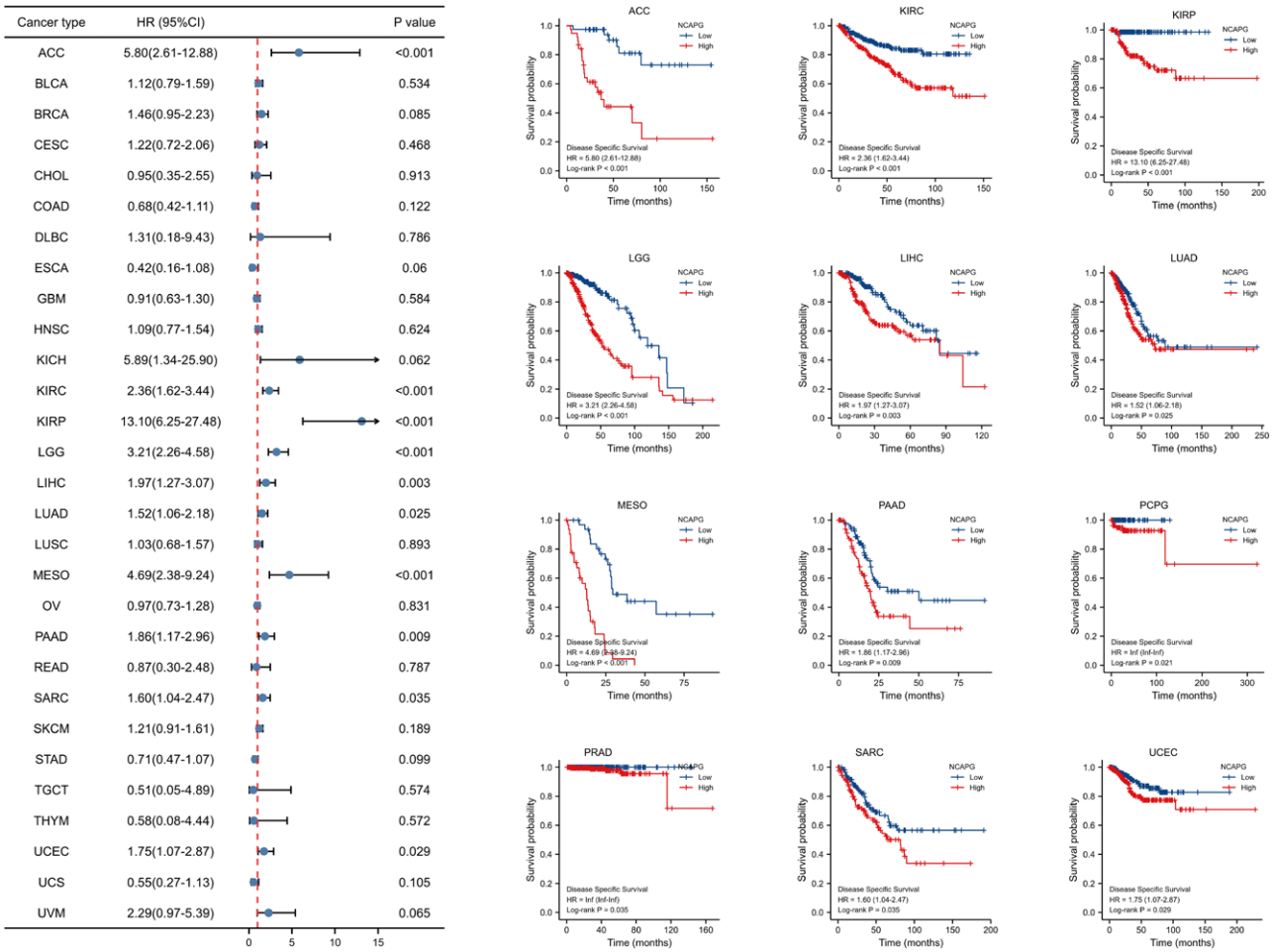


**Figure 7.** The relationship between non-SMC condensin I complex subunit G (NCAPG) expression and disease-specific survival (DSS). ACC = adrenocortical carcinoma, BLCA = bladder urothelial carcinoma, BRCA = breast invasive carcinoma, CESC = cervical squamous cell carcinoma and endocervical adenocarcinoma, CHOL = cholangiocarcinoma, COAD = colon adenocarcinoma, DLBC = lymphoid neoplasm diffuse large B-cell lymphoma, ESCA = esophageal carcinoma, HNSC = head and neck squamous cell carcinoma, KICH = kidney chromophobe, KIRC = kidney renal clear cell carcinoma, KIRP = kidney renal papillary cell carcinoma, LGG = brain lower grade glioma, LIHC = liver hepatocellular carcinoma, LUAD = lung adenocarcinoma, LUSC = lung squamous cell carcinoma, MESO = mesothelioma, OV = ovarian serous cystadenocarcinoma, PAAD = pancreatic adenocarcinoma, PRAD = prostate adenocarcinoma, READ = rectum adenocarcinoma esophageal carcinoma, SKCM = skin cutaneous melanoma, STAD = stomach adenocarcinoma, TGCT = testicular germ cell tumor, THCA = thyroid carcinoma, THYM = thymoma, UCEC = uterine corpus endometrial carcinoma, UCS = uterine carcinosarcoma, UVM = uveal melanoma.

SARC ($P$ = .008), THCA ($P$ = .022), UCEC ($P$ = .023), and UVM ($P$ = .001), while it observed good PFI in patients with COAD ($P$ = .034) and STAD ($P$ = .007; Fig. 8).

### 3.5. Immune cell infiltration analyses

Here, the connection between NCAPG expression and immune cell infiltration was analyzed using TIMER2. We observed that the expression of NCAPG was positively correlated with $CD4^+$T and myeloid-derived suppressor cells, but it is difficult to determine whether NCAPG expression was positively or negatively correlated with other immune cells including $CD8^+$ T cells, Tregs, neutrophils and monocytes, B cell, macrophages, dendritic cell, natural killer (NK) cell, mast cells, cancer-associated fibroblast, common lymphoid progenitor, common myeloid progenitor, endothelial cells, eosinophil, granulocyte-monocyte progenitor, hematopoietic stem cells, T-cell follicular helper, T-cell gamma delta, and NK T cells (Fig. 9).

Moreover, TISIDB databases were applied to analyze the correlation between NCAPG expression and tumor-infiltrating lymphocytes/immune inhibitors/immune stimulators/MHC molecule/chemokines/chemokines receptors in cancers using the TISIDB database. The results exhibited that the NCAPG expression was positively related to Act CD4 and helper T-cell 2 (Th2) infiltration but negatively related to most other immune cells' infiltration in 33 cancers (Fig. 10A). It was worth noting that in KIRC and THCA, there existed an apparently positive correlation between NCAPG expression and almost all immune cells' infiltration (Fig. 10A). In addition, NCAPG expression was negatively connected with immune inhibitors in most cancers, especially in ESCA, GBM, LUSC, READ, and UCS; however, a strongly positive correlation was found in KIRC and THCA (Fig. 10B). More interestingly, the negative relationships between NCAPG expression and immune stimulators/MHC molecule/chemokines/chemokines receptors were observed in most cancers, but, in KIRC and THCA, we also observed notably positive relationships (Fig. 10C through 10F).

### 3.6. Immune characteristics of NCAPG, KIRC, and THCA

Based on the results of Figure 10, we summarized that NCAPG expression was most closely related to immunoregulation in KIRC and THCA. Therefore, our attention was concentrated on the role of NCAPG in immune cell infiltration in KIRC and THCA. Our results revealed that NCAPG expression was positively connected with most immune cell infiltration in KIRC especially activated CD4 T cells (Act_CD4; $\rho = 0.732$; $P < 2.2\times10^{-16}$), Th2 ($\rho = 0.472$; $P < 2.2\times10^{-16}$), memory B cells ($\rho = 0.43$; $P < 2.2\times10^{-16}$), activated CD8 T cells ($\rho = 0.397$; $P < 2.2\times10^{-16}$), and myeloid suppressor cells ($\rho = 0.351$; $P = 6.35\times10^{-17}$). In addition, we observed a stronger positive correlation between NCAPG and most immune cell infiltration in THCA, especially Act_CD4 ($\rho = 0.722$; $P < 2.2\times10^{-16}$), Th2 ($\rho = 0.519$; $P < 2.2\times10^{-16}$), γδt cells (Tgd; $\rho = 0.515$; $P < 2.2\times10^{-16}$), effector memory CD4 T cells (Tem_CD4; $\rho = 0.512$; $P < 2.2\times10^{-16}$), and natural killer T cell ($\rho = 0.501$; $P < 2.2\times10^{-16}$; Figs. 11 and 12).

As presented in Figure 13 and Figure S3, Supplemental Digital Content, http://links.lww.com/MD/O466, we identified

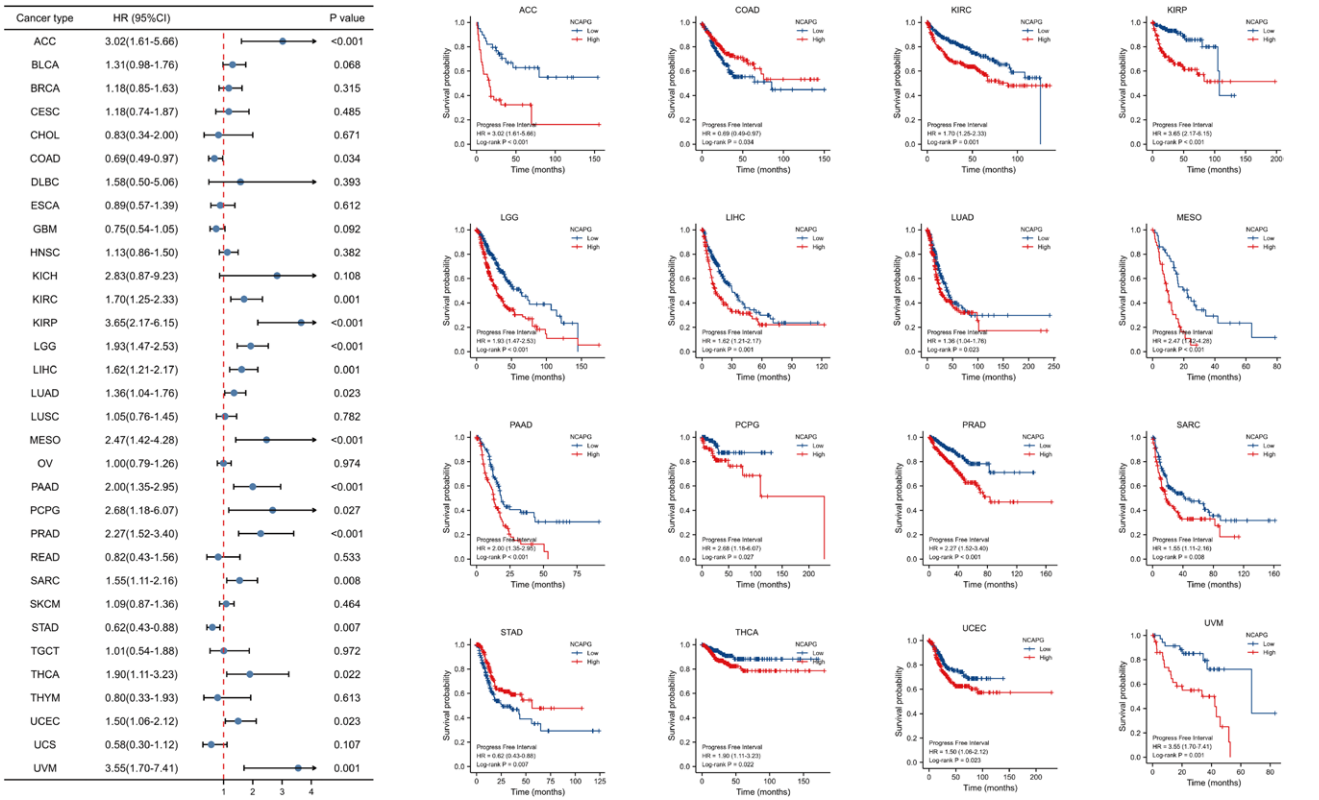


**Figure 8.** The relationship between non-SMC condensin I complex subunit G (NCAPG) expression and progression-free interval (PFI). ACC = adrenocortical carcinoma, BLCA = bladder urothelial carcinoma, BRCA = breast invasive carcinoma, CESC = cervical squamous cell carcinoma and endocervical adenocarcinoma, CHOL = cholangiocarcinoma, COAD = colon adenocarcinoma, DLBC = lymphoid neoplasm diffuse large B-cell lymphoma, ESCA = esophageal carcinoma, HNSC = head and neck squamous cell carcinoma, KICH = kidney chromophobe, KIRC = kidney renal clear cell carcinoma, KIRP = kidney renal papillary cell carcinoma, LGG = brain lower grade glioma, LIHC = liver hepatocellular carcinoma, LUAD = lung adenocarcinoma, LUSC = lung squamous cell carcinoma, MESO = mesothelioma, OV = ovarian serous cystadenocarcinoma, PAAD = pancreatic adenocarcinoma, PRAD = prostate adenocarcinoma, READ = rectum adenocarcinoma esophageal carcinoma, SKCM = skin cutaneous melanoma, STAD = stomach adenocarcinoma, TGCT = testicular germ cell tumor, THCA = thyroid carcinoma, THYM = thymoma, UCEC = uterine corpus endometrial carcinoma, UCS = uterine carcinosarcoma, UVM = uveal melanoma.

that high NCAPG expression led to a poor prognosis in KIRC but regrettably here had no impact on prognosis in THCA, regardless of the enrichment of immune cells. Furthermore, the enrichment of a single type of immune cells in KIRC and THCA was taken into consideration for the prognosis of NCAPG expression. The results displayed the enrichment of most immune cells including basophils, B cells, $CD4^+$ memory T cells, $CD8^+$ T cells, macrophages, mesenchymal stem cells, NK T cells, type 1 T-helper cells, and type 2 T-helper cells, while a decrease in eosinophils, regulatory T cells, and type 1 T-helper cells promoted high NCAPG expression-mediated poor prognosis in KIRC. Intriguingly, both enrichment and reduction of type 1 T-helper cells exacerbated the poor prognosis of KIRC. However, the enrichment or decrease of immune cells had no effects on NCAPG expression for prognosis in THCA.

## 4. Discussion

The pan-cancer analysis aims to identify genes, pathways, biomarkers, or other characteristics common to multiple cancer types, explore the commonality and personality of cancer, and provide more valuable information for cancer prevention, diagnosis, and treatment.[20,21] In recent years, the analysis of key genes in the field of pan-cancer has mushroomed,[22–24] which provided more valuable reference information for experimental investigation and clinical application in the future. In this study, we expounded comprehensively the features of NCAPG from several aspects, including interactive molecules, related diseases and phenotypes, expression profiles in pan-cancer, diagnostic value, prognostic effects in pan-cancer, and immune effects in pan-cancer, especially in KIRC and THCA. Our results showed that the expression of NCAPG was abnormally increased in most tumor types and was negatively correlated with the prognosis, which had a certain prognostic value. Furthermore, the relationships between NCAPG and immune cell infiltration/immune-related molecules partly explained the correlation between NCAPG and patient prognosis, especially in KIRC and THCA. To our knowledge, our study provided comprehensive data mining and analysis of NCAPG expression and biological function from a pan-cancer perspective.

First, our study indicated that NCAPG expression was significantly upregulated in most cancer types compared with normal tissues, suggesting that overexpression of NCAPG was a common phenotype in cancers, consistent with current experimentally relevant findings.[8–11,25] Furthermore, NCAPG expression elevated obviously in an advanced stage of ACC, BRCA, KIRC,

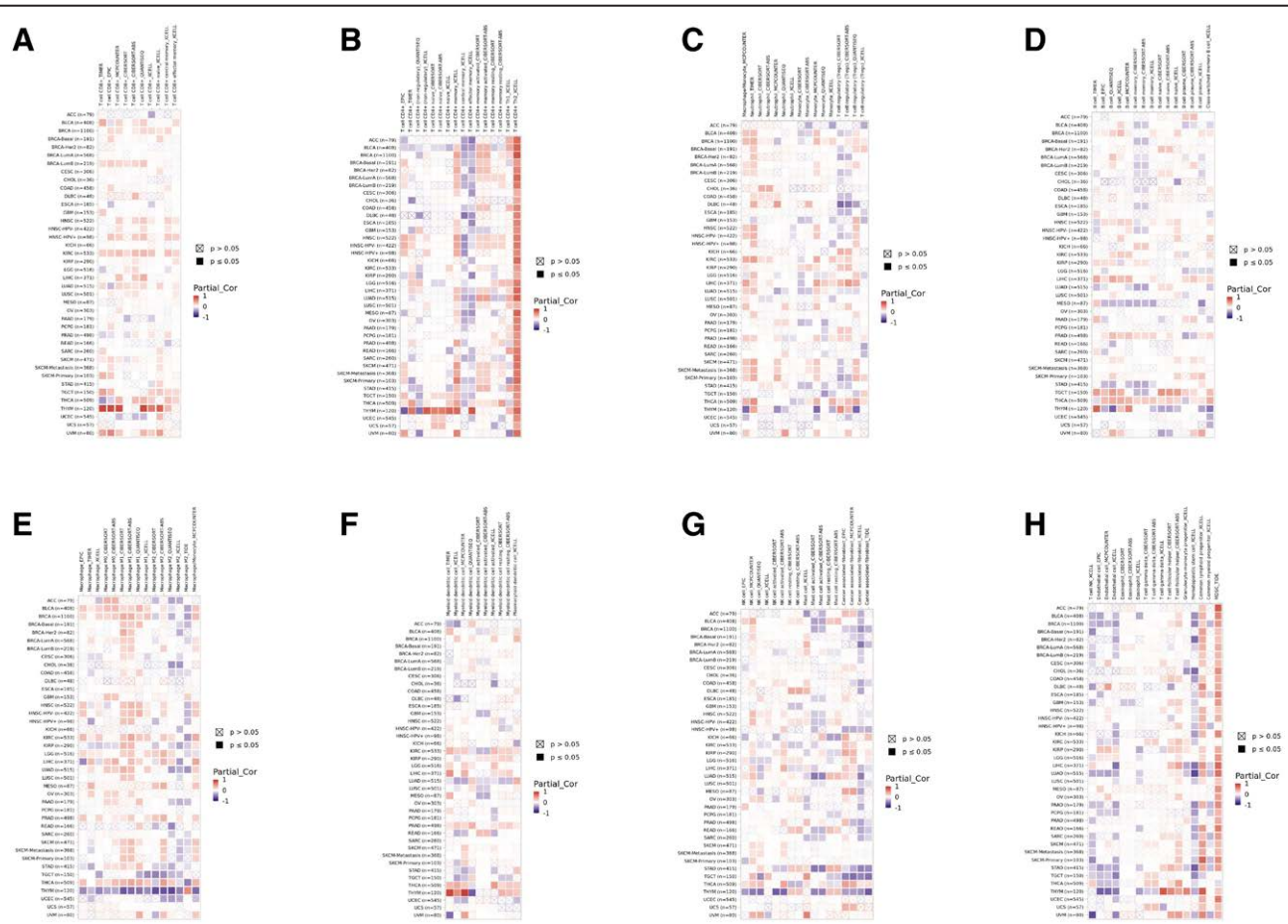


**Figure 9.** The relationships between non-SMC condensin I complex subunit G (NCAPG) and immune cell infiltration. (A–H) The correlation of NCAPG expression with T-cell CD8+, CD4+, Tregs, neutrophil, and monocyte, B cells, macrophage, dendritic cell (DC), natural killer (NK) cell, mast cell, cancer-associated fibroblast, common lymphoid progenitor, common myeloid progenitor, endothelial cell, eosinophil, granulocyte-monocyte progenitor, hematopoietic stem cell, T-cell follicular helper, T-cell gamma delta, NK T-cell, and myeloid-derived suppressor cell infiltration level. ACC = adrenocortical carcinoma, BLCA = bladder urothelial carcinoma, BRCA = breast invasive carcinoma, CESC = cervical squamous cell carcinoma and endocervical adenocarcinoma, CHOL = cholangiocarcinoma, COAD = colon adenocarcinoma, DLBC = lymphoid neoplasm diffuse large B-cell lymphoma, ESCA = esophageal carcinoma, HNSC = head and neck squamous cell carcinoma, KICH = kidney chromophobe, KIRC = kidney renal clear cell carcinoma, KIRP = kidney renal papillary cell carcinoma, LGG = brain lower grade glioma, LIHC = liver hepatocellular carcinoma, LUAD = lung adenocarcinoma, LUSC = lung squamous cell carcinoma, MESO = mesothelioma, OV = ovarian serous cystadenocarcinoma, PAAD = pancreatic adenocarcinoma, PRAD = prostate adenocarcinoma, READ = rectum adenocarcinoma esophageal carcinoma, SKCM = skin cutaneous melanoma, STAD = stomach adenocarcinoma, TGCT = testicular germ cell tumor, THCA = thyroid carcinoma, THYM = thymoma, UCEC = uterine corpus endometrial carcinoma, UCS = uterine carcinosarcoma, UVM = uveal melanoma.

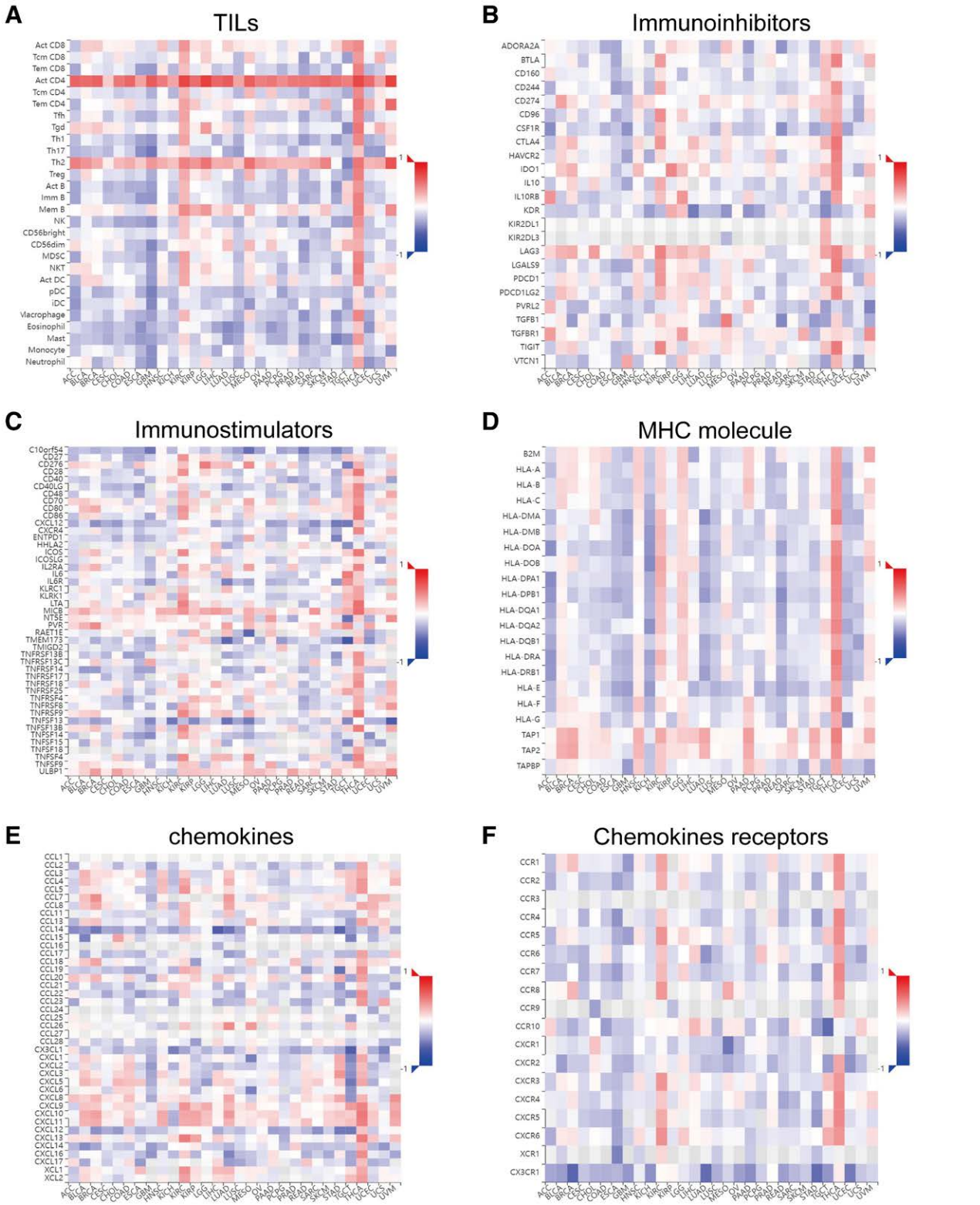


**Figure 10.** The correlation between non-SMC condensin I complex subunit G (NCAPG) expression and tumor-infiltrating lymphocytes (TILs)/immune inhibitors/immune stimulators/major histocompatibility complex (MHC) molecule/chemokines/chemokines receptors in cancers. (A–F) Spearman correlations between expression of NCAPG and TILs, immune inhibitors, immune stimulators, MHCs, chemokines, and receptors (Y axis) across human cancers (X axis). ACC = adrenocortical carcinoma, BLCA = bladder urothelial carcinoma, BRCA = breast invasive carcinoma, CESC = cervical squamous cell carcinoma and endocervical adenocarcinoma, CHOL = cholangiocarcinoma, COAD = colon adenocarcinoma, DLBC = lymphoid neoplasm diffuse large B-cell lymphoma, ESCA = esophageal carcinoma, HNSC = head and neck squamous cell carcinoma, KICH = kidney chromophobe, KIRC = kidney renal clear cell carcinoma, KIRP = kidney renal papillary cell carcinoma, LGG = brain lower grade glioma, LIHC = liver hepatocellular carcinoma, LUAD = lung adenocarcinoma, LUSC = lung squamous cell carcinoma, MESO = mesothelioma, OV = ovarian serous cystadenocarcinoma, PAAD = pancreatic adenocarcinoma, PRAD = prostate adenocarcinoma, READ = rectum adenocarcinoma esophageal carcinoma, SKCM = skin cutaneous melanoma, STAD = stomach adenocarcinoma, TGCT = testicular germ cell tumor, THCA = thyroid carcinoma, THYM = thymoma, UCEC = uterine corpus endometrial carcinoma, UCS = uterine carcinosarcoma, UVM = uveal melanoma.

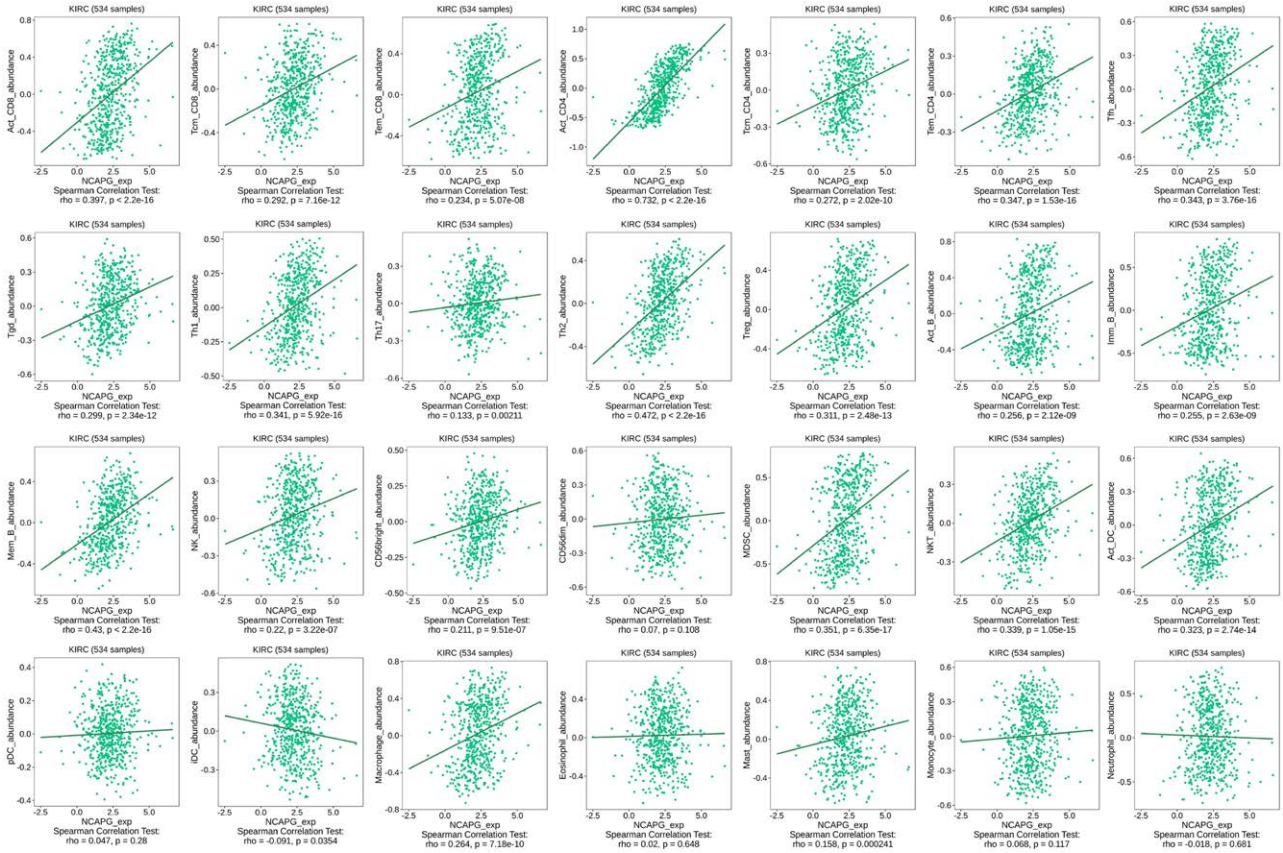


**Figure 11.** Spearman correlations between expression of non-SMC condensin I complex subunit G (NCAPG) and immune cell infiltration in kidney renal clear cell carcinoma (KIRC).

KIRP, LGG, LIHC, PRAD, READ, and SKCM. Cai et al[7] summarized the abnormal higher expression and promoting role in various cancers including hepatocellular carcinoma, PC, BC, GC, glioma, lung cancer, CRC, OV, and endometrial cancer. It was worth noting that NCAPG expression was lower in LAML compared with the normal group in our study. Intriguingly, there was a study, supporting that NCAPG expression was abnormally inhibited in LAML.[26] In addition to lower expression of NCAPG in LAML, our data exhibited that NCAPG expression was overexpressed in the remaining 32 tumor types, which was also supported by IHC results from the HPA database in some cancer types.

Corresponding to the abnormal expression of NCAPG in pan-cancer, growing evidence recommended NCAPG could act as a prognostic marker in multicancers, such as GC, nonsmall cell lung carcinoma, and OV, in which higher NCAPG expression had a positive relationship with unwanted prognosis in these cancer types.[11,16,27] In our study, we explored the prognostic value of NCAPG in 33 tumor species, supporting NCAPG as a tumor diagnostic marker for the vast majority of tumors. Furthermore, lower NCAPG expression could achieve a favorable prognosis in various cancers.

There was evidence suggesting NCAPG participated in immune cell infiltration to affect cancer progression, such as glioma and STAD.[28,29] We also explored the relationship between NCAPG expression and immunity. The results suggested that NCAPG plays a key role in cancer immunity of pan-cancer. In general, tumor microenvironment features were used as markers to assess tumor cell response to immunotherapy and influence clinical outcomes.[30,31] As previously documented, different tumor-infiltrating immune cells could inhibit or promote the development of tumors.[32,33] However, the influence mechanism of tumor microenvironment on tumors is complicated. In our study, NCAPG was mainly positively related to $CD4^+$T and myeloid-derived suppressor cells and was positively or negatively related to other immune cells. In addition, as for immune-related molecules, NCAPG expression was positively related to Act CD4 and Th2 infiltration but negatively related to most other immune cells' infiltration in 33 cancers. Overall, NCAPG was probably involved in the progression of pan-cancer by influencing immune cell infiltration and expression of immune molecules.

Notably, our results displayed that NCAPG expression was negatively connected with immune inhibitors in most cancers; however, a strongly positive correlation was found in KIRC and THCA. Further data displayed that in KIRC, NCAPG expression was positively related with Act_CD4, Th2, memory B cells, activated CD8 T cells, and myeloid suppressor cells and, in THCA, was positively related with Act_CD4, Th2, Tgd, Tem_CD4, and NKT. With particular attention, our results exhibited, in KIRC, enrichment of most immune cells' including basophils, B cells, $CD4^+$ memory T cells, $CD8^+$ T cells, macrophages, mesenchymal stem cells, NK T cells, type 1 T-helper cells, and type 2 T-helper cells, while the decrease in eosinophils, regulatory T cells, and type 1 T-helper cells resulted in higher NCAPG expression-generated poor prognosis. More interestingly, the enrichment and reduction of type 1 T-helper cells exacerbated the poor prognosis of KIRC. Currently, some previous studies reported that immune cell infiltration was closely related to KIRC progression.[34,35] However, there are no experimental studies to explore the further influence of NCAPG on KIRC by regulating immune cell infiltration, which provides the direction for our research. Regrettably, we failed to acquire similar results in KIRC. Collectively, we strongly suggested that NCAPG expression was strongly related to immunoregulation in KIRC, which merits further investigation in the future.

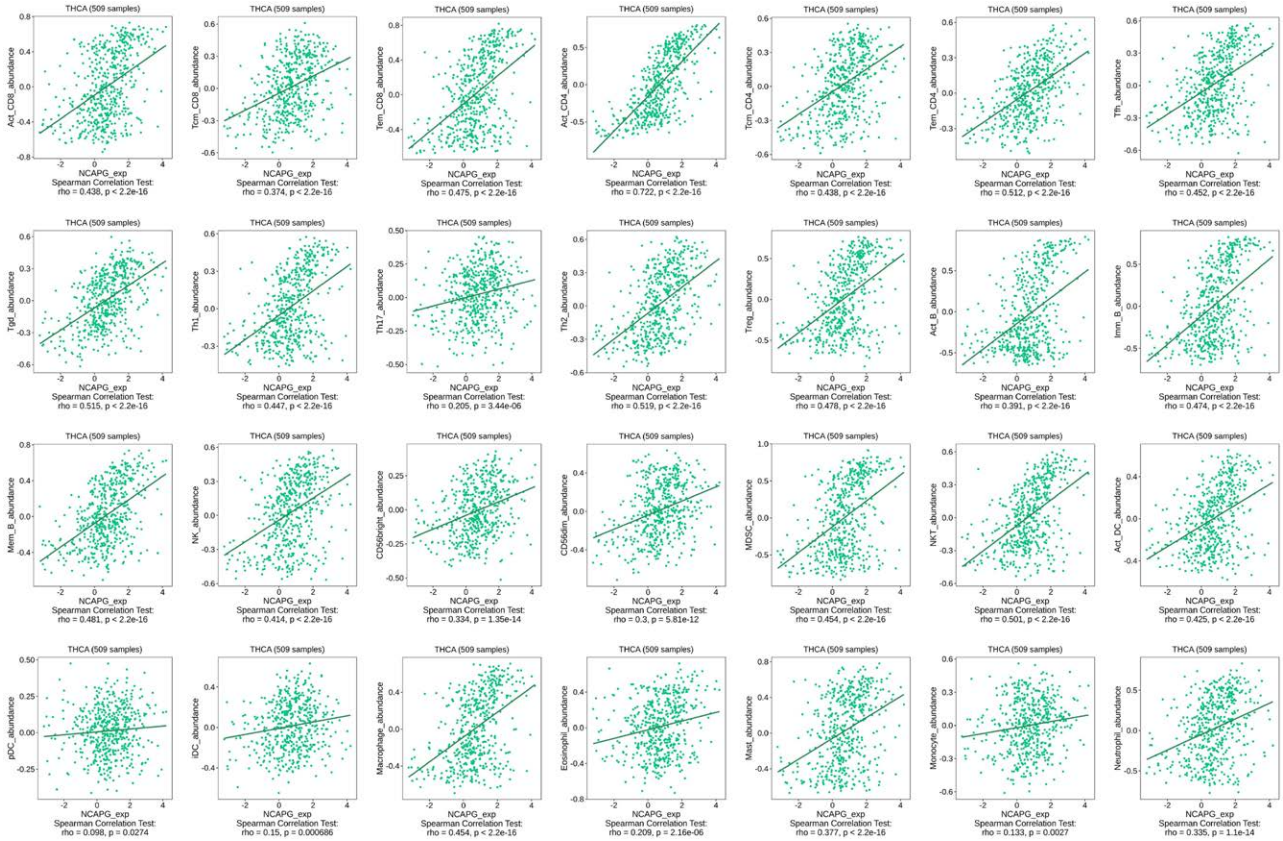


**Figure 12.** Spearman correlations between expression (exp) of non-SMC condensin I complex subunit G (NCAPG) and immune cell infiltration in thyroid carcinoma (THCA).

In addition, we investigated the interacted molecules of NCAPG by searching STRING, GeneMANIA, and GPS-Prot databases. Cyclin-dependent kinase 1, non-SMC condensin I complex subunit D2, non-SMC condensin I complex subunit H, structural maintenance of chromosome 2, and structural maintenance of chromosome 4 were predicted as interacted molecules of NCAPG, and these molecules were reported to exert a promoting influence on different cancer types.[36–40] In the future, the interactions of NCAPG and other 5 molecules need to be validated by conducting experiments. NCAPG has been widely investigated for various types of diseases including cancers and idiopathic pulmonary hypertension.[7,41] Noteworthy, research on NCAPG in the field of diseases has focused on the study of various tumors. Our results found that NCAPG was correlated with 132 diseases and had intimate relationships with cancers, suggesting that researchers could probe the role of NCAPG in other types of diseases besides tumors to explore the value of NCAPG in other diseases. Furthermore, the correlations between NCAPG and 14 cancer functional states were analyzed, and the data indicated that NCAPG mainly mediated cell cycle, DNA repair and damage, proliferation, and invasion to be involved in most tumors. The influences of NCAPG on cell cycle, DNA repair and damage, proliferation, and invasion in cancers were supported by published studies.[42,43]

The current study has several limitations. First, the data were all from open-access databases, which inevitably introduced the heterogeneity of different study samples in different databases, resulting in system bias. Second, some results of data mining could be supported by published literature, but many were still lacking substantive experimental evidence and needed to be verified by experiments. Third, the correlation between NCAPG expression and human tumor prognosis and immune cell infiltration was complex and lacked direct evidence.

In summary, this study comprehensively analyzed the expression of NCAPG in pan-cancer, revealing the correlation between NCAPG expression and clinical prognosis, immune cell infiltration, expression of immune-related molecules, and interacting molecules. This study provided insight into the potential role of NCAPG as a prognostic indicator in tumors through a comprehensive analysis of NCAPG in various cancer subtypes. From our results, we suggested that NCAPG played a nonnegligible role in a variety of cancers. Moreover, our bioinformatics analysis showed that NCAPG participated in the biological activities of cell cycle, DNA repair and damage, proliferation, invasion, and other biological activities of various tumors. At the same time, NCAPG was abnormally high expressed in most tumor types and led to poor prognosis in a variety of tumors. NCAPG might be used as a diagnostic marker for a variety of tumors. Moreover, the expression of NCAPG was closely related to the infiltration of multiple immune cells in the tumor microenvironment in various tumors, especially in KIRC and THCA. Our results suggested that NCAPG played an essential role in pan-cancer, and its exact role in a variety of tumors will be further confirmed by more cell studies, animal studies, and clinical trials. It is hoped that one day, NCAPG can be developed into a diagnostic marker for a variety of tumors, and drugs targeting NCAPG can be developed for tumor therapy.

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## Author contributions

**Conceptualization:** Min Fang.
**Formal analysis:** Min Fang, Zhu Wu, Zhi Xia, Jian Xiao.

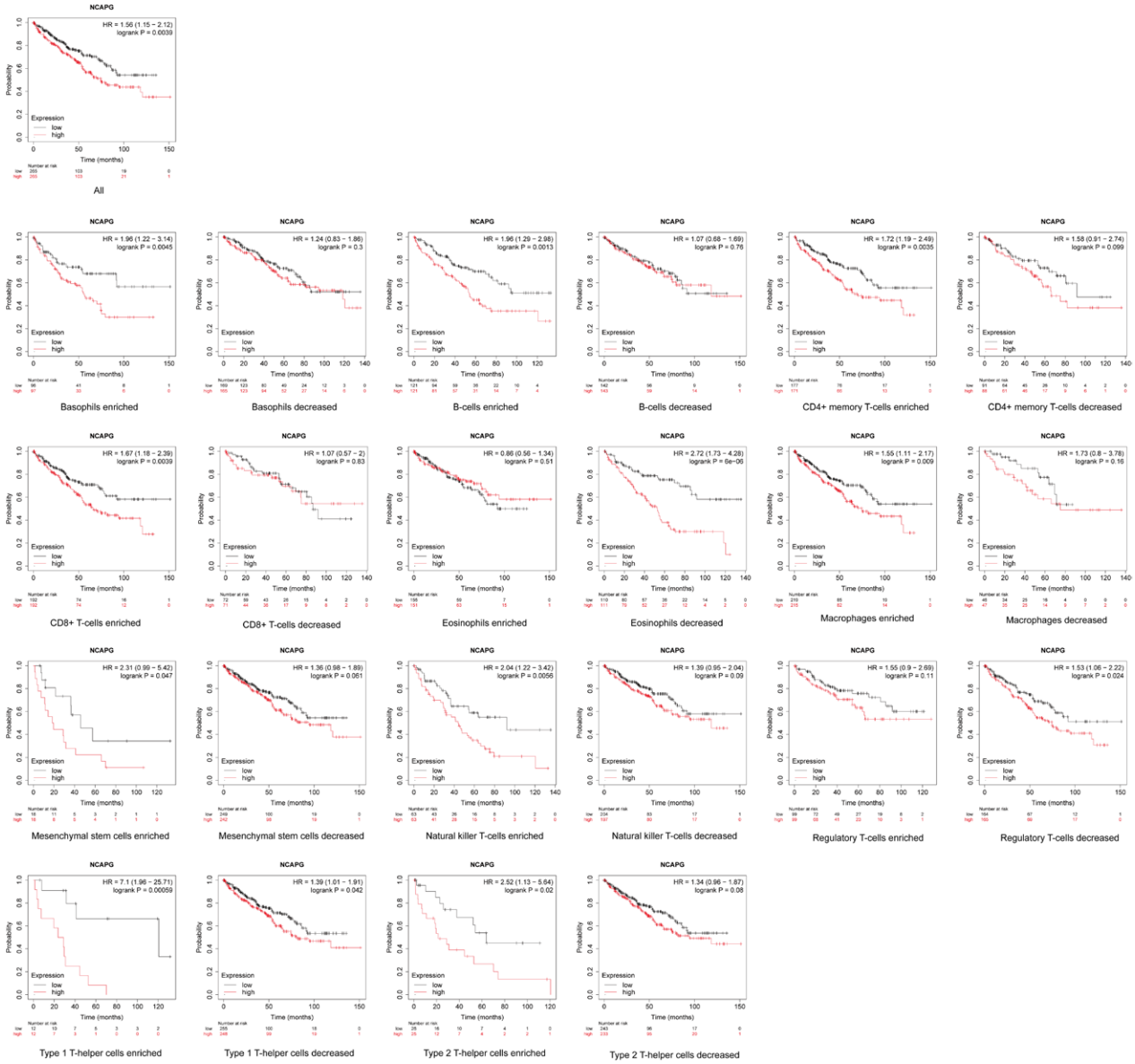


**Figure 13.** Effects of different immune cell infiltration on overall survival (OS) prognosis of kidney renal clear cell carcinoma (KIRC). NCAPG = non-SMC condensin I complex subunit G.

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**Methodology:** Min Fang, Zhu Wu, Zhi Xia.
**Software:** Min Fang, Zhi Xia, Jian Xiao.
**Validation:** Min Fang.
**Writing – original draft:** Min Fang.
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**Visualization:** Zhu Wu, Zhi Xia.
**Writing – review & editing:** Zhu Wu, Zhi Xia, Jian Xiao.
**Funding acquisition:** Jian Xiao.
**Project administration:** Jian Xiao.
**Resources:** Jian Xiao.

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