Pan-cancer Analysis Reveals the Small Nuclear Ribonucleoprotein Polypeptide C (SNRPC) Its Significance in Human Cancers

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Abstract

Background

SNRPC is cloned on human chromosome 6p21.31, which encodes a special protein component of U1 snRNP granules. Although SNRPC played an important role in the pre-mRNA splicing starting and adjustment, but in the tumor biological function is still unknown.

Method

Through the pan-cancer analysis of SNRPC, and our data sets, phosphorylation and functional network analysis based on TCGA (Cancer Genome Map) and GEO (Integrated Gene Expression Database), also from the western blot, qRT-PCR and CCK-8, cloning-forming experiment, scratch experiment to prove SNRPC biological function.

Results

SNRPC is related to the regulation of RNA, shear, and protease signals and has an important effect on ovarian cancer prognosis. Through a series of biological information data mining and basic experiments, we found that SNRPC plays an important role in the proliferation and migration of ovarian cancer. SNRPC expression is positively correlated with the immersion of $CD4^{+}$T cells, macrophages, and neutrophils ($p< 0.05$), as obtained through the TIMER database (Tumor Immunological Assessment Resources) database.

Conclusion

Our pan-cancer research provides SNRPC in different tumors, especially the relatively comprehensive understanding of the carcinogenic potential of ovarian cancer.

Background

Cancer is one of the leading causes of death worldwide and is becoming increasingly young [1]. As a result of the growth and aging of the global population, the number of cancer patients continues to expand [2]. This has led many researchers to devote much of their efforts to cancer prevention, early diagnosis, and treatment to reduce the burden of disease [3]. But the sudden new crown outbreak has hampered research into the diagnosis and treatment of cancer. Therefore, we should speed up the study of the occurrence and development of cancer [4]. Given the complexity of tumor occurrence, the process of studying the carcinogenic effect is a top priority, and we intend to analyze the pan-cancer expression of meaningful genes and evaluate their molecular mechanisms associated with survival prognosis and related.

SNRPC,small nuclear ribonucleoprotein polypeptide C is cloned on human chromosome 6p21.31, which encodes a special protein component of U1 snRNP granules [5]. It plays a significant role in the initiation and adjusting of pre-mRNA splicing [6]. Sequence alignment among SNRPC members showed that the first 40 amino acids were highly conserved, however, the C-side sequence is very different [7]. Studies have shown that SNRPC is related to the gender of systemic autoimmune diseases. SNRPC takes part in the pathogenesis of spinal muscular atrophy by regulating the alternative splicing of coactivator-associated arginine methyltransferase [8]. However, SNRPC in cancer biology effect and the mechanism is still unclear., which will become the focus of our exploration.

Our research is to analyze the pan cancerization of SNRPC using TCGA and GEO databases. Our research includes a series of terms, such as gene expression, survival prognosis, gene mutation, protein phosphorylation levels, immune infiltration, and related cellular pathways, to dig SNRPC at different levels of cancer or potential molecular mechanisms and clinical prognosis of immune. In the course of the study, we found that SNRPC is highly expressed in ovarian cancer, and may be involved in inhibiting the movement of ovarian cancer cells.

Methods

The level of gene expression

The analysis of TIMER2 (http://timer.comp-genomics.org/) is the expression matrix data of gene TCGA, in the GENE option, we can study the Spearman correlation between single genes expression and different immune cell immersion levels [9]. You can use TIMER's Gene_DE options to observe differences in SNRPC expression in specific tumor subtype TCGA projects in different tumors or tumors and adjacent normal tissues. Using the "Box Plots" module of the GEPIA2 (http://gepia2.cancer-PKU.cn/), We obtained differences in the expression of molecules between these tumor tissues and the corresponding normal tissue, with the P-value cut-off of 0.01, log2FC (folding change) cut-off of 1 and the setting of "Matching TCGA normal and GTEx data", setting by p-value Cutoff = 0.01, |Log2FC| Cutoff =1 [10]. Through GEPIA2's Pathological Staging Map module, we obtained violin diagrams of SNRPC expressions for all tumors of TCGA at different pathological stages (□,□□, and □). The expression data of the conversion of the log2 [TPM (one in a million transcripts) is applied to the block or violin plots.

Clinical Proteomic Tumor Analysis Consortium (CPTAC) and UALCAN

The UALCAN portal, a comprehensive analysis of the interactive web resource database of cancer, allows us to perform protein expression analysis of CPTAC (Clinical Oncology Proteomics Analysis Consortium) data sets. Here, we explore the total protein expression level or phosphorus protein between SNRPC primary tumors and normal tissue, by entering "SNRPC." Five alternative data set options for tumors, including breast cancer, colon cancer, ovarian cancer, UCEC (endometrial cancer in the uterine corpus), and RCC (renal cell carcinoma).

We also explored the protein expression of SNRPC in pots through the UALCAN cancer subtype. Mass-spectrometry-based CPTAC Confirmatory/Discovery total protein found that legions were used to classify 532 cases (representing five tissue cancer types) to ten different pan-cancer cancer subtypes (K1 to K10) [11].

Survival curves analysis

We used GEPIA2 database module survival curves to study SNRPC molecules in the different tumor prognoses. We could get a high and low expression SNRPC influence on the survival prognosis of the different tumors, and limit P<,0.05 the conditions it is concluded that there is a statistically significant prognostic curve. Or verify with the Kaplan Meier plotter (https://kmplot.com/analysis/).

Evaluation of tumor-infiltrating immune cells

TIMER (https://cistrome.shinyapps.io/timer/) is a comprehensive online database for systematic analysis of various types of cancer immune immersion. In this study, we conducted TIMER to determine the relationship between SNRPC expression in ovarian cancer and six immune infiltrates (B cells, $CD4^+$T cells, $CD8^+$T cells, neutrophils, macrophages, and dendritic cells) [12].

We used TIMER's "Immune Genes" module to research the link among SNRPC expression with all different tumor immunity infiltration. Explore the potential relationship between the immersion levels of different immune cells and the levels of SNRPC gene for different cancer kinds of TCGA.

The genetic variation analysis

By using the cBioPortal website (http://www.cbioportal.org/) [13, 14], we could study "SNRPC" genetic variation characteristics of the gene, The frequency of change, copy number change, and mutation type results of various tumors was observed in the Cancer Type Summary module. To see this website is interesting SNRPC gene mutation of protein structure diagram and three-dimensional structure, this could be more image observed SNRPC the gene research direction. We also compared the statistics of the prognosis of cancer cases have SNRPC genetic changes. Then it shows some information about SNRPC specific to ovarian cancer, such as mutation/survival/co-expression, and produces Kaplan-Meier plots with a peer rank P-value.

STRING: functional protein association networks

STRING (https://www.string-db.org/) could be used to predict protein function each other websites, including the direct and indirect correlation. We first searched and correlated with SNRPC expression genes, take before 50 genes. In "Settings" the set needs to limit conditions, then click "update" proteins related to the function of the network could be calculated out. Finally, the SNRPC binding protein used for experimental determination was obtained.

Correlation between genetic

We use GEPIA2 "a similar genetic testing module, based on all the TCGA data sets, tumor cells, and normal tissues before 100 targeted SNRPC related genes. We also use the GEPIA2 module for SNRPC "correlation analysis" and the selected genes were paired with Pearson correlation analysis. The log2 TPM is suitable for the figure. The P-value and correlation coefficient are pointed out. Venn diagram (http://bioinformatics.PSB.ugent.be/web tools/Venn/) is used to show things in different groups (set) the mathematical or logical link between, especially suitable for used to denote the set (or) the general relationship between the classes [15, 16]. We used cross analysis to compare SNRPC combination and the interaction of genes.

Gene set enrichment analysis

We combined data for KEGG (https://www.genome.jp/kegg/kegg2.html) path analysis. We then uploaded the list of genes to DAVID [17]. The "tidy" and "ggplot2" R packages eventually show the rich path. Furthermore, we used the "cluster profile" R package for GO Journal pre-proof Journal pre-proof (gene ontology) enrichment analysis. R-language software (https://www.r-project.org) is used in the analysis. $P<0.05$ is considered statistically significant.

Cell Lines and Cell Culture

The normal epithelial ovarian cancer cell line OVCRA3 purchase from the American Type Culture Collection (ATCC) (https://www.atcc.org/). OVCRA3 cells are cultured in RPMI-1640 (Biological Industries 01-100-1B) with 10% FBS (Biological Industries #1928703). All cells are cultured at 37℃ and 5% CO2.

SNRPC knockdown and construction of stably transfected cell line

SNRPC Scarmble and SNRPC shRNA were designed by Sigma online database (https://www.sigmaaldrich.com/china-mainland/zh/life-science/functional-genomics-and-rnai/shrna/individual-genes.html) of validated shRNA. shRNAs (1 μg) were subcloned between the BamHI and NotI site of the pGreenPuro shRNA Expression Lentivector (System Biosciences, SI505A-1). OVCAR3 cells were transfected the plasmid using the jetPRIME Transfection Reagent (101000046, Polyplus) following the manufacturer's protocol and 48 h after transfection, cells were treated with puromycin dihydrochloride (sc-108071, santa cruz) for selection of stably transfected cells.

The sequence of SNRPC shRNA is SNRPC-F: gatccCTGGATGATGATGATAATGAATTCAAGAGATTCATTATCATCATCATCCAGttttttg, SNRPC-R: aattcaaaaaCTGGATGATGATGATAATGAATCTCTTGAATTCATTATCATCATCATCCAGg. The sequence of the scramble is scramble-F: gatccCTGGCATCGGTGTGGATGATTCAAGAGATCATCCACACCGATGCCAGttttttg, scramble-R: aattcaaaaaCTGGCATCGGTGTGGATGATCTCTTGAATCATCCACACCGATGCCAGg.

Western blotting analysis

Protein were extracted from stably transfected OVCAR3 cell in both SNRPC Scramble and SNRPC shRNA groups, with a strong lysate RIPA cantaining protease inhibitor and phosphatase inhibitor. sample proteins (40 μg) are subjected to 10% of the SDS polyacrylamide gel for electrophoresis and transferred to the PVDF membrane. After blocking with 5% non-fat milk for 1 h at room temperature, the membranes were incubated with anti-SNRPC (1:1000, AP2841b, Abcepta) and anti-GAPDH (1:1000, RK-200-301-A33, Rockland) antibodies at 4°C overnight. The membranes were washed with TBST for three times

and incubated with HRP-conjugated anti-rabbit or anti-mouse secondary antibodies (1:5000, #7074, #7076, CST) for 1 h at room temperature. Blots were displayed using an enhanced chemiluminescence kit (WBULS0500, Millipore).

RNA Extraction and Quantitative Reverse Transcription Polymerase Chain Reaction

Trizol liquid (B511311, Sangon Biotech) was used for extracting the total RNA. cDNA were synthesized using HiScript III 1st strand cDNA synthesis Kit (+gDNA wiper) (R312, Vazyme). qPCR was performed on a StepOne/StepOnePlus Real-Time PCR Systems (Applied Biosystems, ThermoFisher), using SYBR-Green (Q711, Vazyme). The thermocycling conditions were as follows: 40 Cycles of denaturation at 94˚C for 20 sec, annealing at 61˚C for 13 sec and extension at 72˚C for 15 sec. The relative amount of transcripts was calculated using the $2^{-\Delta\Delta Cq}$ method. The following is the primer sequence: SNRPC (forward primer: TGTGACTACTGCGATACATACCT; reserved primer: GCCTGCTCTTCCATCCATTTCT); GAPDH (forward primer: CTGGGCTACACTGAGCACC; reserved primer: AAGTGGTCGTTGAGGGCAATG). GAPDH was used as an internal control.

Cell Proliferation Assay

Two stably transfected OVCAR3 cell (2000/well) were cultured in a 96-well plate for 24 hours and, using cell count kit 8 (CCK-8) to observe the survival of cells in 0 h, 24 h, 48 h, 72 h, 96 h, respectively [18]. For clonogenic assays, a 6-well plate containing 150 stably transfected OVCAR3 cell line (SNRPC shRNA and scramble) were incubated for 14 d. Colonies formed were fixed with 4% polyformaldehyde, stained with 0.5% crystalline purple. Plates were took pictures.

Detection of cell migration ability with a scratch test

In wound healing cell migration assay, two stably transfected OVCAR3 cell were cultured in low FBS (≤2%) media and incubated for 72 h to monitor wound closing after being scratched with a 100 μl pipette. Then wounded monolayer cells were washed three times with PBS and culture it continuously with a medium. Cells were photographed at 0, 24, 48, and 72 hours and cell growth were recorded at different times at the same site [19].

Statistical analysis

Data were presented as Mean ± standard error and statistically analyzed using Student's *t*-test, ANOVA. All statistical calculations were performed using GraphPad Prism 7. A p value of<0.05 was determined to considered a significant difference.

Results

The expression pattern of SNRPC from the perspective of pan-cancer

To assess mRNA expression patterns in different cancer types SNRPC, we collected about 30 cancer types from TIMER2. As shown in Figure 1a, there was a significant increase in SNRPC in 12 cancer types of all cancer types compared to normal tissue. These data show that SNRPC gene expression is abnormally expressed in different cancer types (Fig. 1a) For the TIMER2 database, because some cancer species do not have normal tissue, the remaining cancer species are supplemented by the GEPIA2 database (Fig. 1b).

The UALCAN portal, a friendly, well-known analysis of tumor histologic network database, permits us to perform the expression of protein analysis by using the CPTAC (Clinical Proteomic Tumor Analysis Alliance) dataset. Total protein expression level or phosphorus protein among primary tumors with normal tissue was analyzed with UALCAN. Five available data sets for tumors were accessible, including breast cancer, ovarian cancer, colon cancer, RCC (renal cell carcinoma), and UCEC (endometrial cancer in the uterine corpus). Using the UCLAN database, SNRPC was classified into 10 different canceling subtypes (K1 to K10) with mass spectrometry-based protein data from the CPTAC confirmation/discovery group (Fig. 1c).

Through GEPIA2's Pathological Staging Map module, we obtained violin diagrams expressed by SNRPC for all corresponding pathological stages (□,□□ and □) of all TCGA tumors. The expression data of the conversion of the log2 [TPM (Transcripts per million) +1] is used to the block or violin diagram (Fig. 1d).

Prognosis survival curve

Through GEPIA2 database website data, we analyze the survival prognosis of tumor-associated with SNRPC model, including the OS (overall survival) and DFS (disease-free survival), including BRCA, ESCA, KICH, LGC, LIHC, LUSC, OVC, and SARC. The survival prognosis of GEPIA2 module could get we want prognosis curve directly, only we needed to set up the corresponding parameters. The higher the prognosis of SNRPC expression in ovarian and LUSC, the better, as opposed to ESCA, LGC, and KICH (Fig. 1e-f).

To explore the TCGA contains the genetic variation of different tumor types SNRPC

We could observe SNRPC genetic changes in the tumor samples of different TCGA. As shown in Figure 3a, the most of change in SNRPC (>5%) occurs in patients with "mutation" as the main type of uterine tumor. CNA "amplification" is the main type of uterine tumor case, with a frequency of about 3.5% change (Fig. 2a). What was paid attention to was that all genetically modified bile duct cancers, ovarian cancers, and large b-cell lymphomas have "amplification" (Fig. 2a). As shown in figure 2b, we could observe SNRPC type, size, and genetic change in the number of cases. We observed that the wrong mutation of SNRPC is the core type of genetic change, then X84_splice changes detected in these cases (Fig. 2b) can induce splicing mutations in the SNRPC gene. On the other side, we studied the link between SNRPC related genetic changes and clinical survival prognosis for different types of cancer patients. Figure 2c shows that SNRPC-altered ovarian cancer cases cannot showed a better prognosis overall (P=0.516) and had no special significance in disease-free (P=0.550) survival compared to cases without SNRPC change.

Compare the differences of SNRPC phosphorylation levels in normal tissue and tumor tissue

We compared the SNRPC phosphorylation level differences in various tumors, by comparing the normal and primary tumor tissue. Using the CPTAC data set in UCLAN, breast and ovarian cancer were analyzed, and the SNRPC phosphorylation sites and significant differences were summarized. Compared to normal tissue, the S17 gene base in the ZF-U1 domain of SNRPC showed higher levels of phosphorylation in all primary tumor tissues (Fig. 2d-e, P< 0.05), and the phosphate level of the gene base increased. This observation is worth further molecular testing to mine the potential role of S17 phosphorylation in tumor development.

The potential relationship among the immersion levels of different immune cells with the expression of the SNRPC gene of different cancer types of TCGA

Immersion of tumor immune cells as an important part of the tumor microenvironment, and cancer closely related to the occurrence and development of or transfer [20–22]. We found that using the TIDE algorithm in the database, SNRPC was found to be related to the inhibition of immune cells from myelin sources of many tumors. We also used the TIMER database to explore the potential connection among the inundation levels of different immune cells with the expression of the SNRPC gene in various cancers. After a series of studies, we found that in all or most TCGA tumors of LGG and KIGH, the expression of SNRPC was statistically actively related with the estimated immersion values of cancer-related fibroblasts, but negatively correlated (Fig. 2f-g) in BRCA, LUSC, OVC, STAD, TGCT, and THYM.

Functional enrichment analysis

To explore the proteins and expression-related genes of SNRPC binding, the molecular mechanism of SNPRC in tumor development was studied through enrichment analysis. First, using the STRING database, 50 genes associated with SNRPC

expression were obtained. (Fig. 3a) the first 100 SNRPC expression-related genes were obtained through GEPIA2, the first six genes were taken, and the correlation was analyzed using the "correlation analysis" module (Fig. 3b). The expression of six target genes in each tumor is then demonstrated by a heat map. Six target genes were LSM2, ABT1, CSNK2B, NELFE, KIFC1, TUBB. As shown in the figure, SNRPC is positively correlated with the six target genes mentioned above in most tumors. (Fig. 3c) The two data sets were intersected by the Venn graph, and three common genes SNRPG, SNRPD1, and SNRPB were found in the two groups. (Fig. 3d)

KEGG path enrichment analysis is the strength of *P*-values expressed by the size of bubbles, and SNRPC is mainly involved in RNA splicing and mRNA processing. At the same time, KEGG enrichment analysis determined that these genes were involved in the regulation of mRNA, snRNA, and miRNA binding. (Fig. 3e)

SNRPC inhibits the proliferation and migration of ovarian cancer cells

To further investigate the mechanism of SNRPC's biological functions in ovarian cancer, loss-of-function experiments were performed. OVCAR3 cells transfected with an shRNA against SNRPC was significantly low compared with OVCAR3 cells transfected with a scrambles shRNA in the level of RNA (Fig. 4a) and protein (Fig. 4b) expression. We explored tumor biological function of SNRPC through CCK8, Colony-forming assays, and scratch experiment. The analysis of CCK-8 showed that low expression of SNRPC significantly promoted the proliferation of OVCAR3 cells (Fig. 4c). Colony-forming assays showed that proliferation of SNRPC shRNA cell lines was severely increased (Fig. 4d). Scratch tests detect cell migration, as shown in Fig. 4e, low expression of SNRPC can be found to promote cell migration (Fig. 4e). These results indicated that SNRPC may inhibit tumor proliferation and migration of ovarian cancer.

Relationship between SNRPC expression and TIICs

To study the influence of related genes on tumor prognosis, it is essential to study the immune infiltration of genes [23]. An analysis of the relationship between SNRPC and TIICs in the TIMER database shows that SNRPC expression is positively correlated with CD4+T cells (r=0.098, *P*=3.14e-02), macrophages (r=0.157, *P* =5.56e-04), and neutrophils (r=0.207, *P*=4.78e-06) (Fig. 5a). At the same time, the expression of SNRPC was negatively correlated with T cells (r=-0.045, *P*=0.388), NK cells (r=-0.248, *P*<0.001), $CD8^+$T cells (r=-0.046, *P*=0.373). Instead, the expression of SNRPC is positively correlated with TFH (r =0.049, P =0.338) and Th2 cells (r = 0.130, P=0.011) (Fig. 5b) (table1).

Discussion

Small ribonucleic C peptide (SNRPC), also known as shear body U1C protein, is essential for the start-up and regulation of mRNA pre-cut [24, 25]. In our study, SNRPC was found to be closely related to ovarian cancer. Nevertheless, the function of SNRPC in tumors has not been fully studied. But people never stop studying the molecular mechanisms of SNRPC in cancer. For example, snRNP peptide G is the other important component in the biological occurrence of shear body U snRNPs and plays a vital role in the development and the progress of tumors in the breast, lung, and colon cancer [26]. U1 snRNP particle is the richest of whole snRNP species and plays a significant role in mRNA pre-splicing [27]. In the study of SNRPC enrichment analysis, it was found that SNRPC and RNA splicing play an important role. Some proteins are associated with SNRPC and participate in splicing regulation. For instance, transcription factors are a transcription factor the FLI agreed with another or nuclear receptor gene NOR1 associated, by contact with SNRPC involved in splicing regulation [28].

In studying the pan-cancer of the SNRPC gene, it was found that SNRPC is highly expressed on many tumors and has a strong association with tumor prognosis, but there is no actual clinical data to support this hypothesis. We focused on the effects of SNRPC on the proliferation and migration of ovarian cancer, consistent with the corresponding significance of the KM survival curve, our "homologous gene" and system development tree analysis data proved the conservative structure of SNRPC protein in different species, showing that SNRPC normal physiological effect may have parallel mechanisms. There is a functional connection among SNRPC with clinical diseases, in especial tumors. SNRPC can through some common molecular mechanisms in the pathogenesis of tumors to play a role, it remains to be answered [29]. Moreover, according to

TCGA, CPTAC, and GEO database data, and the change of gene expression, gene, DNA methylation, and molecular characteristics of protein phosphorylation, we thoroughly examined a total of 30 different SNRPC genes in tumors. Enrichment analysis showed that SNRPC is involved in the biological generation, the assembly and the tissue, RNA shearing, and mRNA processing of RNA complexes, consistent with SNRPC's speculative physiological function [30].

The latest success in the clinical practice of cancer immunotherapy makes it necessary to study the interaction among cancer cells with the host immune system [31–33]. Immune immersion of tumors is necessary to understand tumor-immune interaction, and it is also a hot topic in today's research [34, 35]. We use TIMER to analyze SNRPC expression and the correlation between immune cells infiltration. The results were represented that $CD4^{+}$T cells, macrophages, and neutrophils were more impregnated in highly expressed ovarian cancer tissue of SNRPC. This has a great effect on our study of SNRPC immune mechanism and in exploring the immune correlation.

Tumor proliferation and migration have a close connection with the survival prognosis of patients [36–38]. We study the effects of SNRPC on the proliferation and migration of ovarian cancer cells by down-regulated of SNRPC to study the prognosis. We found that low-expression SNRPC promotes the movement of OVCAR3 cells and also promotes proliferation. We can venture to assume that SNRPC high expression is a better sign of the prognosis of ovarian cancer.

Conclusion

Through the pan-cancer study of small nuclear ribonucleoprotein polypeptide C (SNRPC), we found that the SNRPC gene is highly expressed in most tumor tissues, discussed the relationship between SNRPC gene expression change and prognosis, and discussed the correlation between SNRPC and TIICs. In addition, SNRPC is involved in RNA splicing and mRNA processing and is involved in the regulation of mRNA, snRNA, and miRNA binding. And experiments show that in ovarian cancer, low-expression SNRPC promotes the movement of OVCAR3 cells and also promotes proliferation. The study suggests that SNRPC could be a potential new target for ovarian cancer treatment.

Abbreviations

BRCA: Breast invasive carcinoma; CCK-8: Cell counting kit-8; ESCA: Esophageal carcinoma; KICH: Kidney Chromophobe; LGC: **large gastric cancer;** LIHC: Liver hepatocellular carcinoma; LUSC: Lung squamous cell carcinoma; OV: Ovarian serous cystadenocarcinoma; PVDF: Polyvinylidene difluoride; SARC: Sarcoma; SNRPC: small nuclear ribonucleoprotein polypeptide C.

Declarations

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Authors' contributions

YJC, MHH had full access to all of the data in the study and takeresponsibility for the integrity of the data and the accuracy of the data analysis; YJC, JLC designed this study; YJC YWQ and KX collected all data and conducted research; YJC, MHH and JLC analyzed and interpreted data of this study; YJC, MHH and JLC drafted the manuscript; MHH got the funding for this study; YJC and MHH gave technical or logistic support for conducting this study; All authors agreed with the results and conclusions of this article. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on request.

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Table

Due to technical limitations, table 1 is only available as a download in the Supplemental Files section.

Figures

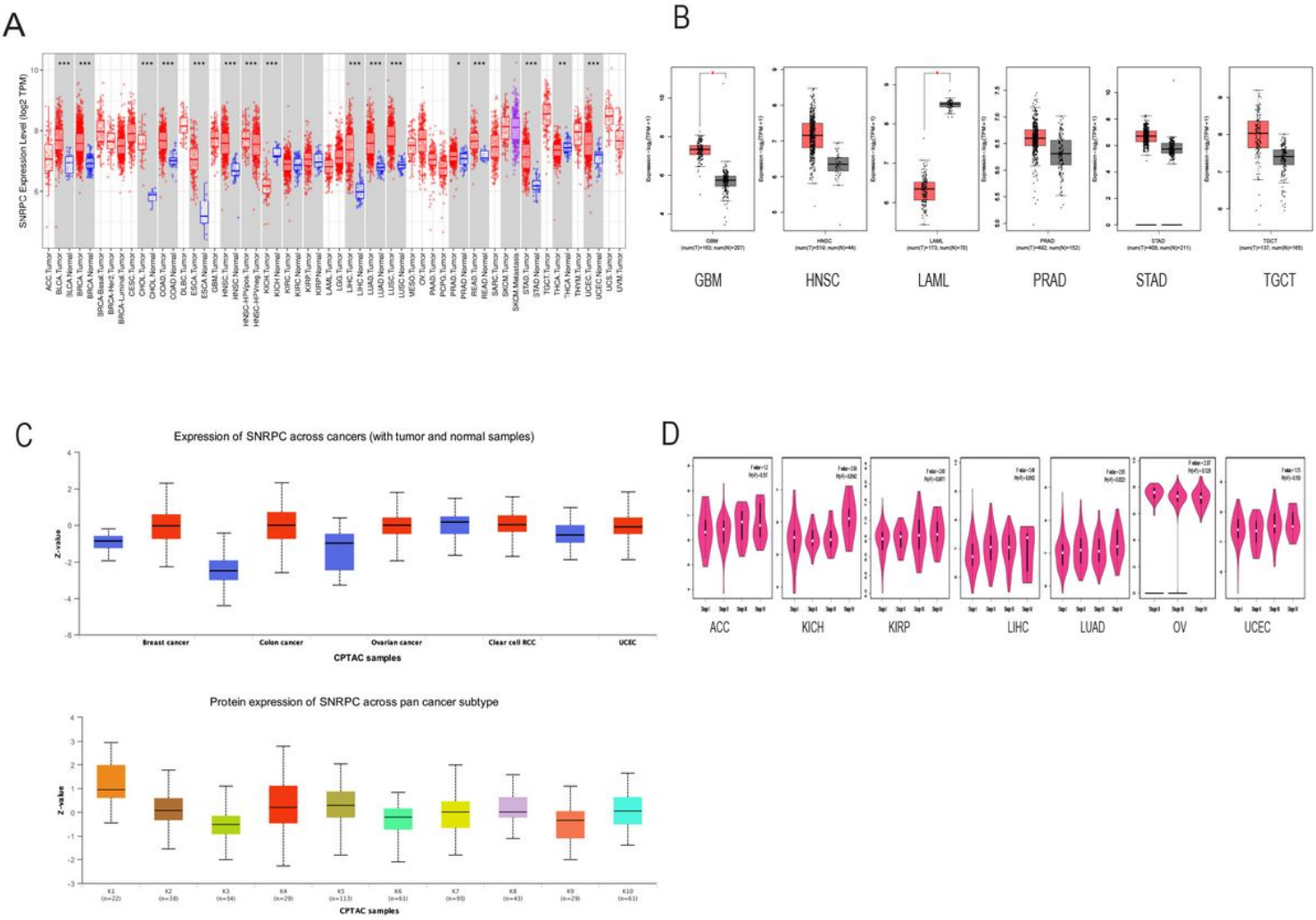

Figure 1

The expression pattern of SNRPC from the perspective of pan-cancer and analysis of tumor survival predictions associated with the SNRPC model a. Analysis of mRNA expression of SNRPC genes in different cancer or specific cancer subtypes. b. For the TIMER2 database because some cancer species do not have normal samples, so the use of GEPIA supplement ($p<0.01$). c. About the expression of SNRPC proteins in different cancers. d. The relationship between the expression of SNRPC and the clinical stage of different cancers was explored by the GEPIA database. e. Using GEPIA, SNRPC expression was analyzed separately and correlated with cancer Overall Survival. f. SNRPC expresses the relationship between high and low levels and the Disease Free Survival of cancer.

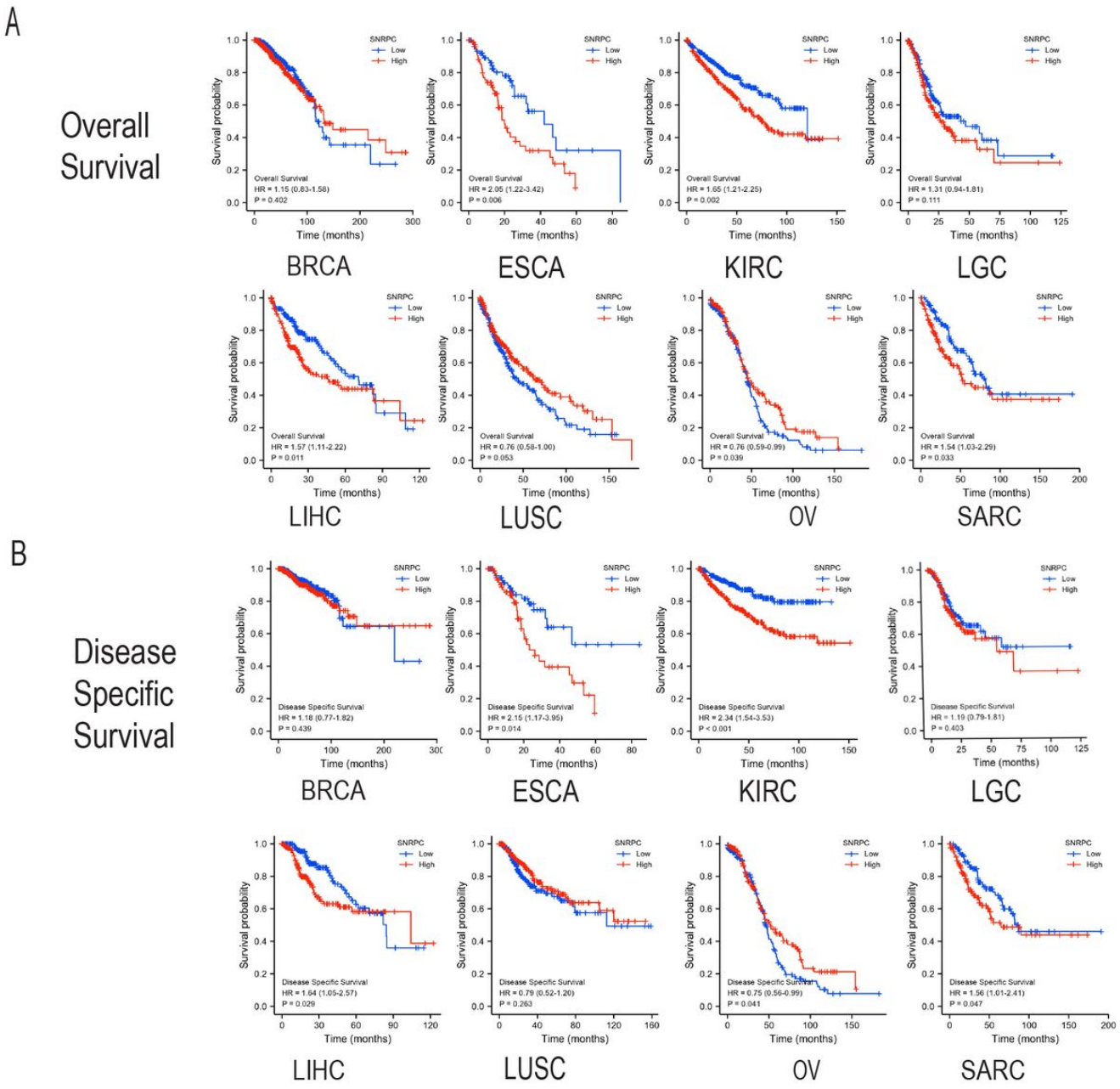

Figure 2

Explore TCGA containing the genetic variant SNRPC and protein phosphorylation levels for different tumor types. a. Mutations of SNRPC in 30 tumors. b. Specific mutations on each domain. (c): Relationship between SNRPC mutation and survival of ovarian cancer. d. Differences in phosphate levels in SNRPC in breast cancer. e. Differences in phosphorylation levels in ovarian cancer of SNRPC are provided, and protein expression levels at SNRPC phosphorylation sites are provided. f-g. Explore the potential relationship between the inundation levels of different immune cells and the level of SNRPC gene expression in different cancer types of TCGA.

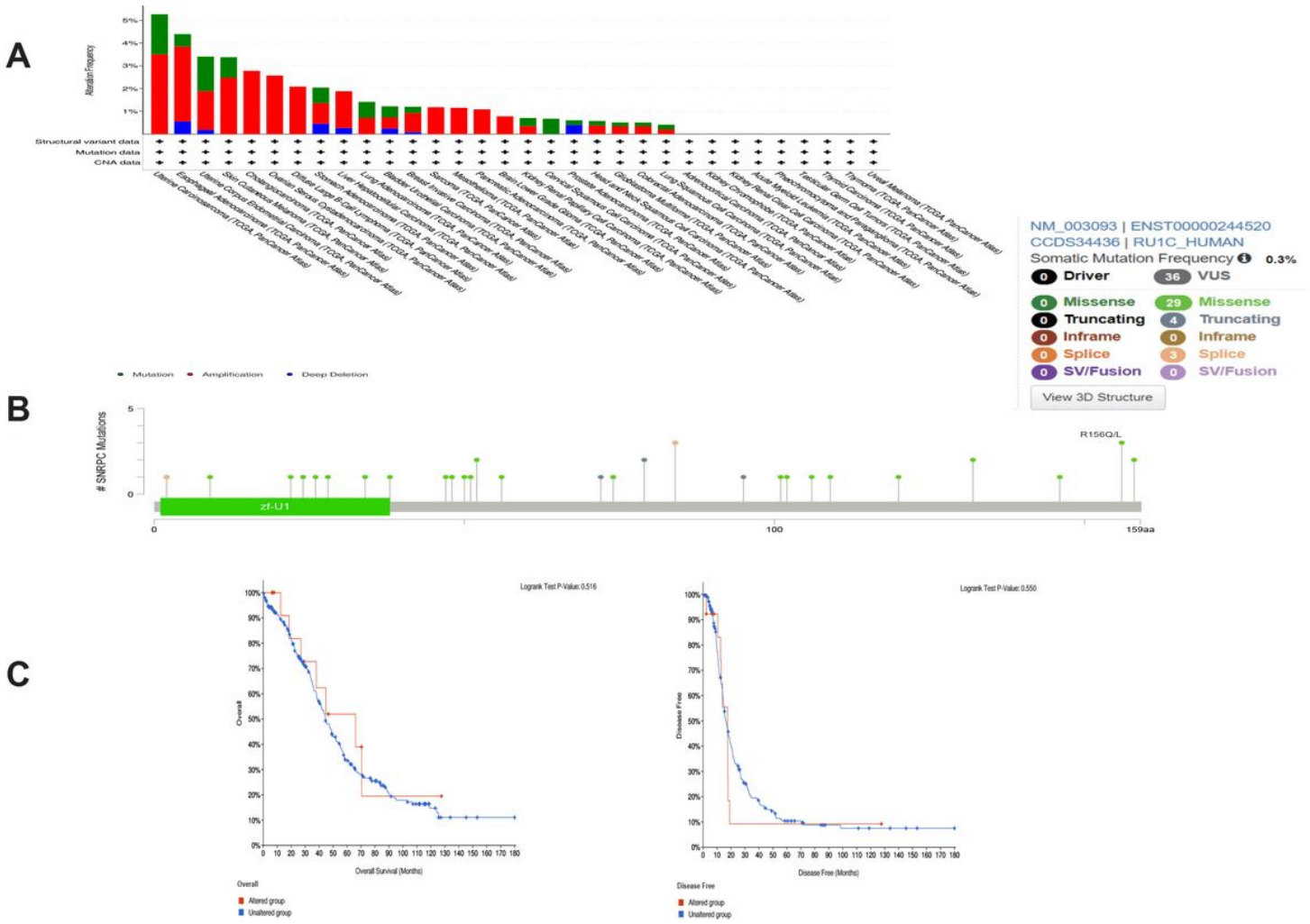

Figure 3

Explore genes associated with SNRPC binding proteins and SNRPC expression. a. Using the STRING database, 50 genes associated with SNRPC expression were obtained. b. The first 100 genes associated with SNRPC expression were obtained using GEPIA2, and the first five genes were taken. c. The expression of five target genes in each tumor was shown using heat maps. d. Using the Venn diagram, 50 proteins and 100 target genes were intersected. e. KEGG and GO analysis were done using proteins that bind or interact with SNRPC.

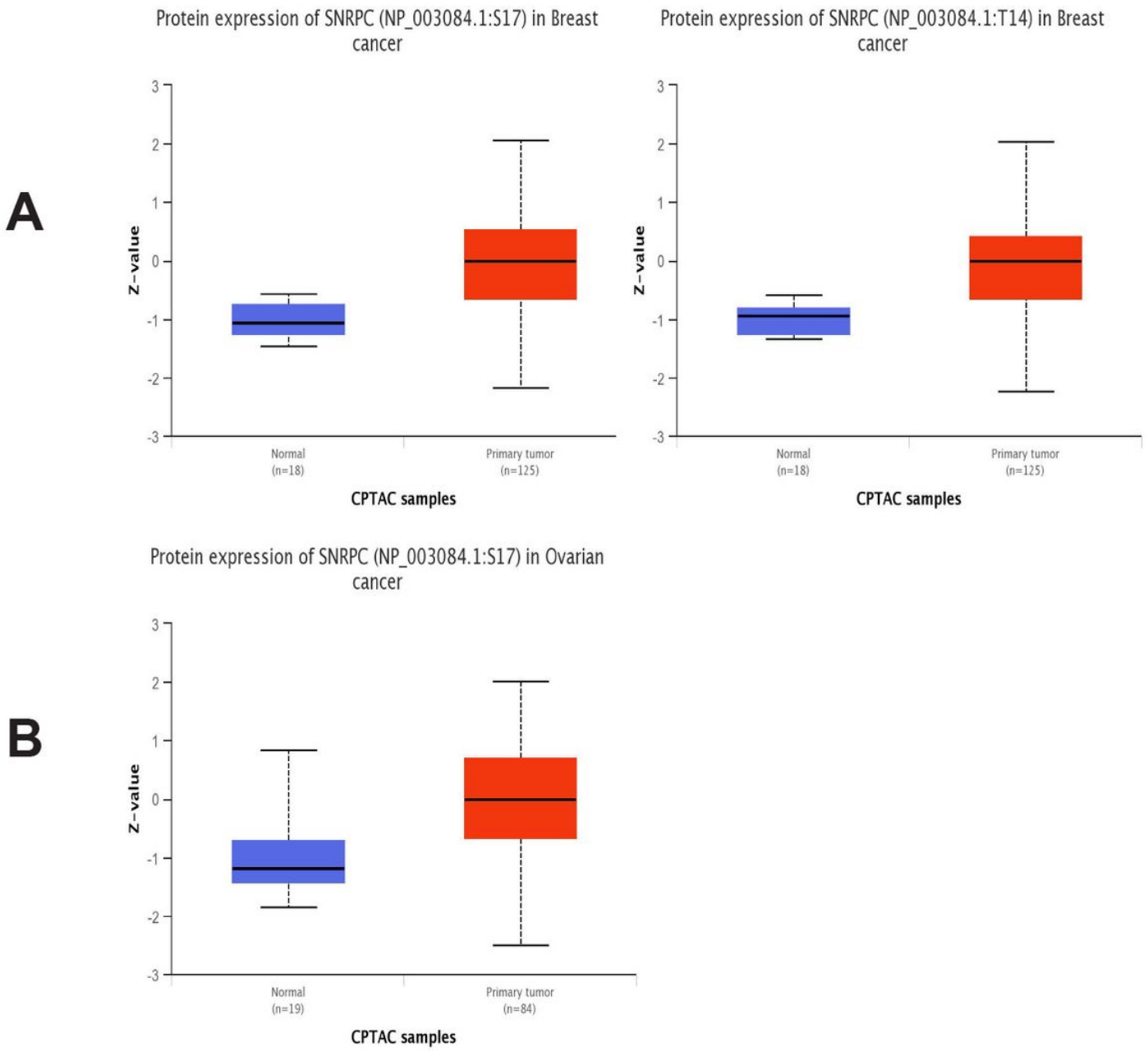

Figure 4

SNRPC inhibits the proliferation and migration of ovarian cancer cells. a. qRT-PCR to detect the shRNA mediated knockdown of SNRPC expression in OVCAR3 cells. b. Western blot to detect the shRNA mediated knockdown of SNRPC expression in OVCAR3 cells. c-d. Cell proliferation is assayed through CCK-8 and clonogenic assays. e. Scratch experiments assess cell migration capabilities at indicated times.

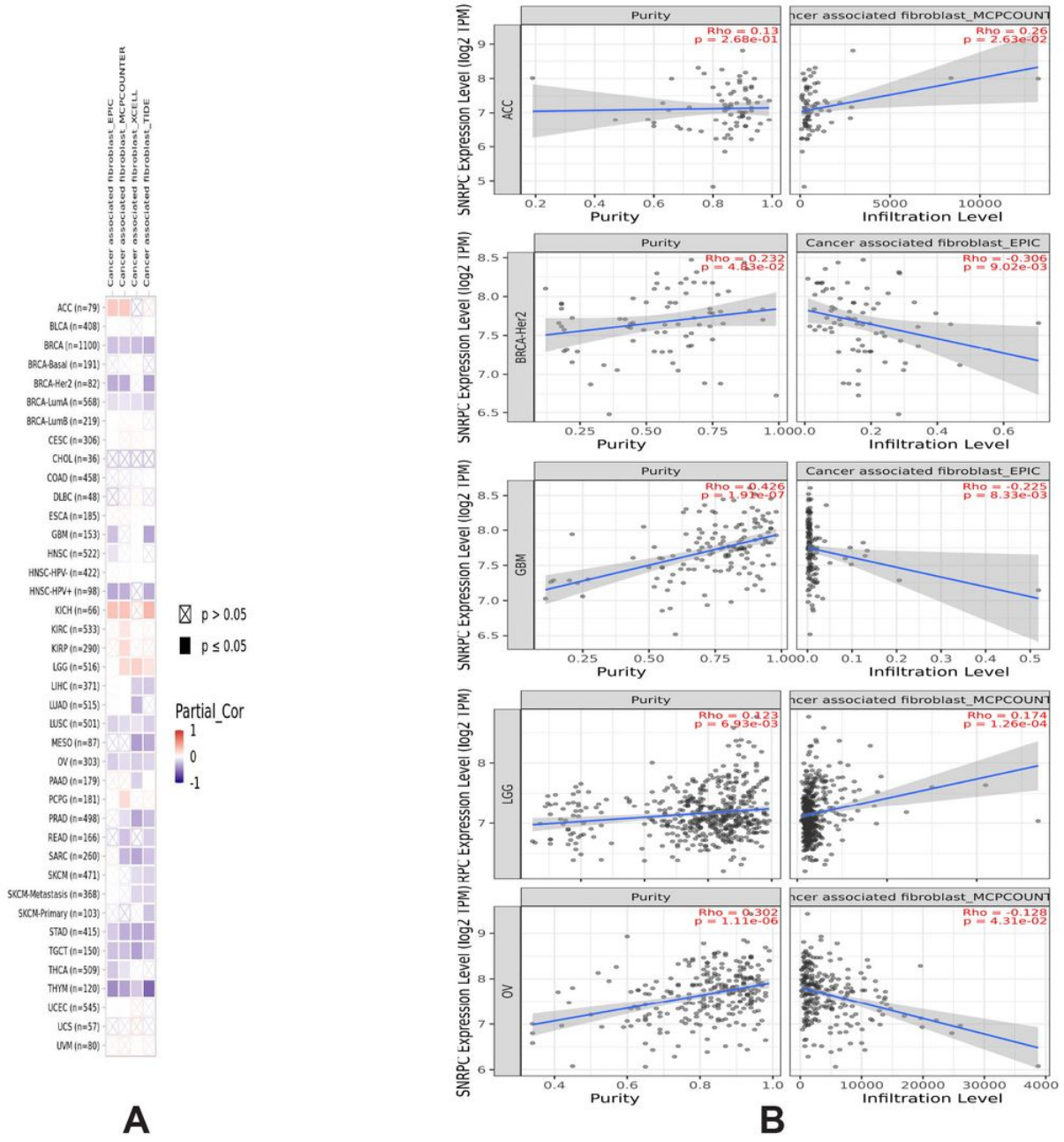

Figure 5

Relationship between SNRPC expression and TIICs (tumor-infiltrating immune cell) a. Correlation between SNRPC expression and 6 types of immune cells in the TIMER database. b. The relationship between SNRPC expression and TIICs was compared in the form of lollipop charts.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- table11.xlsx