

Analysis of CEP55 pan cancer immune and prognostic based on TCGA and GTEx databases

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Overview

Background

The CEP55 encoded product is a centrosome protein.Participate in the development of the cranioskeletal system,Establish protein localizationand intermediate separation。 Acting on the upstream or internal division of mitotic cells. CEP55 can affect the prognosis of various tumor types through multiple pathways. We evaluated the role of CEP55 using pan cancer data from the Cancer Genome Atlas (TCGA) and Genotyping Tissue Expression (GTEx) projects.

Method

The potential role of CEP55 in different tumors was explored based on the Cancer Genome Atlas (TCGA) and Genotyping Tissue Expression (GTEx) datasets.CEP55 related expression differences, tumor mutation burden (TMB), microsatellite instability (MSI), MATH, clinical staging and gene expression, immune cell analysis (xCELL, CIBERSORT, Timer), gene expression prognosis, etc.Our research findings indicate that CEP55 affects the prognosis of cancer patients, and its role varies among different types of cancer. In some cases, elevated CEP55 gene expression is detrimental to survival, while the opposite is true in other tumor types.

Result

There are differences in the expression of CEP55 in various tumor tissues and normal tissues adjacent to cancer.CEP55 is correlated with common immunotherapy targets such as TMB and MIS,Developing drugs related to CEP55, with the potential to treat related tumors through TMB and MSI immune targets. CEP55 is related to clinical staging of tumors and can serve as a basis for adjusting treatment plans for different stages of clinical tumors. CEP55 has shown significant correlation in various immune cell analyses, indicating that CEP55 widely affects tumor immune cells, which may in turn affect the immune regulation of various tumors, and may participate in the regulation of the immune microenvironment of multiple tumors, with the potential to serve as a potential immunotherapy target. CEP55 may be a potential new therapeutic target.

conclusion

The expression of CEP55 varies among different tumors and is correlated with the clinical staging of various tumors. It may be involved in immune regula-

tion of multiple tumors and may serve as a potential therapeutic target.

Keywords: CEP55, pan cancer analysis, immune microenvironment, immune checkpoint.

1. Introduction

One of the important characteristics of malignant tumors is that gene mutations lead to the appearance of new antigens on the surface of cancer cells. Cancer cells have evolved complex immune escape mechanisms, which are obstacles to effective cancer immunotherapy. At present, multiple immune escape mechanisms have been discovered, such as the expression of endogenous "immune checkpoints", which can alleviate immune responses after antigen activation. These immune escape mechanisms have promoted the development of immune checkpoint inhibitors as anticancer drugs. In previous studies, we found that CEP55 is associated with the prognosis of various tumors. However, there are currently no relevant reports on its prognostic role in pan cancer immune infiltration. Therefore, this article conducted a pan cancer analysis on it.

In this study, sequencing data from tumor tissue and normal tissue from databases such as The Cancer Genome Atlas1 (TCGA) and The Genome Tissue Expression 2,3(GTEx) were combined to perform multi pathway pan cancer analysis of CEP55. We evaluated the expression of CEP5 and its relationship with the prognosis of cancer patients. We further examined the differential expression of CEP55, tumor mutation burden 5,6 (TMB), microsatellite instability (MSI), MATH, clinical staging and gene expression, immune cell analysis (xCELL, CIBERSORT, Timer), and gene expression prognosis. Our research findings provide new insights into the functional role of CEP55 in pan cancer, highlighting the potential mechanisms by which CEP55 affects the tumor microenvironment and cancer immunotherapy. These data suggest that CEP55 may serve as a prognostic marker for tumor immune infiltration.

2. Materials and Methods

2.1 Data download. We downloaded the standardized pan cancer dataset from UCSC7(<https://xenabrowser.net/>): TCGA TARGET GTEx (PANCAN, N=19131, G=60499) , and then extracted the expression data of CEP55 gene in various samples from it.

2.2 CEP55 gene expression analysis. We used R software (version 3.6.4) to calculate the expression differences between normal and tumor samples in each tumor, and performed significant difference analysis using non paired Wilcoxon Rank Sum and Signed Rank Tests. We observed significant upregulation in 33 tumors.

2.3 TMB and MSI analysis of CEP55. We screened CEP55 related samples and expression profiles from the pan cancer dataset, and obtained them from

GDC(<https://portal.gdc.cancer.gov/>) We downloaded the level 4 Simple Nucleotide Variation dataset of all TCGA samples processed by MuTect28 software from cancer. We used the tmb function of the R software package maftools (version 2.8.05) to calculate the tumor mutation burst (TMB) of each tumor. We integrated the TMB and gene expression data of the samples and obtained the MSI (Microsatellite instability) score of each tumor from previous studies. Furthermore, we performed a $\log_2(x+0.001)$ transformation on each expression value. We calculated their Pearson correlation in each type of tumor.

2.4 The expression data of CEP55 gene was extracted, and MATH (Mutant all tumor heterogeneity) of each tumor was calculated using the infraHeterogeneity function of R software package maftools (version 2.8.05). The TMB and gene expression data of the samples were integrated, and further $\log_2(x+0.001)$ transformation was performed on each expression value to eliminate cancer types with less than 3 samples in a single cancer type. Finally, expression data of 37 cancer types were obtained.

2.5 Clinical staging and gene expression analysis of CEP55. Extract CEP55 gene related expression data and samples, further perform $\log_2(x+0.001)$ transformation on each expression value, eliminate cancer species with less than 3 samples in a single cancer species, and finally obtain expression data for 37 cancer species. Then their Pearson correlation was calculated for each type of tumor.

2.6 Immunocyte analysis of CEP55 (xCELL9). Extract the expression data of CEP55 in various tumors. Re evaluated the aDC/Adipocytes/Astrocytes/B-cells/Basophils/CD4+_memory_T-cells/CD4+_naive_T-cells/CD4+_T-cells/CD4+_Tcm/CD4+_Tem/CD8+_naive_T-cells/CD8+_T-cells/CD8+_Tcm/CD8+_Tem/cDC/Chondrocytes/Class-switched_memory_B-cells/CLP/CMP/DC/Endothelial_cells/Eosinophils/Epithelial_cells/Erythrocytes/Fibroblasts/GMP/Hepatocytes/HSC/iDC/Keratinocytes/ly_Endothelial_cells/Macrophages/Macrophages_M1/Macrophages_M2/Mast_cells/Megakaryocytes/Melanocytes/Memory_B-cells/MEP/Mesangial_cells/Monocytes/MPP/MSC/mv_Endothelial_cells/Myocytes/naive_B-cells/Neurons/Neutrophils/NK_cells/NKT/Osteoblast/pDC/Pericytes/Plasma_cells/Platelets/Preadipocytes/pro_B-cells/Sebocytes/Skeletal_muscle/Smooth_muscle/Tgd_cells/Th1_cells/Th2_cells/Tregs/ImmuneScore/StromaScore of each patient in each tumor based on gene expression. The Pearson's correlation coefficient between genes and immune cell infiltration scores in various tumors was calculated using the corr.test function of the R software package psych (version 2.1.6) to determine significantly correlated immune infiltration scores.

2.7 Immunocyte analysis of CEP55 (CIBERSORT). Extract CEP55 expression data and use the deconvo-CUBERSOR method of R software package IOBR Re evaluated the B-cells/B_cells_naive/B_cells_memory/Plasma_cells/T_cells_CD8/T_cells_CD4_naive/T_cells_CD4_memory_resting/T_cells_CD4_memory_activated/T_cells_follicular_helper/T_cells_regulatory_(Tregs)/T_cells_gamma_delta/NK_cells_resting/NK_cells_activated/Monocytes/Mac-

rophages_M0/Macrophages_M1/Macrophages_M2/Dendritic_cells_resting/Dendritic_cells_activated/Mast_cells_resting/Mast_cells_activated/Eosinophils/Neutrophils infiltration score of each patient in each tumor based on gene expression. The Pearson's correlation coefficient between genes and immune cell infiltration scores in various tumors was calculated using the `corr.test` function of the R software package `psych` (version 2.1.6) to determine significantly correlated immune infiltration scores.

2.8 CEP55 immune cell analysis (Timer). Extract the expression profile of CEP55 in various tumors, and use the Timer method of R software package IOBR to re evaluate the B cell, T cell CD4, T cell CD8, Neutrophil, Macrophage, and DC infiltration scores of each patient in each tumor based on gene expression.

The Pearson's correlation coefficient between genes and immune cell infiltration scores in various tumors was calculated using the `corr.test` function of the R software package `psych` (version 2.1.6) to determine significantly correlated immune infiltration scores.

2.9 Prognostic analysis of gene expression of CEP55. Extract the expression data of CEP55 gene in various samples, obtain TARGET follow-up data as a supplement, and exclude samples with follow-up time less than 30 days. Further, perform $\log_2(x+0.001)$ transformation on each expression value, exclude cancer types with less than 10 samples in a single cancer type, and finally obtain the expression data of 44 cancer types and the corresponding Overall survival data of the samples: TCGA-GBM/TCGA-GBMLGG/TCGA-LGG/TCGA-UCEC/TARGET-LAML/TCGA-BRCA/TCGA-CESC/TCGA-LUAD/TCGA-ESCA/TCGA-STES/TCGA-SARC/TCGA-KIRP/TCGA-KIPAN/TCGA-COAD/TCGA-COADREAD/TCGA-PRAD/TCGA-STAD/TCGA-HNSC/TCGA-KIRC/TCGA-LUSC/TCGA-THYM/TCGA-LIHC/TARGET-WT/TCGA-SKCM-P/TCGA-SKCM/TCGA-BLCA/TCGA-SKCM-M/TCGA-THCA/TARGET-NB/TCGA-MESO/TCGA-READ/TCGA-OV/TCGA-UVM/TCGA-PAAD/TCGA-TGCT/TCGA-UCS/TCGA-LAML/TARGET-ALL/TCGA-PCPG/TCGA-ACC/TARGET-ALL-R/TCGA-DLBC/TCGA-KICH/TCGA-CHOL. Establish a Cox proportional hazards regression model using the `Cox` function of the R software package `Survival` (version 3.2-7) to analyze the relationship between gene expression and prognosis in each tumor. Logrank test was used for statistical analysis to obtain prognostic significance.

3.Result

3.1 CEP55 gene expression analysis. We observed significant upregulation in 33 types of tumors, such as GBM/GBMLGG/LGG/UCEC/BRCA/CESC/LUAD/ESCA/STES/KIRP/KIPAN/COAD/COADREAD/PRAD/STAD/HNSC/KIRC/LUSC/LIHC/WT/SKCM/BLCA/THCA/READ/OV/PAAD/UCS/ALL/LAML/PCPG/ACC/KICH/CHOL. As shown in **Figure 1**.

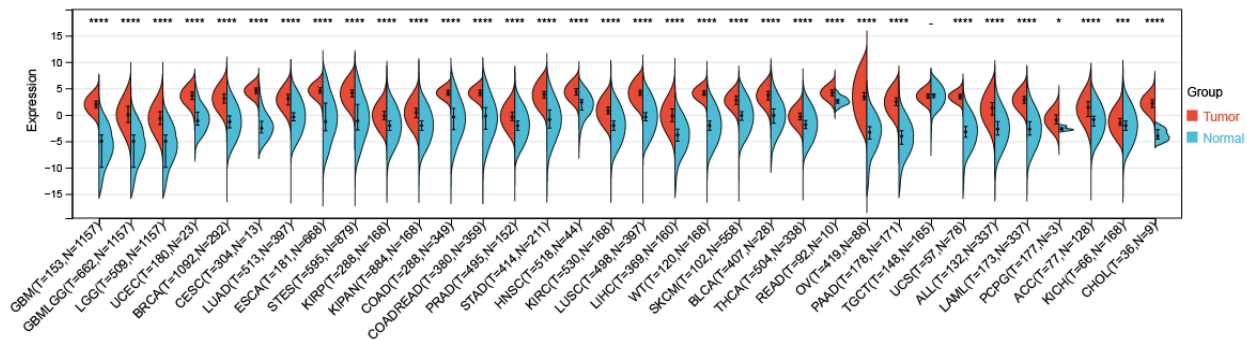


Figure 1. Expression analysis of CEP55 gene in various tumors.

3.2 The TMB and MSI analysis results of CEP55. Through TMB analysis of CEP55, we observed significant correlations among 18 tumors, with significant positive correlations observed in all 18 tumors, such as:GBM/GBMLGG/LGG/LUAD/COAD/COADREAD/BRCA/STES/SARC/KIPAN/STAD/PRAD/KIRC/TGCT/PCPG/BLCA/ACC/KICH. As shown in **Figure 2**. MSI analysis results of CEP55. Through MSI analysis, we found that CEP55 was significantly correlated in 12 tumors, with a significant positive correlation in 9 tumors, such as:GBM/COAD/COADREAD/STES/SARC/STAD/LUSC/LIHC/READ, There is a significant negative correlation among three types of tumors, such as:GBM-LGG/KIPAN/DLBC. As shown in **Figure 3**.

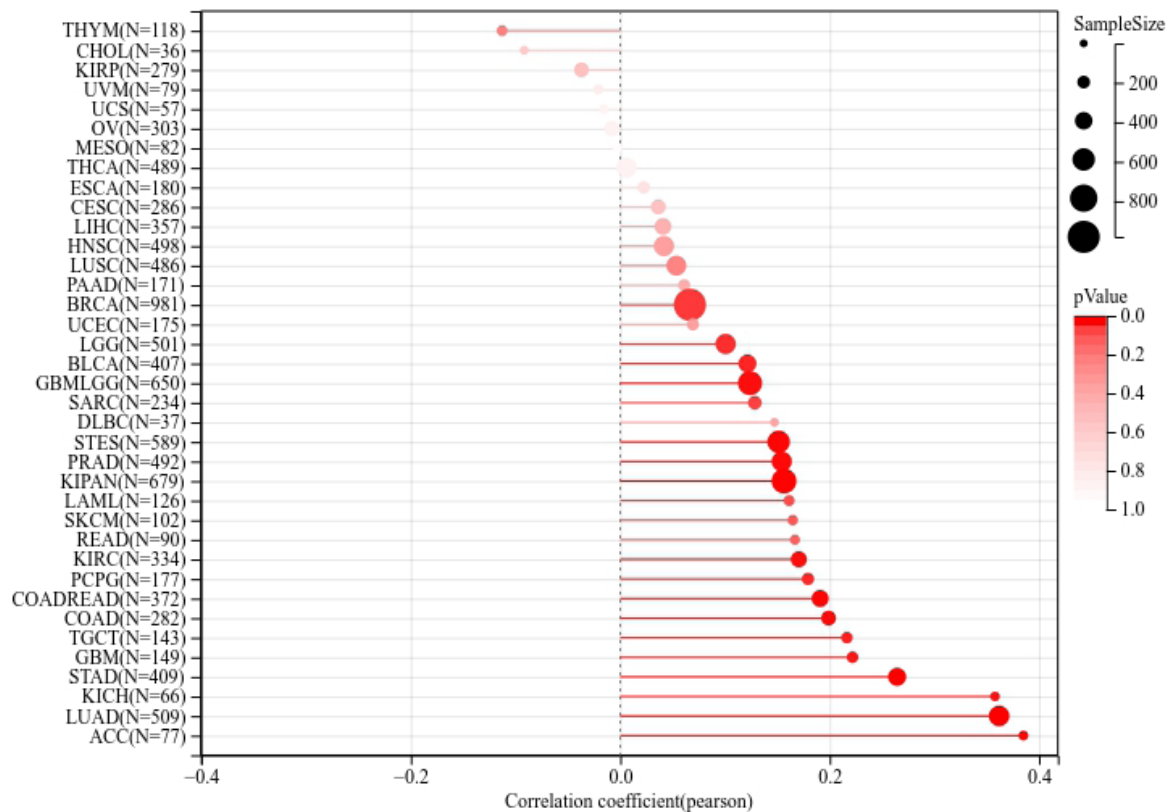


Figure 2.CEP55 TMB

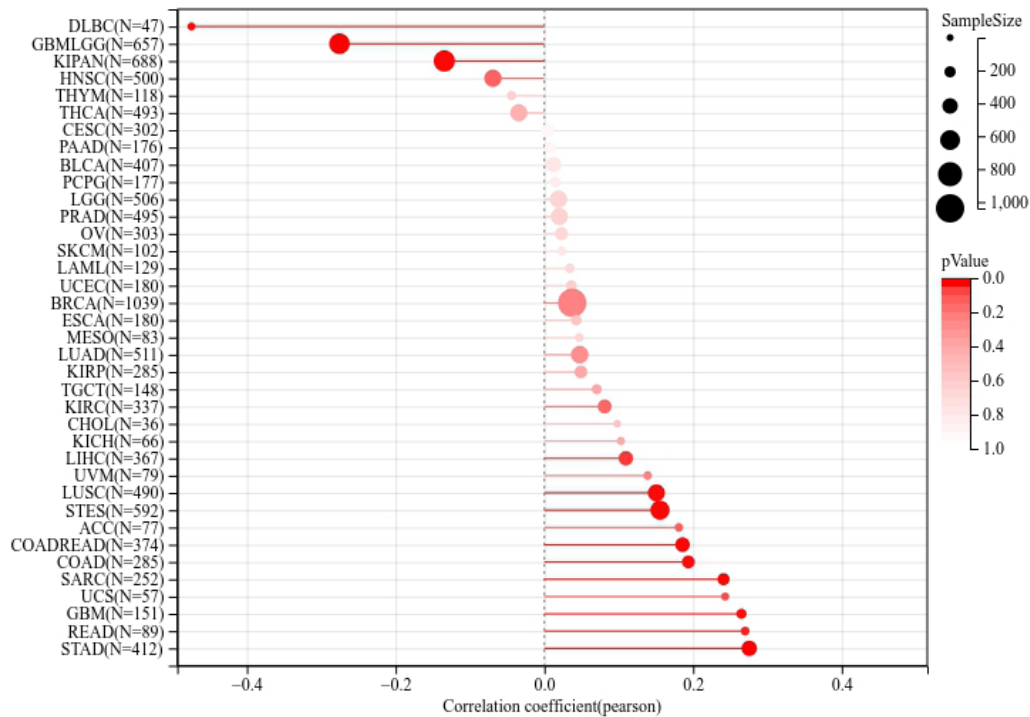


Figure 3. MSI analysis of CEP55.

3.The MATH analysis results of CEP55. Through MATH analysis of CEP55, we observed significant correlations in 12 tumors, with significant positive correlations in 8 tumors, such as:LUAD/BRCA/ESCA/STES/SARC/LUSC/ LIHC/BLCA,There is a significant negative correlation among the four types of tumors, such as:GBMLGG/LGG/KIPAN/THCA。 As shown in **Figure 4**.

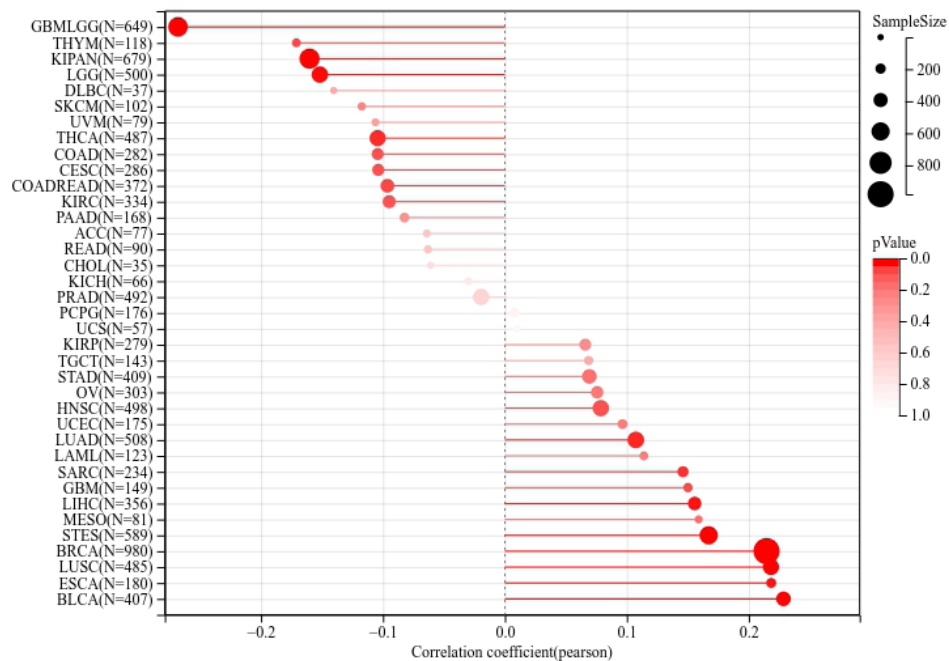


Figure 4. MATH analysis of EP55.

3.4 Clinical staging and gene expression analysis results of CEP55. Significant differences in CEP55 expression were observed in four types of tumors, such as:GBM/GBMLGG/KIRP/UCS。 As shown in **Figure 5**.

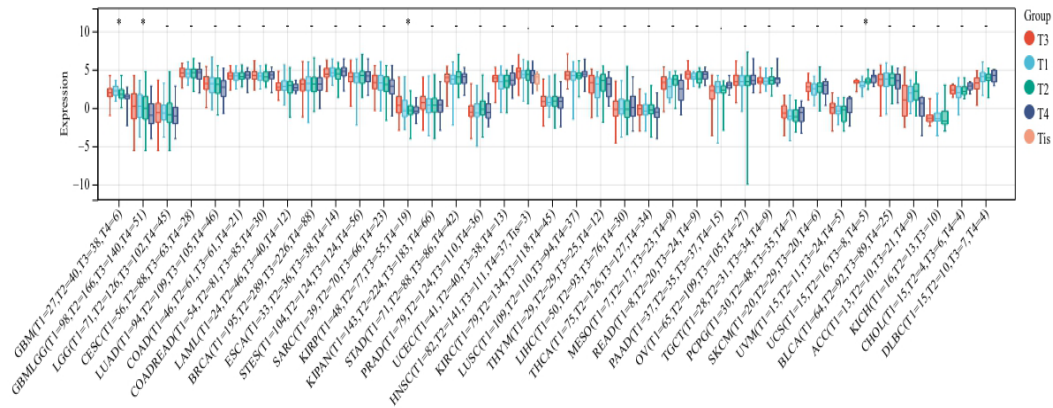


Figure 5. Clinical staging and gene expression analysis of CEP55.

3.5 The immune cell analysis (xCELL9) results of CEP55. Through immune cell analysis (xCELL), it was observed that CEP55 gene expression was significantly correlated with immune infiltration in 44 cancer types. such as:GBM/GBMLGG/LGG/UCEC/TARGET-LAML/BRCA/CESC/LUAD/ESCA/STES/SARC/KIRP/KIPAN/COAD/COADREAD/PRAD/STAD/HNSC/KIRC/LUSC/THYM/LIHC/TARGET-WT/SKCM-P/SKCM/BLCA/SKCM-M/TTHCA/TARGET-NB/MESO/READ/OV/UVM/PAAD/TGCT/UCS/LAML/TARGET-ALL/PCPG/ACC/TARGET-ALL-R/DLBC/KICH/CHOL. As shown in **Figure 6**.

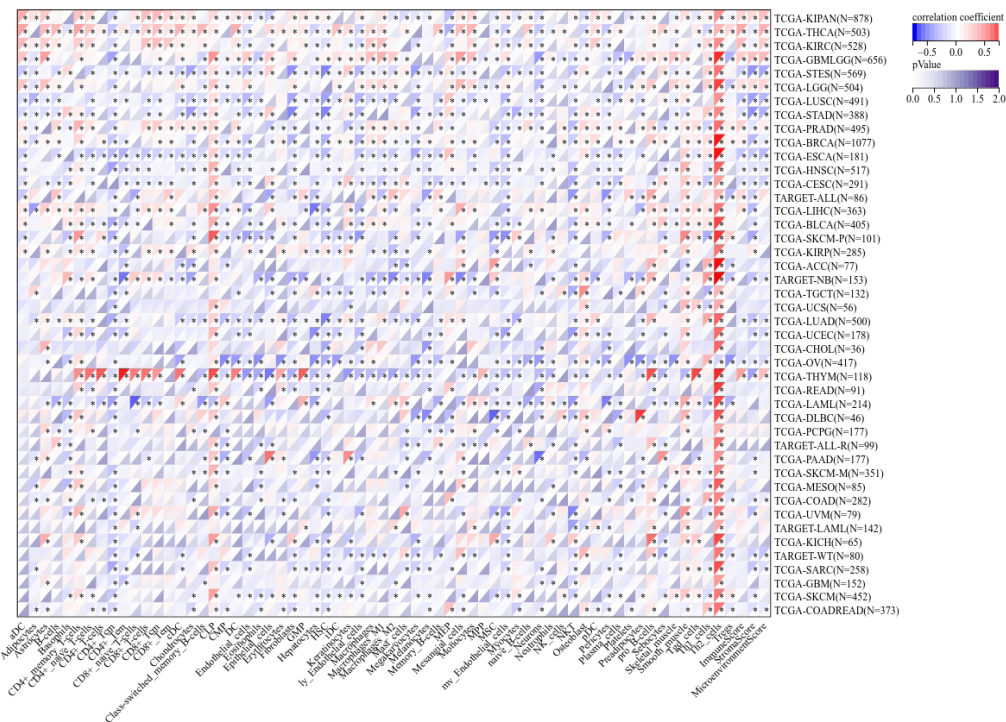


Figure 6. Immune cell analysis (xCELL) of CEP55.

3.6 The immune cell analysis (CIBERSORT) results of CEP55. Through analysis, it can be found that CEP55 gene expression is significantly correlated with immune infiltration in 44 cancer types, such as GBM/GBMLGG/LGG/UCEC/TARGET-LAML/BRCA/CESC/LUAD/ESCA/STES/SARC/KIRP/KIPAN/COAD/COADREAD/PRAD/STAD/HNSC/KIRC/LUSC/THYM/LIHC/TARGET-WT/SKCM-P/SKCM/BLCA/SKCM-M/THCA/TARGET-NB/MESO/READ/OV/UVM/PAAD/TGCT/UCS/LAML/TARGET-ALL/PCPG/ACC/TARGET-ALL-R/DLBC/KICH/CHOL. As shown in **Figure 7**.

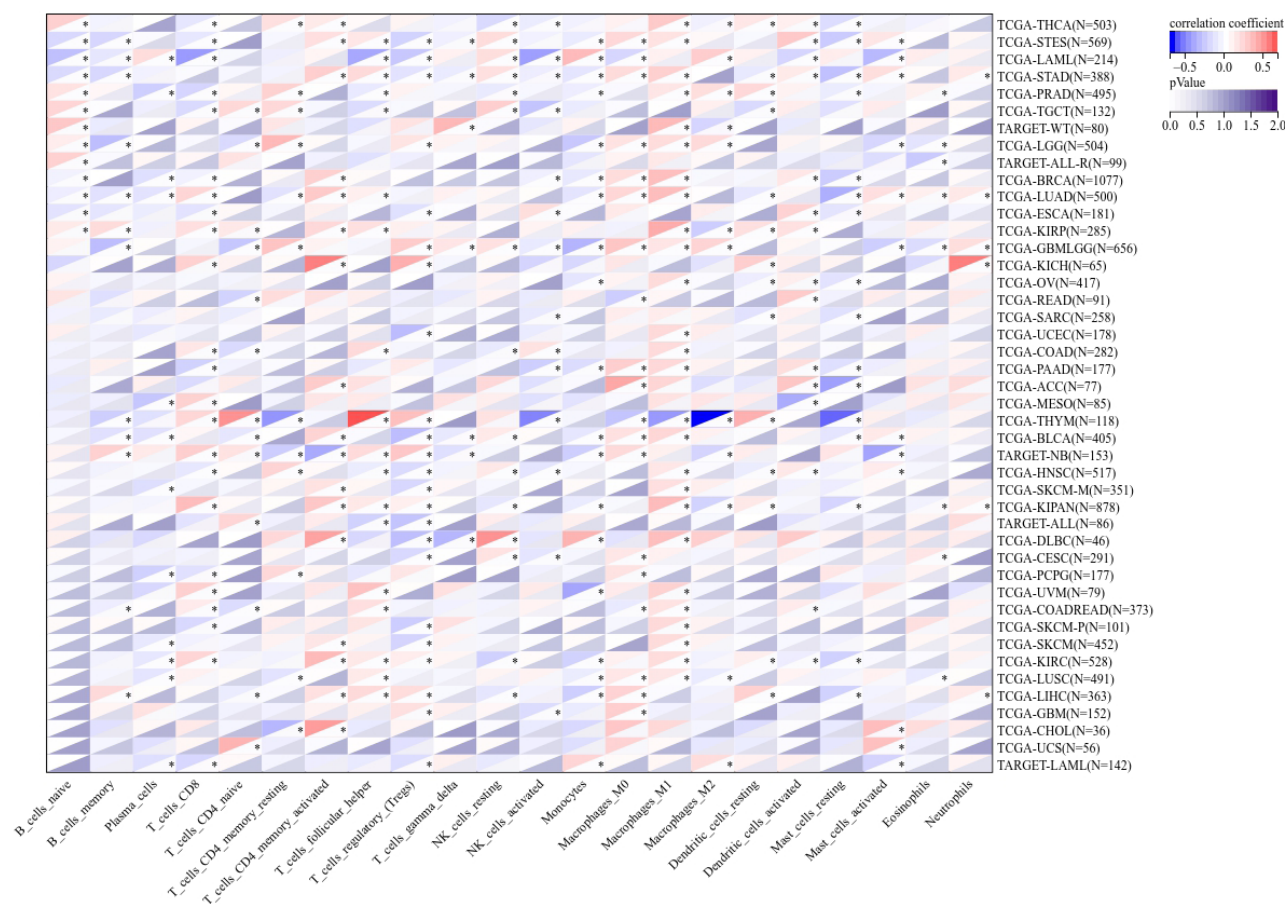


Figure 7. Immune cell analysis of CEP55 (CIBERSORT).

3.7 The immune cell analysis (Timer) results of CEP55. Through Timer analysis, it was observed that CEP55 gene expression was significantly correlated with immune infiltration in 38 cancer types (ACC/BLCA/BRCA/CESC/CHOL/COAD/COADREAD/DLBC/ESCA/GBM/GBMLGG/HNSC/KICH/KIPAN/KIRC/KIRP/LGG/LIHC/LUAD/LUSC/MESO/OV/PAAD/PCPG/PRAD/READ/SARC/SKCM-M/SKCM-P/SKCM/STAD/STES/TGCT/THCA/THYM/UCEC/UCS/UVM). As shown in **Figure 8**.

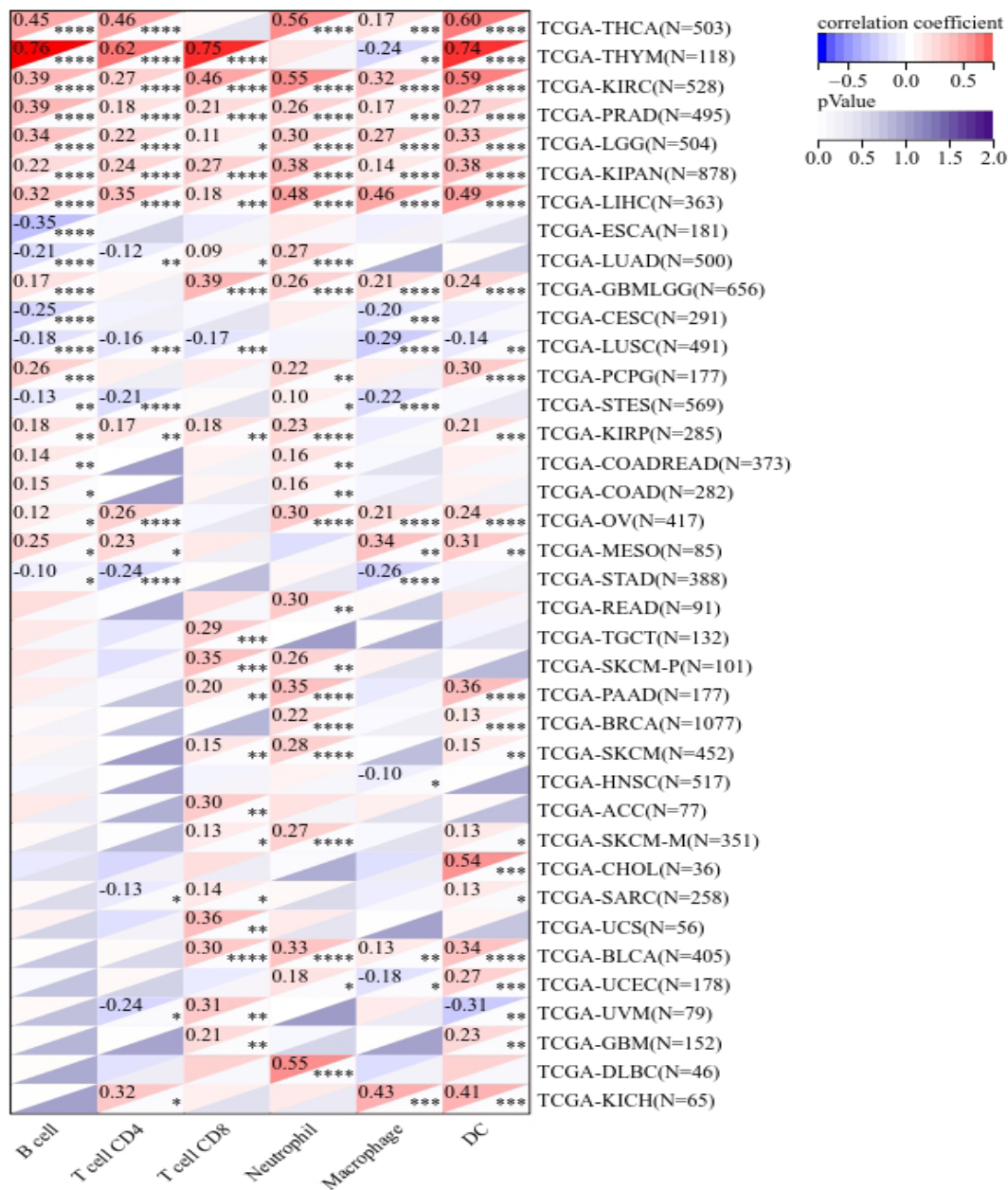


Figure 8 Immune cell analysis of CEP55 (Timer).

3.8 Prognostic analysis results of CEP55 gene expression. The pan cancer prognosis analysis of CEP55 found that high expression had poor prognosis in 14 tumor types (GBMLGG/LGG/TARGET-LAML/LUAD/KIRP/KIPAN/KIRC/LIHC/MESO/UVM/PAAD/LAML/ACC/KICH), and low expression in THYM had poor prognosis in 1 tumor type. As shown in **Figure 9**.

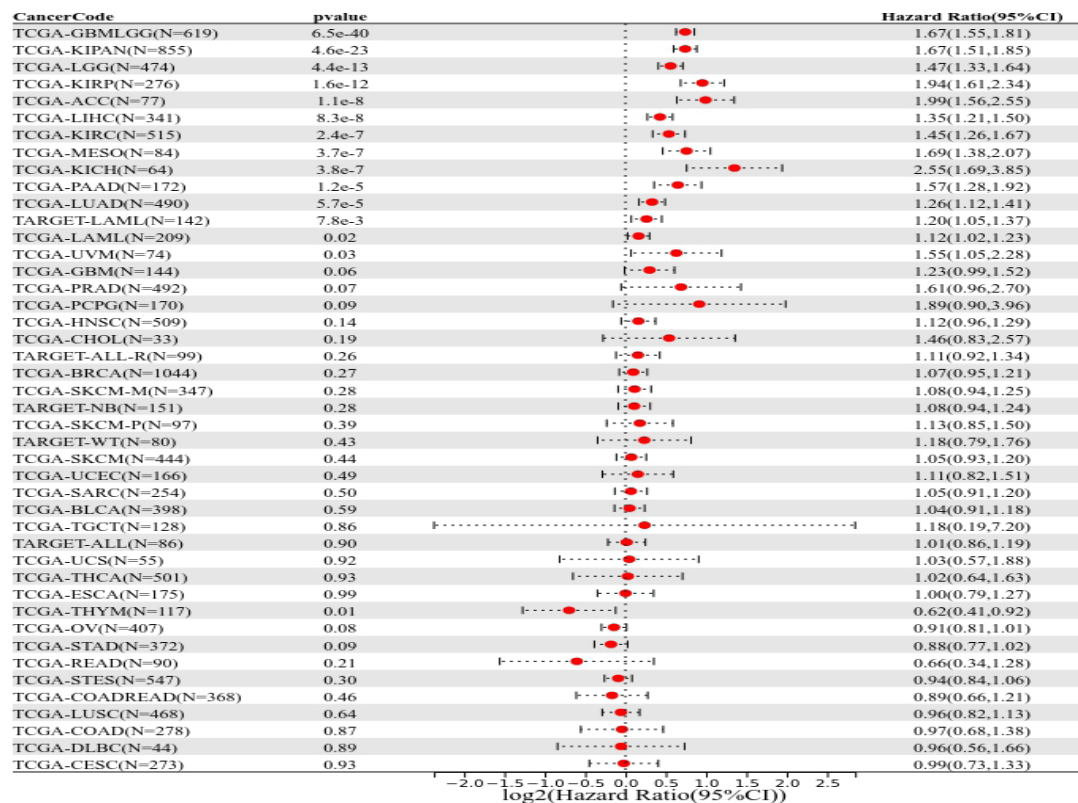


Figure 9. Prognostic analysis of gene expression of CEP55.

Discussion

The CEP55 encoded product is a centrosome protein. Participate in the development of the cranioskeletal system; Establish protein localization; Separate from the intermediate layer. Acting on the upstream or interior of mitotic cell division. Previous studies have found that CEP55 is involved in the occurrence and development of various tumors, and may be associated with their prognosis. CEP55 may be associated with the prognosis of renal cancer 10-13, poor prognosis of esophageal squamous cell carcinoma 14,15, and may also affect the proliferation and migration of esophageal cancer cells 16. Some studies have also found that it may be involved in promoting the occurrence of neuroblastoma 17,18. Other studies have found that CEP55 may be involved in the regulation of a variety of tumors, and affect the prognosis and development of a variety of tumors, such as: the prognosis of lung cancer 19-24, the prognosis of rectal cancer 25,26, the prognosis of liver cancer 27-30, the prognosis of astrocytoma 31, the development of anaplastic thyroid cancer (ATC) 32, the regulatory role of colloid cell tumor 33-35, the progress of prostate cancer (PCa) 36,37, the progress of breast cancer 38,39 the proliferation and invasion of osteosarcoma 40, the occurrence and development of bladder cancer 41, and the prognosis of cervical cancer 42. These studies demonstrate that CEP55 is widely involved in and affects the prog-

nosis and development of various tumors, and has the potential to serve as a therapeutic target.

This study investigated the differential expression, tumor mutation burden (TMB), microsatellite instability (MSI), clinical staging, and gene expression of CEP55 MATH/ Immune cell analysis (xCELL, CIBERSORT, Timer), gene expression prognostic analysis. Further explore its role in pan cancer.

Through differential expression analysis of CEP55, we found that it was significantly upregulated in 33 types of tumors, including: GBM/GBMLGG/LGG/UCEC/BRCA/CESC/LUAD/ESCA/STES/KIRP/KIPAN/COAD/COADREAD/PRAD/STAD/HNSC/KIRC/LUSC/LIHC/WT/SKCM/BLCA/THCA/READ/OV/PAAD/UCS/ALL/LAML/PCPG/ACC/KICH/CHOL. This indicates that there are significant differences in the expression of CEP55 in most tumors.

The TMB analysis of CEP55 found that it is associated with 18 tumors, which are:GBM/GBMLGG/LGG/LUAD/COAD/COADREAD/BRCA/STES/SARC/KIPAN/STAD/PRAD/KIRC/TGCT/PCPG/BLCA/ACC/KICH. The microsatellite instability (MSI) analysis of CEP55 showed a significant correlation with 12 types of tumors, among which 9 types were significantly positively correlated, such as:GBM/COAD/COADREAD/STES/SARC/STAD/LUSC/LIHC/READ,There is a significant negative correlation among three types of tumors, such as:GBMLGG/KIPAN/DLBC. There is a significant negative correlation among three types of tumors, such as:

The clinical staging and gene expression analysis of EP55 showed significant differences among four types of tumors: GBM/GBMLGG/KIRP/UCS. The MATH analysis of CEP55 observed a significant correlation among 12 tumors, with a significant positive correlation among 8 tumors, such as:LUAD/BRCA/ESCA/STES/SARC/LUSC/LIHC/BLCA,There is a significant negative correlation among the four types of tumors, such as:GBMLGG/LGG/KIPAN/THCA. This indicates that CEP55 may be related to the clinical staging of various tumors, and has potential guidance for the treatment of tumors in different stages.

The immune cell analysis (xCELL9, CIBERSORT 43) of CEP55 suggests that its gene expression is significantly correlated with immune infiltration in 44 cancer types: GBM/GBMLGG/LGG/UCEC/LAML/BRCA/CESC/LUAD/ESCA/STES/SARC/KIRP/KIPAN/COAD/COADREAD/PRAD/STAD/HNSC/KIRC/LUSC/THYM/LIHC/WT/SKCM-P/SKCM/BLCA/SKCM-M/THCA/NB/MESO/READ/OV/UVM/PAAD/TGCT/UCS/LAML/ALL/PCPG/ACC/ALL-R/DLBC/KICH/CHOL.The immune cell analysis (Timer44) of CEP55 suggests that we observed a significant correlation between gene expression and immune infiltration in 38 cancer types: ACC/BLCA/BRCA/CESC/CHOL/COAD/COADREAD/DLBC/ESCA/GBM/GBMLGG/HNSC/KICH/KIPAN/KIRC/KIRP/LGG/LIHC/LUAD/LUSC/MESO/OV/PAAD/PCPG/PRAD/READ/SARC/SKCM-M/SKCM-P/SKCM/STAD/STES/TGCT/THCA/THYM/UCEC/UCS/UVM.Through the above analysis of multiple immune cells of

CEP55 in up to 44 types of tumor tissues. We found that CEP55 may be correlated in the immune microenvironment of up to 44 types of tumors and may affect the prognosis of these tumors. Therefore, this study found that CEP55 may serve as a potential new immune checkpoint and can be used to develop related inhibitors as anti-tumor drugs.

The prognostic analysis of CEP55 gene expression suggests that high expression in 14 tumor types (GBMLGG/LGG/LAML/LUAD/KIRP/KIPAN/KIRC/LIHC/MESO/UVM/PAAD/LAML/ACC/KICH) leads to poor prognosis, while low expression in one tumor type THYM leads to poor prognosis. Through prognostic analysis of CEP55 and various tumors, it can be found that it has a significant correlation with the prognosis of multiple tumors. This suggests that CEP55 may affect the prognosis of multiple tumors.

Although this study used multiple databases and multidimensional analysis to draw relevant conclusions, it lacks specific experimental verification. To increase the reliability of the above research, we can conduct further experimental verification of the results to enhance their reliability and provide a basis for future drug research.

In summary, our research results indicate that there are differences in the expression of CEP55 in various tumor tissues and normal tissues adjacent to cancer. CEP55 is correlated with common immunotherapy targets such as TMB and MIS. Developing drugs related to CEP55 has the potential to treat related tumors through TMB and MSI immunotherapy targets. CEP55 is related to clinical staging of tumors and can serve as a basis for adjusting treatment plans for different stages of clinical tumors. CEP55 has shown significant correlation in various immune cell analyses, indicating that CEP55 widely affects tumor immune cells, which may in turn affect the immune regulation of various tumors, and may participate in the regulation of the immune microenvironment of multiple tumors, with the potential to serve as a potential immunotherapy target. CEP55 may be a potential new therapeutic target.

Conclusion

The expression of CEP55 varies among different tumors and is correlated with the clinical staging of various tumors. It may be involved in immune regulation of multiple tumors and may serve as a potential therapeutic target.

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