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Comprehensive bioinformatic analysis of the expression and prognostic significance of TSC22D domain family genes in acute myeloid leukemia

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ABSTRACT

Background:

TSC22D domain family genes, including Tsc22d1-4, have been extensively reported to be involved in tumors. However, their expression profiles and prognostic significance in acute myeloid leukemia (AML) remain unknown.

Methods:

The present study investigated the expression profiles and prognostic significance of TSC22D domain family genes in AML through the use of multiple online databases, including the CCLE, EMBL-EBI, HPA, Oncomine, GEPIA2, UALCAN, BloodSpot, and GSCALite databases. The cBioPortal and GSCALite databases were used to explore the genetic alteration and copy number variation (CNV) of the Tsc22d3 gene. The TRRUST (Version 2) database was used to explore the gene ontology biological process, disease ontology, and Kyoto Encyclopedia of Genes and Genomes (KEGG) pathways associated with the Tsc22d3 gene. The AnimalTFDB3.0, STRING, and Harmonizome databases were used to investigate the protein-protein interaction (PPI) network of the Tsc22d3 gene. The Harmonizome database was used for Tsc22d3 gene regulatory kinase analysis. The TargetScanHuman 7.2, MiRDB, and ENCORI databases were used to execute the analysis of the Tsc22d3 gene regulatory miRNAs. Then, the GSCALite and GEPIA2021 databases were used to investigate the correlation between Tsc22d3 expression and immune infiltration.

Results:

The expression of the Tsc22d3 gene was upregulated markedly in AML cells relative to normal hematopoietic stem cells. The expression of the Tsc22d3 gene was increased in AML tumor samples compared with healthy bone marrow samples. And overexpression of the Tsc22d3 gene was associated with poor OS in AML patients. This study implied that the Tsc22d3 gene is a new biomarker for

predicting the prognosis of AML. Furthermore, gene ontology analysis showed that Tsc22d3 was involved in leukemia. Functional enrichment analysis showed that the Tsc22d3 gene has many biological functions, including the regulation of many genes, kinases, miRNAs, signaling pathways, and immune infiltration. Therefore, this study suggests that the Tsc22d3 gene may be a potential therapeutic target for AML.

Conclusions:

Tsc22d3 gene expression was upregulated in AML, and overexpression was associated with poor OS in AML patients. Therefore, the Tsc22d3 gene may serve as a novel prognostic biomarker and therapeutic target for AML.

Keywords: acute myeloid leukemia, prognostic biomarker, therapeutic target, tumor infiltration, bioinformatics analysis

INTRODUCTION

Acute myeloid leukemia (AML) is an aggressive hematopoietic malignancy with high biological and clinical heterogeneity[1]. Despite advances made in the diagnosis and treatment of AML, the increased risk of relapse and low 5-year survival rate after diagnosis remain significant challenges[2]. Authentication of new AML biomarkers can help to clarify the pathogenesis of the disease and guide the diagnosis, treatment, and prognosis evaluation of AML[3]. TSC22D domain family genes have been extensively reported to play an essential role in tumors[4-8]. Nonetheless, their expression profiles and significance in prognosis prediction for AML remain unclear. Herein, we conducted an analysis of the expression and prognostic value of TSC22D domain family genes in AML through the use of multiple online databases. See Supplementary Material 1 for the details of each online database login site.

RESULTS

Comprehensive bioinformatics analysis of the TSC22D domain family genes in AML was performed by using data from TCGA and GEO databases. The specific analysis process is shown in Figure 1.

Analysis of TSC22D domain family gene expression in AML cell lines, normal hematopoietic stem cells, AML tissues and healthy bone marrow

First, three different databases, including the CCLE, EMBL-EBI, and HPA databases, were used to authenticate the expression levels of TSC22D domain family genes in AML cell lines. We found that TSC22D domain family genes were abnormally expressed in AML cell lines at different levels (Figure 2). Second, we used the OncoPrint and BloodSpot databases to check the expression levels of TSC22D domain family genes in AML cells and normal hematopoietic stem cells and performed statistical analysis. As shown in Figure 3, analysis from three different datasets revealed that the expression of the Tsc22d3 gene was upregulated markedly in AML cells relative to normal hematopoietic stem cells, whereas the

expression of the Tsc22d1 gene was the opposite. Third, the OncoPrint, BloodSpot, and GEPIA2 databases were used to authenticate the expression levels of TSC22D domain family genes in AML tissues and healthy bone marrow. As depicted in Figure 4, the expression of the Tsc22d1 gene and Tsc22d3 gene increased in the AML tumor tissues compared with the healthy bone marrow.

Survival analysis according to the expression of the TSC22D domain family genes in AML

We found that high expression of the Tsc22d3 gene was associated with poor OS in AML patients by survival analysis of GEPIA2, UALCAN, BloodSpot, and GSCALite datasets (Figure 5).

Genetic alteration and CNV analysis of the Tsc22d3 gene

Analysis of the TCGA PanCancer Atlas dataset from the cBioPortal database revealed that the mutation rate of the Tsc22d3 gene was 8%, and Tsc22d3 gene alteration did not affect the OS of AML patients. Likewise, CNV analysis of the GSCALite database showed that the incidence of CNV of the Tsc22d3 gene was low in AML and did not affect the OS of AML patients (Figure 6).

Functional enrichment analysis of the Tsc22d3 gene

We analyzed the gene ontology biological processes, disease ontology terms and KEGG pathways associated with the Tsc22d3 gene by using the TRRUST database. The results showed that the Tsc22d3 gene has many biological functions (Figure 7A). Disease ontology analysis revealed that the Tsc22d3 gene was involved in tumors, including leukemia (Figure 7B). Furthermore, KEGG pathway analysis indicated that the Tsc22d3 gene was involved in the regulation of multiple signaling pathways (Figure 7C).

PPI analysis of the Tsc22d3 gene

PPI analysis from the AnimalTFDB3.0, STRING, and Harmonizome databases indicated that the Tsc22d3 protein interacted with many proteins related to the regulation of cell proliferation and differentiation, including FOS, JUN, and NFkB, etc. (Figure 8A, 8B, 8C).

Analysis of kinases regulated by the Tsc22d3 gene

We analyzed the predicted kinases regulated by the Tsc22d3 gene through the use of the Harmonizome database. We found that the Tsc22d3 gene regulated many kinases, including TGFBR2, MAPKs, and JAKs, etc. (Figure 8D).

Analysis of miRNAs regulated by the Tsc22d3 gene

Analysis of the Tsc22d3-regulated miRNAs from the TargetScanHuman 7.2, MiRDB, and ENCORI databases indicated that the Tsc22d3 gene regulated many miRNAs, including hsa-miR-101-3p, hsa-miR-125b-5p, hsa-miR-135b-5p, hsa-miR-182-5p, hsa-miR-193a-3p, hsa-miR-216b-5p, hsa-miR-362-5p, hsa-miR-370-3p, and hsa-miR-98-5p, etc. (Figure 9).

Immune infiltration analysis of the Tsc22d3 gene

Analysis of Tsc22d3 gene expression and immune infiltration in AML from the GSCALite database indicated that the Tsc22d3 gene was enriched in exhausted T cells, macrophages and monocytes. Analysis of Tsc22d3 gene expression and immune infiltration in AML samples from the GEPIA2021 database revealed that the Tsc22d3 gene was enriched in resting memory CD4⁺ T cells, CD8⁺ T cells, resting NK cells, plasma cells, monocytes, etc. (Figure 10).

DISCUSSION

TSC22D domain family genes, including Tsc22d1-4, belong to the leucine zipper TF family and have been reported to be involved in regulating cell proliferation and differentiation[9]. Tsc22d1, also called transforming growth factor- β -stimulated clone-22, was reported to play a tumor suppressor role in tumors[10]. Tsc22d2 overexpression depends on the TSC22D2-PKM2-CyclinD1 regulatory axis to inhibit tumor cell growth in colorectal cancer[11]. Tsc22d3, also known as glucocorticoid-induced leucine zipper (GILZ), can promote or suppress tumor growth, depending on the type of tumor and its microenvironment. Tsc22d3 plays a dual role in tumors: it not only exerts a tumor-promoting effect by influencing the immune system and tumor microenvironment but also inhibits tumor growth by inducing apoptosis or suppressing the proliferation of cancer cells[12]. Tsc22d4, also known as THG-1, was reported to promote esophageal squamous cell carcinoma cell tumorsphere growth[13]. However, the expression of TSC22D domain family genes and their prognostic value in AML remain unclear.

The present study examined the expression profiles and prognostic significance of Tsc22d1-4 genes in AML using multiple online databases. We found that TSC22D domain family genes were abnormally expressed in AML cell lines at different levels. The expression of the Tsc22d3 gene was markedly upregulated in AML cells relative to normal hematopoietic stem cells, whereas the expression pattern of the Tsc22d1 gene was the opposite. Furthermore, we found that the expression of the Tsc22d1 gene and Tsc22d3 gene increased in AML tumor samples compared with healthy bone marrow samples. Further survival analysis revealed that high expression of the Tsc22d3 gene resulted in poor OS in AML patients. Therefore, we identified the Tsc22d3 gene as a new prognostic biomarker for AML. Next, we analyzed the profiles of the alteration and CNV of the Tsc22d3 gene in AML. We found that the incidence of genetic alterations and CNV of the Tsc22d3 gene were low in AML and did not affect the OS of AML patients.

As a TF, Tsc22d3 is involved in regulating TF activity and signaling pathways. Tsc22d3 promotes tumor growth by downregulating the antiapoptotic protein MCL[14]. Tsc22d3 confers leukemia cells with a proliferative and metabolic advantage by reprogramming glycolytic metabolism in tumor cells[15]. Hence, we analyzed the gene ontology biological process, disease ontology terms, and KEGG pathways associated with the human Tsc22d3 TF using the TRRUST (Version 2) database. We found that the Tsc22d3 TF had many biological functions, including the response to DNA damage stimulus, the

regulation of cell proliferation and cell cycle arrest. Disease ontology analysis revealed that the Tsc22d3 TF was involved in leukemia. Furthermore, KEGG pathway analysis indicated that the Tsc22d3 TF was involved in regulating multiple signaling pathways, including the FoxO signaling pathway, AMPK signaling pathway, PI3K–Akt signaling pathway, JAK–STAT signaling pathway, etc. These signaling pathways play a part in cancer progression. PPI analysis indicated that Tsc22d3 proteins interact with many proteins, including FOS, JUN, and NFκB, etc, which are involved in regulating the proliferation and differentiation of tumor cells. Tsc22d3 promoted tumor cell proliferation by regulating AKT kinase[16]. Therefore, we analyzed Tsc22d3 TF regulatory kinases. The results showed that the Tsc22d3 TF regulated many kinases, including TGFBR2, MAPKs, JAKs, etc. These kinases are involved in various cellular activities, such as cell proliferation and differentiation. Furthermore, we analyzed Tsc22d3 gene-regulated miRNAs. We found that the Tsc22d3 gene regulated many miRNAs, including hsa-miR-101-3p, hsa-miR-125b-5p, hsa-miR-135b-5p, hsa-miR-182-5p, hsa-miR-193a-3p, hsa-miR-216b-5p, hsa-miR-362-5p, hsa-miR-370-3p, and hsa-miR-98-5p, etc. These miRNAs were reported to participate in the occurrence and development of AML[17].

Tsc22d3 has been reported to be involved in the supervision of the cell cycle, differentiation, and apoptosis of immune cells[18]. Tsc22d3 can play an anti-inflammatory and immunosuppressive role in tumor development. Activation of the immunosuppressive Tsc22d3 TF in dendritic cells can result in treatment failure[19]. Tsc22d3 upregulation can subvert therapy-induced anticancer immunosurveillance[20]. As a TF, Tsc22d3 can mediate the immunosuppressive and anti-inflammatory effects of T cells and macrophages by inhibiting nuclear factor-κB (NF-κB)-dependent transcription[21, 22]. Furthermore, Tsc22d3 can play a significant role in tumor progression by mediating the increase in cell quantity and activity of Treg cells through the TGF-β signaling pathway[23, 24]. Tsc22d3 could play an indispensable role in the tumor microenvironment by influencing all immune system cells that infiltrate the tumor microenvironment[25]. In addition, Tsc22d3 could serve as a pivotal regulator of T cell predysfunction[26]. Recent research shows that the proliferation, survival, and drug resistance of AML cells are sustained and modulated by the bone marrow immunosuppressive microenvironment[27]. Hence, we conducted an analysis of Tsc22d3 expression and immune infiltration in AML. The results indicated that the Tsc22d3 gene was enriched in exhausted T cells, CD8⁺ T cells, macrophages and monocytes, etc.

Conclusions

Tsc22d3 may serve as a new prognostic biomarker and therapeutic target for AML. Tsc22d3 may be involved in AML through multiple mechanisms, including the regulation of genes, kinases, miRNAs, signaling pathways and immune infiltration. However, the specific mechanism of the Tsc22d3 gene in AML progression still needs to be further studied.

MATERIALS AND METHODS

Gene expression analysis

Analysis of gene expression in AML cell lines

The Cancer Cell Line Encyclopedia (CCLE)[28] is a multiomics online database collection of 1378 types of cancer cell lines. We downloaded the expression data of TSC22D domain family genes in forty-four types of AML cell lines from the CCLE database (Supplementary Material 2) and then employed the cluster heatmap tool from the website (<http://www.bioinformatics.com.cn>) for visualization.

EMBL-EBI[29] is an integrated bioinformatics research database. We downloaded the data and figures related to TSC22D domain family gene expression in fourteen types of AML cell lines from the EMBL-EBI database (Supplementary Material 3).

The Human Protein Atlas (HPA)[30] is a comprehensive database of proteomics, transcriptomics, and systems biology data. We downloaded pictures of TSC22D domain family gene expression in five types of AML cell lines from the HPA database.

Analysis of gene expression in AML cells and normal hematopoietic stem cells

Oncomine[31] is the world's largest database of oncogene chips and integrated data mining platforms. We downloaded the data of TSC22D domain family gene expression in AML cells and CD34-positive peripheral blood cells from the Valk leukemia dataset[32] in the Oncomine database. We used GraphPad Prism 8 statistical software for statistical analysis (Supplementary Material 4).

BloodSpot[33] is an online open data platform with data from the Cancer Genome Atlas (TCGA) and Gene Expression Omnibus (GEO) databases that provides gene expression profiles and gene traits in healthy and malignant hematopoiesis, as well as Kaplan–Meier survival maps. We downloaded the expression data of TSC22D domain family genes in AML cells and normal hematopoietic stem cells from the normal hematopoiesis with AML dataset (Supplementary Material 5) and the blood pool AML samples with normal cells dataset (Supplementary Material 6) in the BloodSpot database. We used GraphPad Prism 8 statistical software for statistical analysis.

Analysis of gene expression in AML tissues and healthy bone marrow

We downloaded the expression pictures of the TSC22D domain family genes in AML tissues and healthy bone marrow from the Valk leukemia dataset in the Oncomine database.

GEPIA2[34] is an updated and enhanced online publicly accessible database based on TCGA and Genotype-Tissue Expression (GTEx) databases for tumor and normal samples for gene expression analysis. We downloaded pictures of the expression of TSC22D domain family genes in AML tissues and normal tissues from the GEPIA2 database.

We downloaded the expression data of TSC22D domain family genes in AML tissues and healthy bone marrow samples from the Leukemia MILE Study dataset in the BloodSpot database (Supplementary Material 7). We used GraphPad Prism 8 statistical software for statistical analysis.

Survival analysis

UALCAN[35] is an online, accessible, and interactive network resource for cancer omics data analysis.

GSCALite[36] is an open, online, web-based platform for genomic cancer analysis.

We downloaded pictures of TSC22D domain family gene expression and survival analysis in AML from the GEPIA2, UALCAN, BloodSpot, and GSCALite databases.

Genetic alteration and copy number variation (CNV) analysis of the TSC22D3 gene

cBioPortal[37] is a public database to interactively explore multidimensional genomic datasets of cancers. We downloaded figures related to the genetic alterations and the survival data of AML patients grouped according to Tsc22d3 gene expression from the TCGA PanCancer dataset in the cBioPortal database.

We downloaded pictures of the CNV summary and survival analysis of the Tsc22d3 gene in AML from the GSCALite database.

Functional enrichment analysis of the Tsc22d3 gene

TRRUST (Version 2)[38] is an online, open database of human and mouse transcriptional regulatory networks.

We downloaded the gene ontology biological process, disease ontology, and KEGG pathway data associated with human Tsc22d3 transcription factor (TF) from the TRRUST database (Supplementary Material 8). Then, we managed the bar with a color gradient tool from the website (<http://www.bioinformatics.com.cn>) for visualization.

Protein–protein interaction (PPI) analysis of the Tsc22d3 gene

AnimalTFDB3.0[39] is an online database aimed at providing the most comprehensive and accurate information for animal (including human) TFs and cofactors. We downloaded a picture of the PPI analysis of the Tsc22d3 gene from the AnimalTFDB3.0 database.

STRING[40] is an online open database aimed at providing customized protein–protein networks.

We downloaded a picture of the PPI analysis of the Tsc22d3 gene from the STRING database.

Harmonizome[41] is an online database of processed datasets of gene and protein knowledge from more than 70 major online sources. We downloaded the data of PPI analysis of the Tsc22d3 gene from the Harmonizome database (Supplementary Material 9). Then, we used Cytoscape[42] analysis software to visualize the results.

Analysis of regulated kinase of the Tsc22d3 gene

We downloaded the data of the predicted Tsc22d3 kinase interactions from the Harmonizome database (Supplementary Material 10) and managed the circular heatmap tool from the website (<http://www.bioinformatics.com.cn>) for visualization.

Analysis of regulated miRNAs of the Tsc22d3 gene

TargetScanHuman 7.2[43] is an online database that predicts relationships between human miRNAs and target genes. We downloaded the data of the Tsc22d3 conserved miRNA families from the TargetScanHuman 7.2 database (Supplementary Material 11). The flower plot tool from the website (<http://www.bioinformatics.com.cn>) was used for visualization.

miRDB[44] is an online database for the prediction of miRNA target genes.

We downloaded the data of the predicted Tsc22d3 miRNAs from the miRDB database (Supplementary Material 12) and managed the flower plot tool from the website (<http://www.bioinformatics.com.cn>) for visualization.

The Encyclopedia of RNA Interactomes (ENCORI)[45] is an online open source platform for studying data on RNA interactions.

We downloaded the data of the predicted Tsc22d3 miRNAs from the ENCORI database (Supplementary Material 13) and used Cytoscape analysis software to visualize the results.

Immune Infiltration analysis of the Tsc22d3 gene

We downloaded the data of the correlation between the Tsc22d3 gene expression and immune infiltration in AML from the GSCALite database (Supplementary Material 14) and managed the correlation coefficient analysis tool from the website (<http://www.bioinformatics.com.cn>) for visualization.

GEPIA2021[46] is an online database for tumor immune invasion analysis. The CIBERSORT algorithm was used to explore the correlation between Tsc22d3 gene expression and immune infiltration in AML.

Abbreviations

acute myeloid leukemia : AML; Cancer Cell Line Encyclopedia: CCLE; copy number variation: CNV; Kyoto Encyclopedia of Genes and Genomes: KEGG; Human Protein Atlas: HPA; Cancer Genome Atlas: TCGA; Gene Expression Omnibus: GEO; Genotype-Tissue Expression: GTEx; transcription factor: TF; protein–protein interaction : PPI; Encyclopedia of RNA Interactomes: ENCORI; glucocorticoid-induced leucine zipper : GILZ.

Declarations

Ethics approval and consent to participate

This study was approved by the Academic Committee of First Center Clinic College of Tianjin Medical University and conducted according to the principles expressed in the Declaration of Helsinki.

Consent for publication

This study was published with the consent of the Academic Committee of clinical College of The First Center of Tianjin Medical University.

Availability of data and materials

The datasets provided for this study can be found and accessed in online databases. These data are also provided in the Supplementary Materials.

Competing interests

All authors declare that there are no competing interests.

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Authors' contributions

XQX: conceptualization, data analysis, and writing; XJ and JXW: data analysis, and writing; RS, XX, MZ, DNX: editing the article; MFZ: supervision of the project and reviewing, writing, and editing of the article. All authors revised the article.

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XQX is currently a doctoral student in the First Center Clinic College of Tianjin Medical University and work at Fenyang Hospital of Shanxi Province.

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Figures

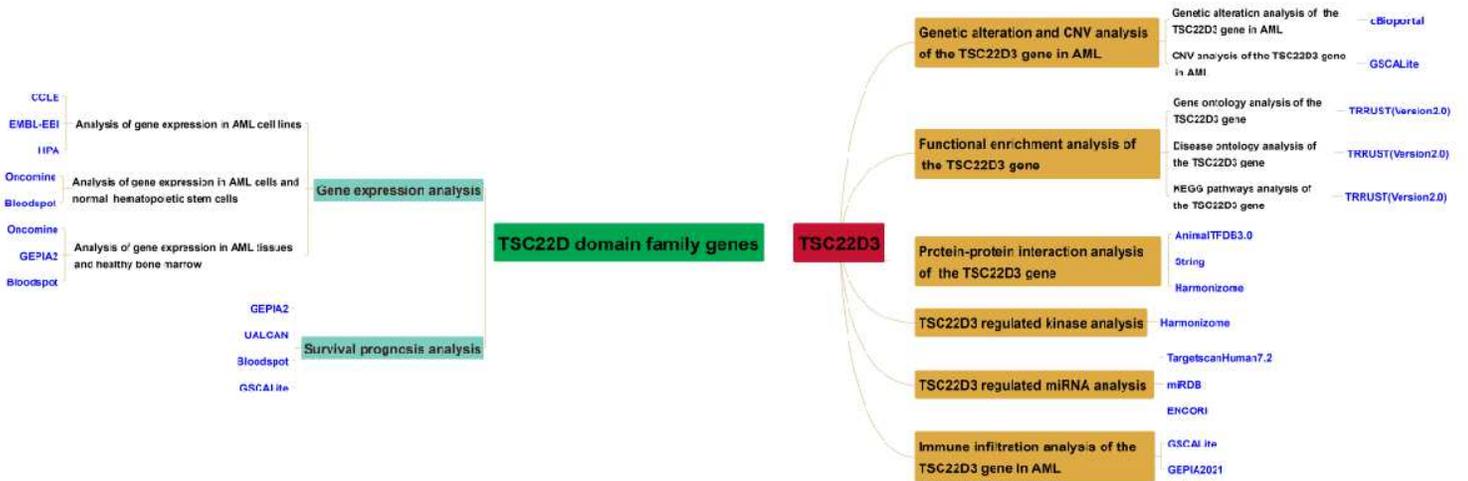


Figure 1 A mind map of the present study. Data were obtained from the TCGA and GEO databases to analyze the expression and clinical significance of the TSC22D domain family genes in AML.

Figure 1

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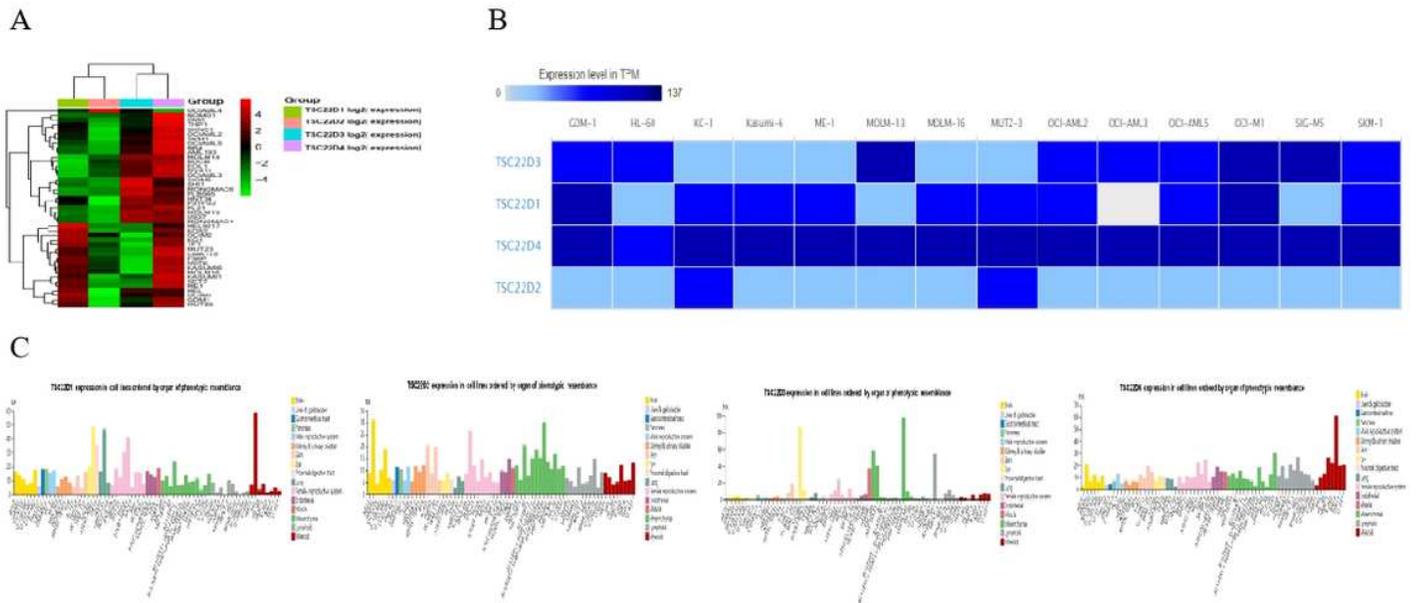
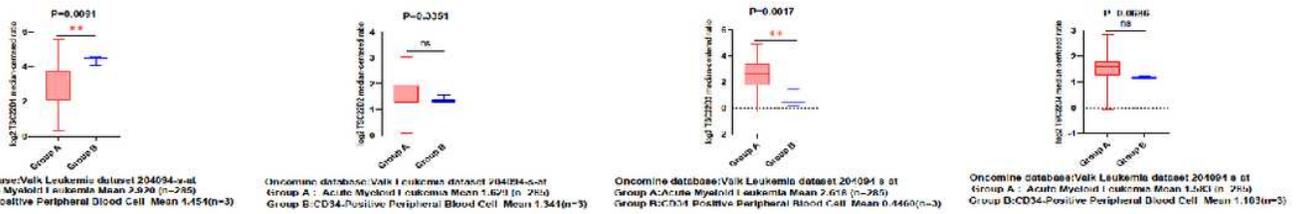


Figure 2. Analysis of TSC22D domain family gene expression in AML cell lines. (A): Heatmap of TSC22D domain family gene expression in forty-four AML cell lines. The red band represents high expression, the black band represents medium expression, and the green band represents low expression. (B): Bar chart of TSC22D domain family gene expression in fourteen AML cell lines. The deep blue band represents high expression, the blue band represents medium expression, and the white band represents low expression. (C): Bar graphs of TSC22D domain family gene expression in human cancers, including five AML cell lines.

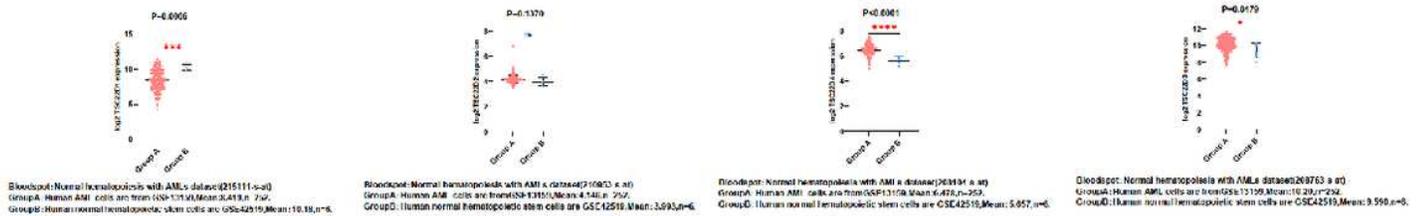
Figure 2

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A



B



C



Figure 3. Analysis of TSC22D domain family gene expression in normal hematopoietic stem cells and AML cells. (A): Boxplots displaying the expression levels of the TSC22D domain family genes, analyzed by using the Valk Leukemia dataset from the OncoPrint database. (B): The treemaps display the expression levels of the TSC22D domain family genes. Analysis was performed by using GEO data from normal hematopoiesis and AML datasets in the BloodSpot database. (C): Violin diagrams displaying the expression levels of the TSC22D domain family genes. Analysis was performed by using GEO and TCGA data from the Bloodpool AML samples with a normal cell dataset in the BloodSpot database. (*P<0.05, **P<0.01, ***P<0.001, ****P<0.0001, ns means no statistical significance).

Figure 3

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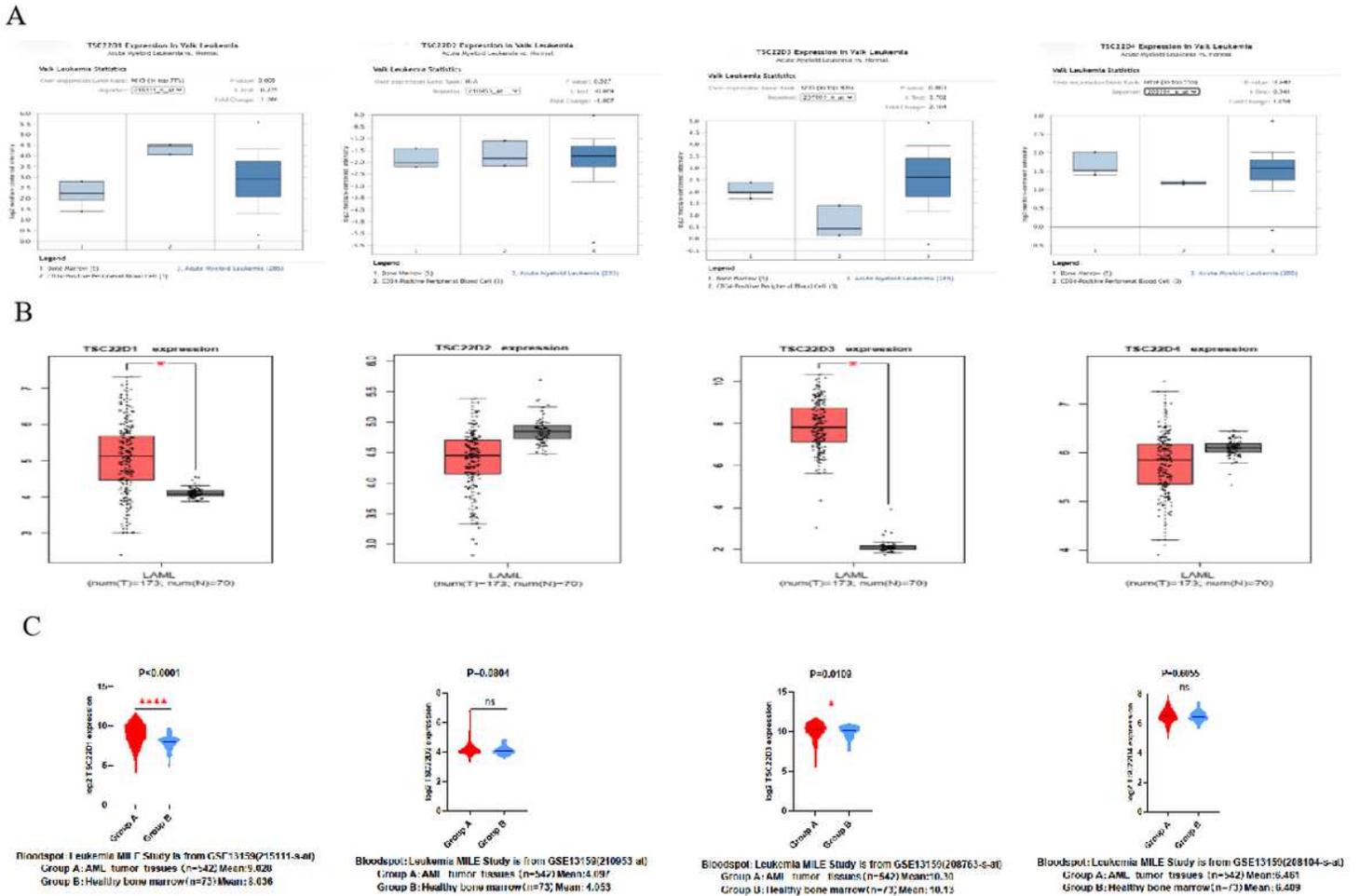


Figure 4. Analysis of TSC22D domain family gene expression in healthy bone marrow samples and AML tissues. (A): The expression levels of the TSC22D domain family genes in healthy bone marrow and AML tissues were analyzed by using the Valk leukemia dataset from the OncoPrint database. Statistical analysis was represented by t test, p value, and fold change. (B): Boxplot displaying the expression levels of the TSC22D domain family genes in healthy bone marrow samples and AML tissues from the TCGA data in the GEPIA2 database. (C): Violin diagram showing the expression levels of TSC22D domain family genes in healthy bone marrow samples and AML tissues from the GEO data in the Leukemia MILE Study dataset from the BloodSpot database (*P<0.05, **P<0.01, ***P<0.001, ****P<0.0001, NS means no statistical significance).

Figure 4

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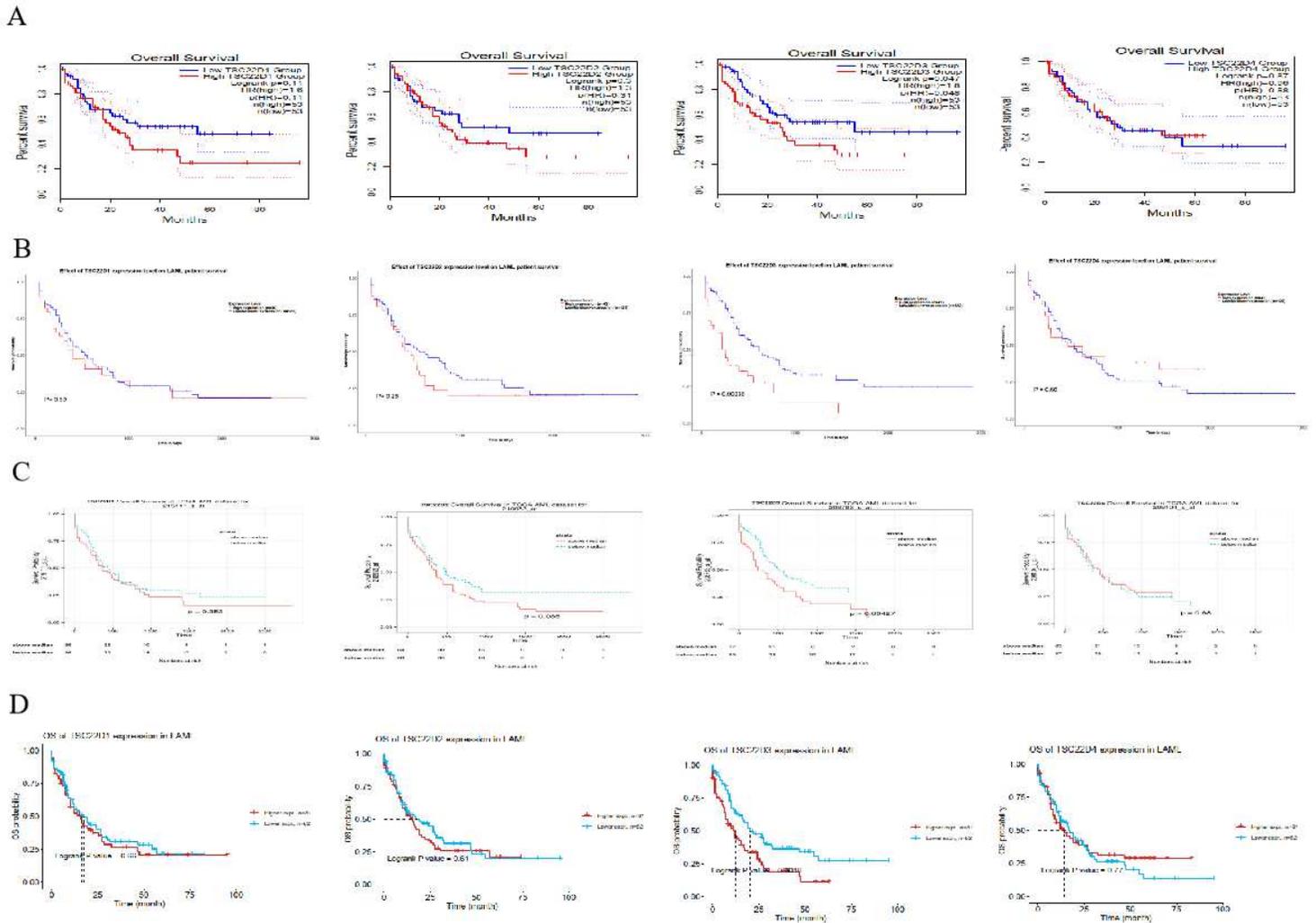


Figure 5. The impact of TSC22D domain family gene expression on the survival of AML patients. (A): The prognostic value of the expression of the TSC22D domain family genes in AML patients was analyzed by using the GEPIA2 database. (B): The prognostic value of the expression of the TSC22D domain family genes in AML patients was analyzed by using the UALCAN database. (C): The prognostic value of the expression of the TSC22D domain family genes in AML patients was analyzed by using the BloodSpot database. (D): The prognostic value of the expression of the TSC22D domain family genes in AML patients was analyzed by using the GSCALite database.

Figure 5

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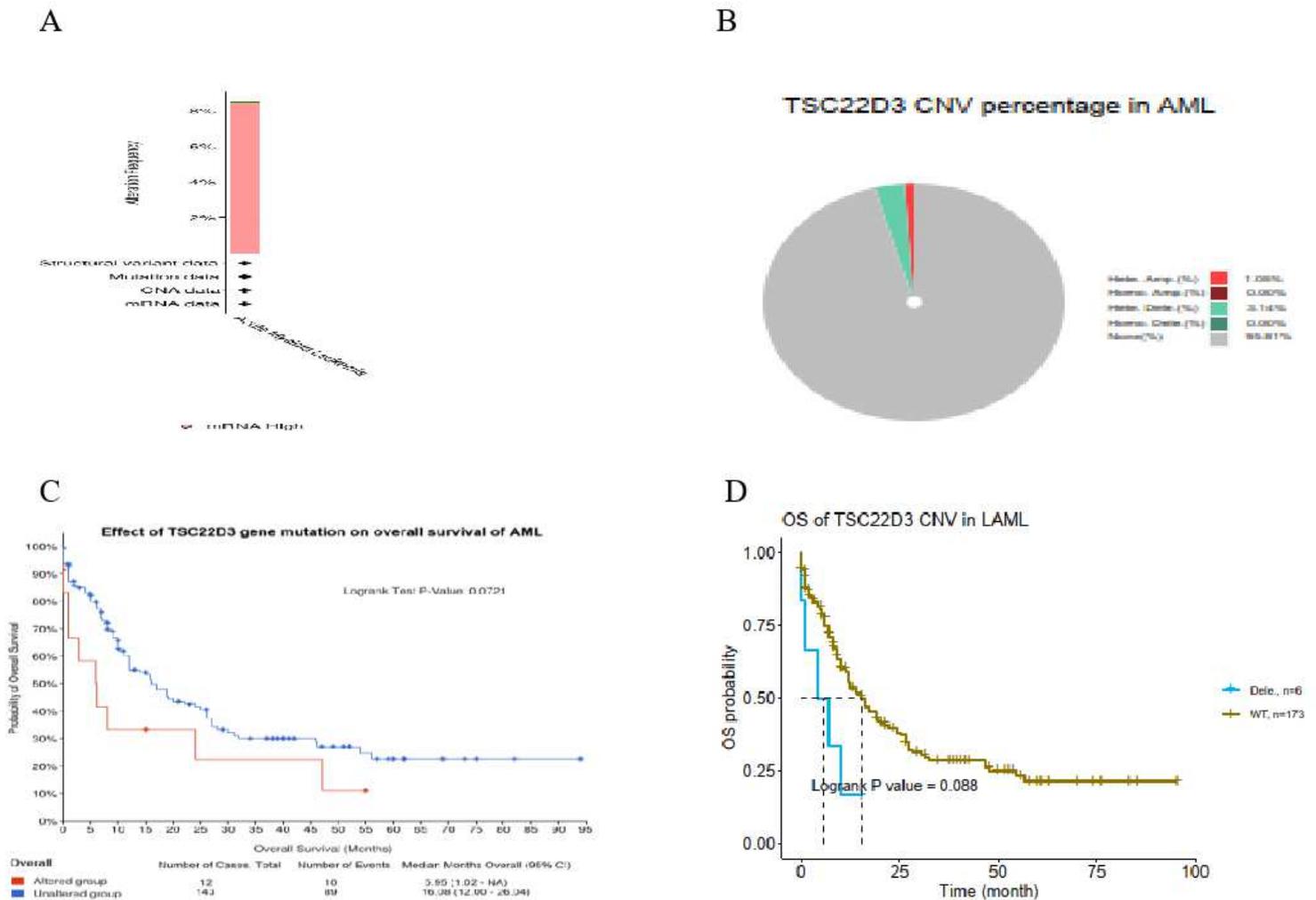
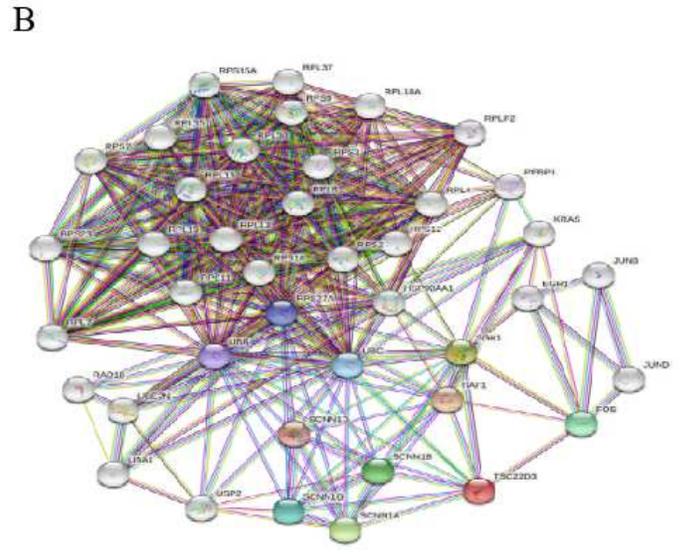
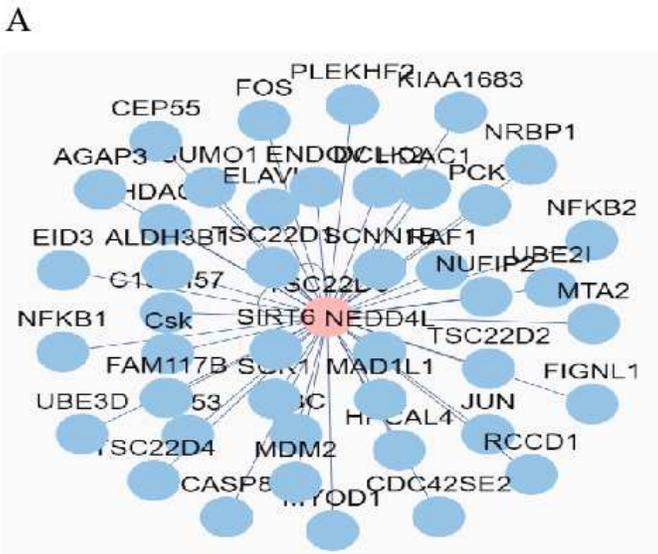


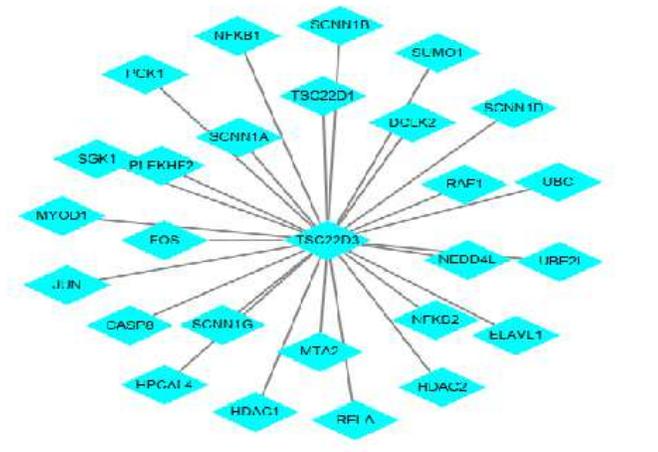
Figure 6. The mutation profiles and summary of CNV of the TSC22D3 gene and the impact on the survival of AML patients. (A) The mutation rate of the TSC22D3 gene based on 165 AML samples from the TCGA PanCancer dataset was analyzed by using the cBioPortal database. (B) The summary of CNVs of the TSC22D3 gene in AML was analyzed by using the GSCALite database. (C) The mutation of the TSC22D3 gene impacted AML survival and was analyzed using the cBioPortal database. (D) The CNV of the TSC22D3 gene on the survival of AML patients was analyzed by using the GSCALite database.

Figure 6

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B
Harmonizome database: Protein-Protein Interactions



C
Predicted TSC22D3 kinase interactions (KCA) from harmonizome database

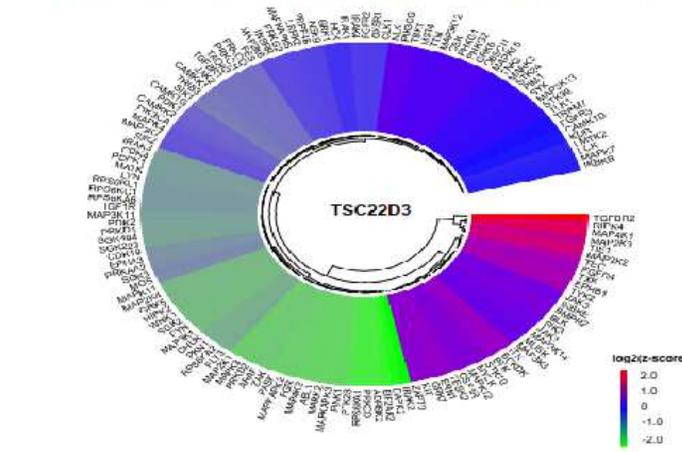


Figure 8. Analysis of PPIs and regulated kinases of TSC22D3. (A) The PPIs of TSC22D3 according to the AnimalTFDB3.0 database. (B) The PPIs of TSC22D3 according to the STRING database. (C) The PPIs of TSC22D3 according to the Harmonizome database. (D) The heatmap of the TSC22D3-regulated kinases generated by using the Harmonizome database. The red band represents high log₂(z score), the blue band represents medium log₂(z score), and the green band represents low log₂(z score).

Figure 8

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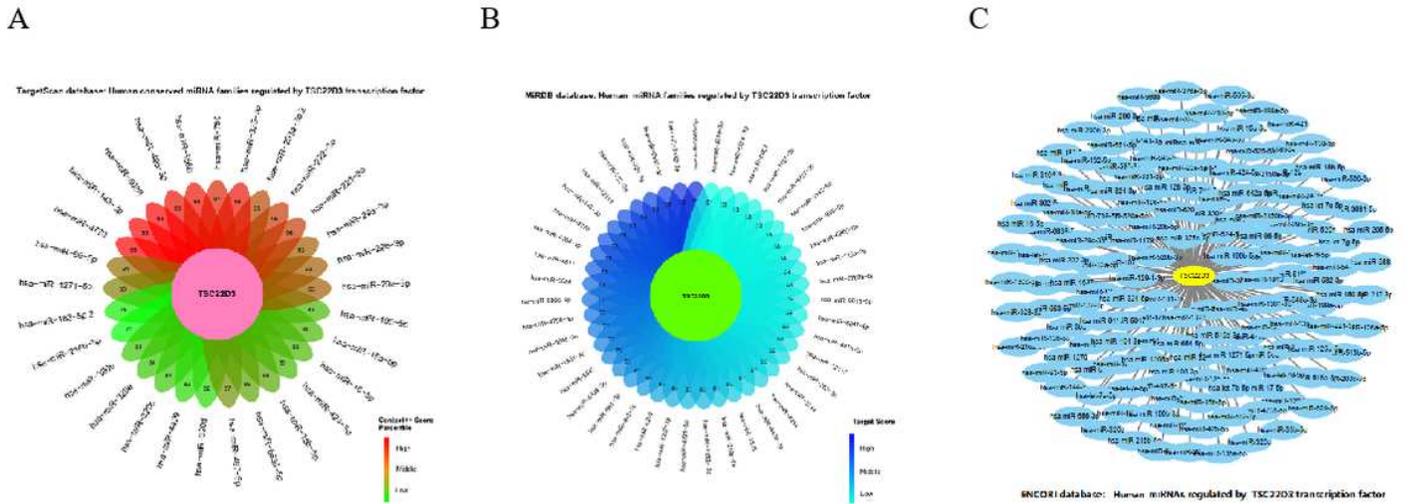


Figure 9. Analysis of miRNAs regulated by TSC22D3. (A) The flower plot of TSC22D3-regulated miRNAs generated by using the TargetScan database. The red petals indicate a high context++ score percentile, and the green petals indicate a low context++ score percentile. (B) The flower plot of the TSC22D3-regulated miRNAs generated by using the MiRDB database. The dark blue petals indicate a high target score, and the cyan petals indicate a low target score. (C) The network diagram of the TSC22D3-regulated miRNAs was generated by using the ENCORI database.

Figure 9

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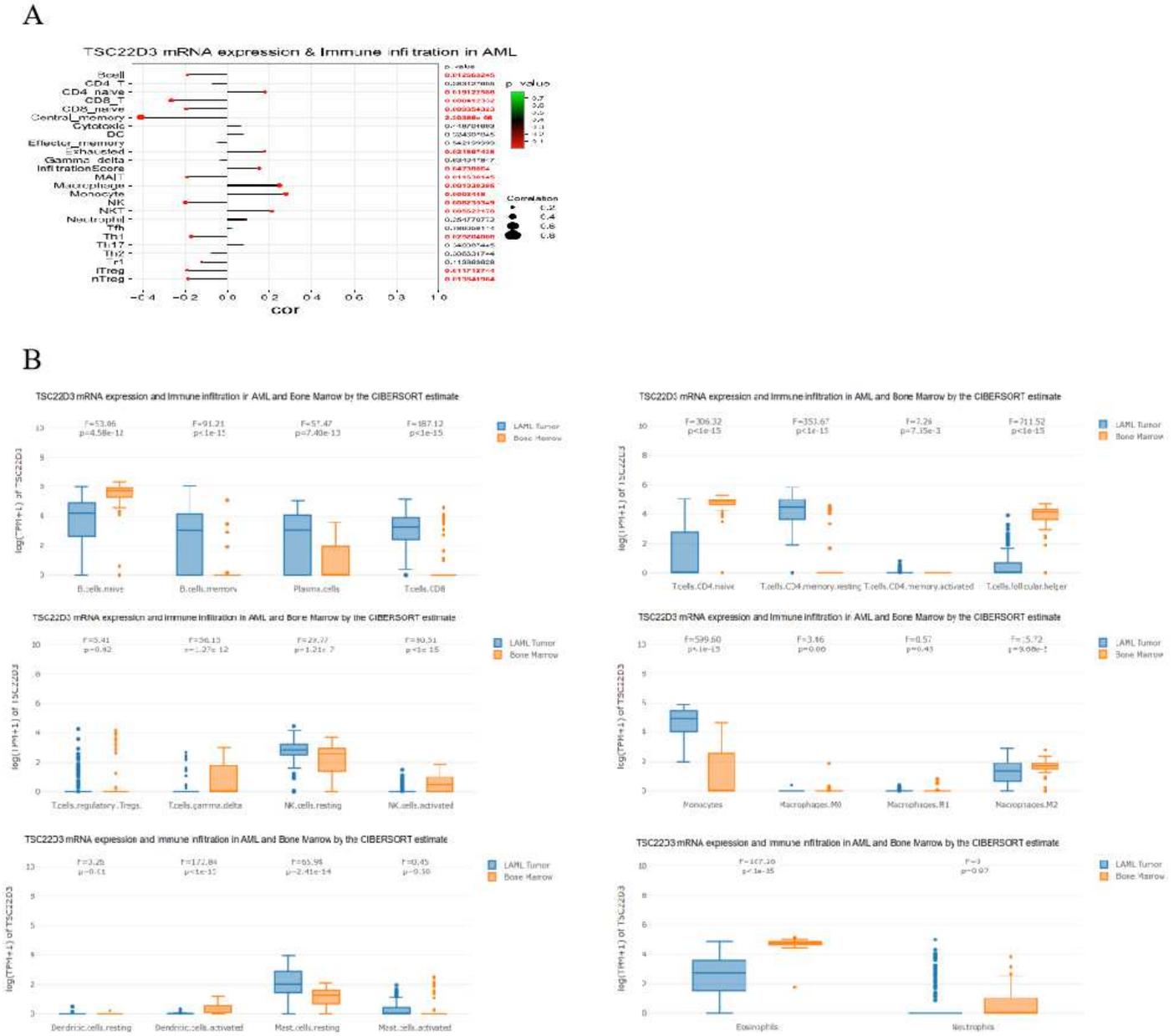


Figure 10. Immune infiltration analysis according to TSC22D3 expression in AML. (A): The correlation of TSC22D3 expression and immune infiltration in AML was analyzed by using the GSCALite database. (B): The CIBERSORT algorithm was used to analyze the relationship between TSC22D3 expression and immune infiltration in AML based on the GEPIA2021 database.

Figure 10

See image above for figure legend

Supplementary Files

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