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ORIGINAL ARTICLE

Comprehensive analysis of Ferroptosis related molecular TFRC in PAAD and its relationship with the tumour microenvironment and patient prognosis

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Abstract.

Background: Pancreatic adenocarcinoma (PAAD) is a highly fatal malignancy worldwide. Recent studies have reported that ferroptosis is a novel type of non-apoptotic cell death. Despite evidence that TFRC, an important participant in intracellular iron transport, is involved in cancer progression, the role of TFRC in PAAD remains unclear.
Methods: The expression levels of TFRC were downloaded from The Cancer Genome Atlas (TCGA) and GTEx databases, the TFRC expression patterns and immunological effects were assessed by pan-cancer analysis, and the results were validated in the GEO and Oncomine datasets. Then, the expression data of TFRC in PAAD were assessed by prognostic survival analysis (overall survival (OS), disease-free survival (DFS), progression-free survival (PFS), and disease-specific survival (DSS)). Subsequently, the correlation of TFRC with factors that reflect the tumour microenvironment, including immune scores, immune checkpoints, tumour-infiltrating immune cells, tumour mutation burden (TMB), microsatellite instability, ferroptosis level, and m6A levels, was assessed. In addition, the clinicopathological features of TFRC and the risk score in PAAD were combined to establish a clinical nomogram and construct a calibration curve for validation. Finally, gene set enrichment analysis was performed to visualize the signalling pathways by which TFRC affects PAAD.
Results: The expression of TFRC was significantly increased in PAAD tissues compared to normal pancreatic tissues. Kaplan-Meier survival analysis showed that PAAD patients with high levels of TFRC had shorter survival than did those with lower levels of TFRC. TFRC expression was associated with different tumour-infiltrating immune cells in PAAD and with factors such as lymph node metastasis and a history of alcohol consumption. The results of the univariate and multivariate Cox regression analyses showed an association of TFRC with prognosis in PAAD patients. GSEA suggested that TFRC acts on pathways related to endothelial cell proliferation and blood vessel endothelial cell migration.
Conclusions: TFRC is a potential prognostic indicator and a novel therapeutic target for human PAAD.
Keywords: TRFC, PAAD, survival, prognosis, tumour microenvironment

Introduction

Pancreatic adenocarcinoma (PAAD) is one of the deadliest gastrointestinal malignancies, with approximately 338,000 pancreatic cancer patients surveyed worldwide in 2012 [1]. Difficulties in early detection and susceptibility of PAAD to metastasis lead to poor prognosis of pancreatic cancer [2]. One study reported that the incidence of pancreatic cancer continues to rise globally, and it is predicted that pancreatic cancer will be the second leading cause of cancer-related death in the US by 2030 [3]. The strong chemoresistance of PAAD poses significant challenges to extending patient survival, and effective treatments are still limited [4-5]. Therefore, there is

an urgent need to explore the molecular targets with regard to the prognosis and treatment if PAAD patients.

TFRC, also known as CD71, is a membrane protein that regulates intracellular iron transport. Recently, an increasing number of studies have shown that TFRC overexpression in certain malignancies plays an important role in tumour development [6-7]. TFRC promotes epithelial ovarian cancer cell proliferation and metastasis [7], and epigenetic silencing of the TFRC gene reduces the growth and survival of hepatocellular carcinoma (HCC) cells [8]. However, the role of TFRC in PAAD remains unclear.

In this study, we observed a significant increase in the expression of TFRC in PAAD patients from The Cancer Genome Atlas (TCGA), Oncomine and GEO databases. This overexpression is associated with a poor prognosis of PAAD. At the same time, our study showed that high TFRC expression was significantly associated with lymphnode metastasis. Furthermore, we performed an enrichment analysis and visualized the signalling pathways enriched by TFRC in PAAD.

Materials and Methods

Data source and processing

We performed mapping from the TCGA (https://cancergenome.nih.gov/) [9] and Genotype Tissue Expression (GTEx) (https://gtexportal.org/) databases, and TFRC expression in multiple cancers was analysed using R software v4.0.3 [10]. We also used the Oncomine database [11] to further validate the difference in TFRC expression between PAAD patients and healthy individuals, and a P-value cut-off value of 0.05 was considered statistically significant.

Survival analysis

We analyzed the relationship between TFRC expression and overall survival (OS) and disease-specific survival (DSS) in multiple cancers by using the R packages survminer with samples from the TCGA database. The relationship between OS, DSS, progression-free survival (PFS) and disease-free survival (DFS) with in PAAD using univariate Cox regression analysis and P values, risk coefficient, hazard ratio (HR) and confidence intervals are visualized by forest plots. We considered $P < 0.05$ in the univariate Cox to be statistically significant. In addition, we investigated the association between TFRC expression and clinicopathological features with Fisher's exact test and drew a three-line table.

Analysis of the tumour microenvironment

To determine the effect of TFRC on immune infiltration in PAAD, we conducted an immune score assessment and immune checkpoint analysis using the TCGA dataset of PAAD patients with R software by R (v4.0.3) [12-15]. Correlation of TFRC expression with various immune cells was performed with the R package ggstatsplot [16]. Next, we analysed the correlation of tumour mutation burden (TMB) and microsatellite instability with TFRC expression in PAAD using the R software package fmsb. In addition, we performed apoptosis and RNA methylation modification (m6A) analysis and further validated our results with the PAAD patient dataset from the GEO database [17-18]. We considered a P-value cut-off of 0.05 to be statistically significant. All the above analyses were implemented using the R software packages ggplot2 and pheatmap.

Gene set enrichment analysis (GSEA)

GSEA is a computational method that correlates gene expression data with diseases or drug response [19]. In this study, all the specimens from PAAD patients were sorted by TFRC expression levels classified into high and low expression groups based on the median amount of TFRC expression. Genome arrangements were set to 100 times and

filtered by their nominal p values and normalized enrichment scores (NESs). The R software package limma, h.eg.db, clusterProfiler, enrichplot and gglot2 were applied to visualize the data. A P-value of 0.05 was statistically significant.

Statistical analysis

Univariate and multivariate Cox regression analyses were used to assess the prognostic significance of TFRC expression in PAAD patients. We presented the variable P values, risk coefficient HRs and confidence intervals with the R package forestplot and using forest plots. The results of multivariate Cox proportional risk analysis were produced using the "rms" R package, and nomograms were generated. The nomogram can not only predict the total 3-year relapse rate but also graphically represent these factors, and the prognostic risk for individual patients can be determined by calculating the points associated with each risk factor [20-21].

Results

Pan-cancer expression pattern and survival correlation

We analysed the TFRC expression data from the TCGA and GTEx databases and showed that TFRC was highly expressed in multiple tumour tissues, especially in PAAD tissues (Figure 1A). To further validate our findings from these databases, we used the Oncomine database for analytical comparison and reported significant differences in TCGA expression between normal tissues and tumour tissues from PAAD patients (Figure 1B). These results suggest that TFRC expression is upregulated in PAAD patients. Next, we investigated the prognostic significance of TFRC expression in human tumours. We performed OS analysis using the "survival" package in the R project and found that high expression of TFRC predicted poor PAAD prognosis (Figure 1C) (p=0.020) with decreased OS with increasing TFRC expression; however, this correlation was not observed in other tumours (Figure 1E-G). Therefore, we further analysed the role of TFRC in PAAD using the Cox regression model. The results showed that TFRC is a high-risk factor for PAAD occurrence (Figure 1H).

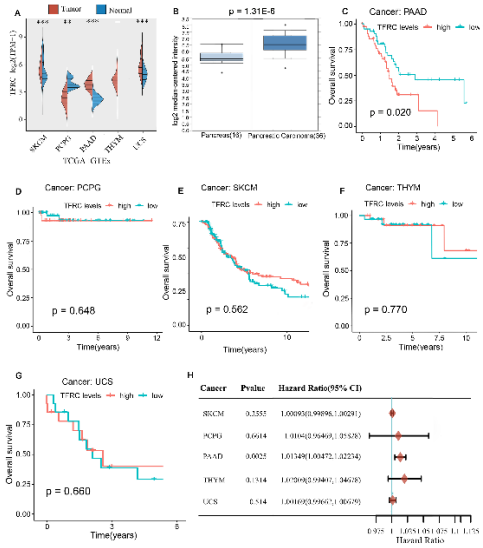

Figure 1. The expression of TFRC in different cancers and comparison of OS survival curves of TFRC high and low expression in different cancers. A: TFRC expression of different tumor types in the TCGA and GTEx database. B: TFRC level in PAAD tissues compared to normal tissues in the Oncomine database. (C-G) Comparison of OS survival curves of TFRC high and low expression in PAAD(C), PCPG(D), SKCM (E), THYM (F) and UCS(G). H: Prognostic HR of TFRC in different cancers for OS.

We then studied PAAD samples using different survival analyses. Kaplan-Meier analysis of PAAD samples from the TCGA database revealed that high levels of TFRC were significantly associated with OS (Figure 2A) (p = 0.025) but not with PFS (p= 0.318).

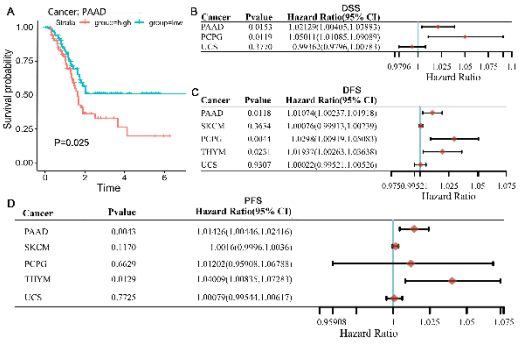

Figure 2. Comparison of DSS, DFI and PFI survival curves of TFRC high and low expression in different cancers. A:

High TFRC expression had poor DSS in PAAD. (B–D) Prognostic HR of TFRC in different cancers for DSS (B), DFI (C), PFI (D).

However, when performing Cox analysis, we found that the P values of DSS, DFS, and PFS derived from Cox analysis were less than 0.05, with HR values greater than 1 (Figure 2B-D), indicating that TFRC expression was associated with the survival of PAAD patients, was a high-risk factor for PAAD and could serve as a prognostic biomarker of PAAD.

Correlation between TFRC and the clinical characteristics of PAAD patients

We used the association between TFRC expression and different clinicopathological features of PAAD using 178 samples from the TCGA database. We divided these samples into high and low expression groups based on the median value of TFRC expression and then analyzed the association between TFRC expression levels and clinical characteristics using Fisher's exact and chi-squared tests. We found that there was no significant relationship between TFRC expression levels and survival, smoking, tumour grade (Figure 3A, p> 0.05), sex, or T stage (Figure 3A, p <0.1).

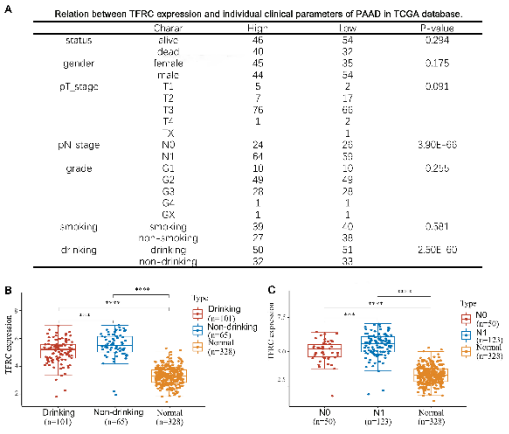

Figure 3. Correlation of TFRC expression and prognosis in PAAD with different clinicopathological factors. A: Relation between TFRC expression and prognosis in PAAD with different clinicopathological factors in in TCGA database. (B-C) TFRC expression was remarkably correlated with PAAD patients'nodal metastasis (B) and a history of alcohol consumption(C).

but a significant relationship with a history of alcohol consumption and lymph node metastasis (Figure 3B-C, p <0.001). Meanwhile, a higher level of lymph node metastasis corresponded with increased TFRC levels in PAAD patients, whereas PAAD patients with a history of alcohol consumption had lower TFRC levels in vivo than those with no history of alcohol consumption.

TFRC expression was correlated with the immune infiltration of the tumour microenvironment in PAAD

We performed an analysis of the correlation between the expression level of TFRC and the level of immune infiltration in PAAD. First, we analyzed differential expression of immune checkpoint genes in high and low TFRC expression samples and we found that the levels of CD274 (p <0.001), CTLA4 (p <0.01), HAVCR2 (p <0.01), PDCD1LG2 (p <0.01) and TIGIT (p <0.01) were significantly higher in the TFRC high expression group, suggesting that high expression of TFRC promoted the inhibition of immune cell function and increased the ability of tumours to evade the immune system (Figure 4A).

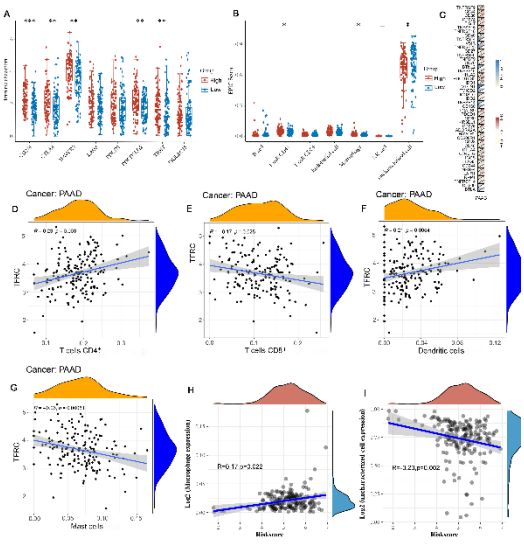

Figure 4. Correlation analysis of TFRC level with immune checkpoint, immune scores and immune cells infiltration levels in human PAAD. A: Correlation analysis of TFRC level and immune checkpoint by TCGA database. B: Correlation analysis of TFRC level and immune scores in human PAAD. C: gene co-expression heatmap. (D-I) Correlation analysis of

TFRC level immune cells infiltration levels. TFRC expression in PAAD tissues correlates with tumor immune infiltration levels of CD4+ T cells (D), CD8+ T cells (E), dendritic cells (F), mast cells (G), macrophages (H) and uncharacterized cells (I).

We also tested our results obtained above with a gene coexpression heatmap. Next, we analysed the differences in the immune cell counts in specimens with high and low TFRC expression using immune scores and showed that the CD4+ content ($p<0.05$), macrophage count ($p<0.05$) and uncharacterized T cells ($p<0.05$) were correlated with TFRC expression (Figure 4B). Finally, we evaluated the correlation of TFRC expression levels with various immune cells using samples from the TCGA database. In PAAD, TFRC expression levels were compared with the counts of the following cells: CD4+ T cells ($R=0.23$, $p<0.01$) (Figure 4D), CD8+ T cells ($R= -0.17$, $p<0.05$) (Figure 4E), dendritic cells ($R=0.21$, $p<0.01$) (Figure 4F), mast cells ($R= -0.26$, $p<0.001$) (Figure 4G), macrophages ($R=0.17$), $p<0.01$) (Figure 4H) and uncharacterized cells ($R=0.23$, $p<0.01$) (Figure 4I) The above analysis suggests that TFRC can act as an immune infiltration modulator in PAAD.

Effects of TFRC expression on the tumour microenvironment

It has been found that TMB can be predicted as a novel effective biomarker for immunotherapy in various tumours. Therefore, we used the TCGA dataset to analyse the relationship between TFRC expression and TMB in PAAD. The results showed that the TMB was positively associated with TFRC expression levels (Figure 5A, $p <0.01$). Next, we investigated the relationship between microsatellite instability and TFRC expression levels but found no correlation between the two (Figure 5B, $p> 0.05$). In addition, we analysed differences in the expression of molecules associated with ferroptosis, such as CDKN1A, HSPA5, EMC2, SLC7A11 and NFE2L2, and found that they were significantly associated with TFRC expression (Figure 5C, $p <0.001$). Next, we identified a significant correlation between TFRC expression and the m6A molecules VTRMA, RBM15, ZC3H13, YTHDC1 and YTHDC2 (Figure 5D, $p <0.001$). Finally, we further validated our results from the GEO database (Figure 5E-F).

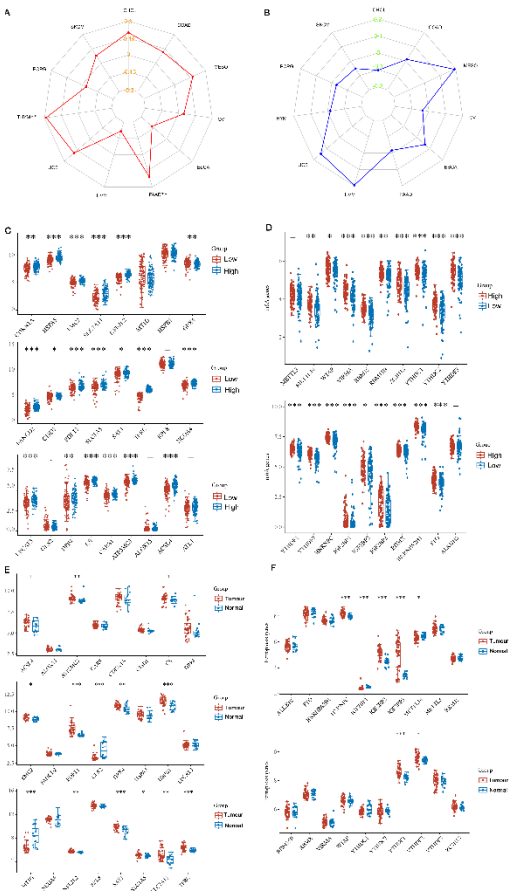

Figure 5. Correlation analysis of TFRC level with TMB, microsatellite instability, the molecules associated with ferroptosis and the m6A molecules in human PAAD. A: The relationship between TFRC expression and TMB in PAAD. B: The relationship between microsatellite instability and TFRC expression levels. C: The expression of molecules associated with ferroptosis were associated with TFRC expression. D: Correlation between TFRC expression and the m6A molecules. (E-F) Correlation analysis of TFRC level with the molecules associated with ferroptosis (E) and the m6A molecules (F) across human PAAD and normal tissue using the GEO database.

Effect of TFRC expression and clinical factors on prognosis

We evaluated 178 PAAD samples from the TCGA database to determine the

correlation of TFRC expression and clinicopathological factors with OS using univariate Cox regression. The results showed a significant correlation of TFRC (HR=1.44, p<0.01), age (HR=1.02, p<0.01), pT_stage (HR=1.55, p<0.05), pN_stage (HR=2.15, p<0.01), grade (HR=1.45, p<0.05) and primary tumour (HR=1.39, p<0.05) with OS of PAAD (Figure 6A). Notably, pN_stage corresponded with the highest risk. Multivariate Cox regression analysis of OS suggested that pT_stage and primary tumour could act as independent prognostic factors (Figure 6B). Next, we generated an OS nomogram in the group that predicted the 1-, 2- and 3-year OS rates of PAAD patients (Figure 6 C). To further verify our results, we also generated calibration curves for the nomogram model. The ideal curve is indicated by diagonal dashed lines, while the nomogram of the observed 1-year OS rate is indicated by red lines (Figure 6D).

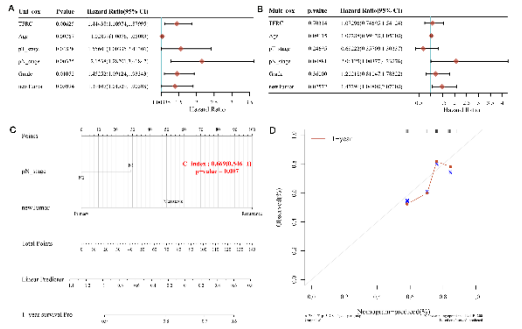

Figure 6. Univariate and multivariate Cox analyses of clinical parameters and a nomogram for overall survival in PAAD. A: Correlation of TFRC expression and clinicopathological factors with OS by univariate Cox regression. B: Correlation of TFRC expression and clinicopathological factors with OS by multivariate Cox regression. C: A nomogram for overall survival in PAAD. D: Calibration curves for the nomogram model.

TFRC affects signalling pathways associated with the occurrence and development of PAAD

We classified the PAAD samples into high and low expression groups based on the median expression value of TFRC, and GSEA of the PAAD samples revealed the five most significantly enriched signal pathways. The results showed that TFRC regulates PAAD occurrence through signalling pathways associated with endothelial cell proliferation, blood vessel endothelial cell migration, G1/S transition of mitotic cell cycle, cell migration involved in sprouting angiogenesis and vascular endothelial cell proliferation (Figure 7).

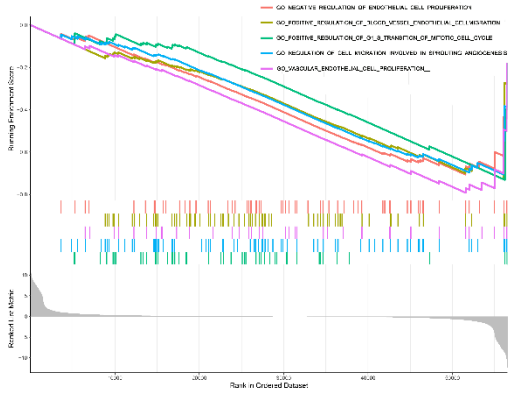

Figure 7. Molecular function of TFRC in PAAD.A-D: Gene ontology (GO).

Discussion

Pancreatic cancer is one of the deadliest tumours of all cancers and has a poor prognosis. Therefore, it is important to understand the prognostic factors and molecular mechanisms underlying the occurrence and development of PAAD in order to promote effective treatment. Ferroptosis is a novel mode of non-apoptotic cell death. Increasing studies have confirmed that ferroptosis plays an important role in cancer progression. Many studies have investigated the role of ferroptosis-related genes in pancreatic cancer. Xu et al. reported that PTGS2 and SQLE have cancer-promoting capacity in PAAD [22]. Another study suggested that the combination of artesunate with GRP78 inhibition is a novel method for effectively killing KRAS-mutant pancreatic ductal adenocarcinoma cells [23]. Subsequent studies identified microsomal glutathione-S-transferase 1 (MGST1) as a target gene of NRF2 involved in inhibiting the progression of ferroptosis in pancreatic cancer cells [24].

Although several studies have revealed the pathophysiological role of ferroptosis-related genes in pancreatic

cancer, it is more likely that there are dozens of combinations of ferroptosis-related genes that affect PAAD, especially in PAAD prognostic studies. TFRC is a membrane protein involved in mediating intracellular iron transport. In this study, we analysed for the first time the prognostic value of TFRC expression in PAAD and its correlation with immune infiltration and the tumour microenvironment using specimens from the TCGA, GTEx, Oncomine and GEO databases.

In this study, we confirmed that the expression of TFRC was significantly increased in tumour tissues from PAAD patients compared with normal tissues.

In addition, overexpression of TFRC correlated with lymph node metastasis in PAAD. Our results suggest an important role of TFRC in the occurrence and development of PAAD. Our results are the same as those described in previous work [6]. TFRC contributes to the proliferation and metastasis of epithelial ovarian cancer cells [7]. These results strongly suggest that TFRC acts by promoting tumour proliferation and metastasis.

The results from the survival analysis showed that patients with high TFRC expression exhibited significantly worse OS, DSS, PFE and DFS than those with low TFRC expression (Figure 1C), which is consistent with previous findings. Epigenetic silencing of the TFRC gene was shown to reduce the growth and survival of HCC cells [8].

It was previously reported that TFRC can regulate the tumour microenvironment. Using an immunocorrelation analysis, we found that TFRC expression is positively associated with macrophages and dendritic cells in PAAD, suggesting that TFRC is involved in the regulation of immune cells in the tumour microenvironment in PAAD. Furthermore, according to our findings, TFRC expression is associated with the expression of multiple immune checkpoint-related genes in PAAD. We hypothesized that TFRC is able to promote immune escape of tumour cells. Consistent with our findings, Sun Jiarui et al. stated that the iron restriction imposed by cancer cells drives the polarization of macrophages toward an immunosuppressive M2 phenotype, thereby inhibiting antitumor immunity [25]. We further discovered a significant association of TFRC expression with TMB, ferroptosis, and m6A in PAAD. Univariate and multivariate Cox analyses showed that low TFRC expression was associated with a good prognosis in PAAD patients, indicating that TFRC may be a potential prognostic indicator for PAAD patients.

Using GSEA, TFRC was observed to regulate PAAD occurrence through multiple pathways (endothelial cell proliferation and blood vessel endothelial cell migration) that have been implicated in cell proliferation and migration. Therefore, we hypothesized that TFRC plays a role in PAAD progression by promoting cancer cell proliferation and metastasis. Although the specific mechanism of action of TFRC in PAAD in this study was not fully clarified and but was rather intimated based on a pure raw data analysis (which may have some limitations), we provide a novel direction for exploring the role of TFRC in PAAD. Taken together, our findings suggest that TFRC is a potential prognostic indicator and a novel therapeutic target for PAAD.

Conclusion

In conclusion, we observed differential expression of TFRC and its associated genes between tumour tissue and normal pancreatic tissue in PAAD patients, suggesting the potential for TFRC as a prognostic indicator and novel therapeutic target in PAAD.

Data Availability

Data collection and processing were performed according to the policies of the TCGA project, GTEx, Oncomine and GEO databases.

Author Contributions

Junshan Xu. Designed this study. Junshan Xu. carried out data acquisition and analysis. Junshan Xu, Wenhua Hu. wrote the manuscript and contributed to preparing and making figures and tables. All authors read and approved of the final manuscript.

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Conflict of Interest

The author(s) declared that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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