Original Article:

Assessment of RPS14 in Kidney Renal Clear Cell Carcinoma Using Bioinformatics Analysis

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Abstract:

Background and aim:
RPS14—a structural gene for ribosomal protein S14—is a critical component of the small ribosomal subunit, and its overexpression has been indicated in many cancers including Kidney Renal Clear Cell Carcinoma (KIRC). Early diagnosis and accurate prognostic assessment remain critical challenges for the treatment of KIRC. This study aimed to investigate differential RPS14 gene expression using bioinformatics tools to examine RPS14 RNA sequencing across pathological parameters, such as cancer stage, sex, age, and genetic alterations of RPS14 in KIRC samples.

Methods:
Comprehensive data analysis was performed using four publicly available cancer OMICS data analysis platforms to examine RPS14 RNA sequencing across KIRC pathological stages, sex, age, and subtype. Additionally, the association between differential RPS14 expression and the KIRC tumour microenvironment was assessed. Furthermore, an analysis of the cancer genomic database was performed to analyse genomic alterations across a set of patients with KIRC.

Results:
More than half of the nurses (55.7%) demonstrated excellent overall competency, with the highest scores in knowledge (63.3%), experience (59.5%), and attitude (58.9%) domains. The skills domain showed the lowest percentage of excellence (48.1%). Competency level was significantly associated with marital status (p = 0.005) and attendance at patient safety training (p = 0.008), whereas age and experience were not significantly associated with both.

Conclusion:
RPS14 was highly upregulated in tumour samples compared to normal tissue (p=1.0 ×10-12). Sex-specific findings revealed significant upregulation in both males (p=1.0×10-12, n=345) and females (p=1.0×10-12, n=188) compared to the normal sample (n=72). RPS14 was upregulated in Caucasians (p=1.62×10-12, n=462), African-Americans (p=1.87×10-9, n=56), and Asian (4.82×10-3, n=8) compared to normal samples. RPS14 was altered in 141 (6%) of 2281 queried patients.
Conclusions This study detected upregulated RPS14 in KIRC samples. Further validation analyses and studies are required to determine the potential role of RPS14 as a diagnostic and prognostic biomarker and therapeutic target for KIRC.

Keywords:
RPS14; cancer; kidney cancer; biomarker; renal cell carcinoma; genomic profile

Introduction:

Kidney Renal Clear Cell Carcinoma (KIRC) is the most common subtype of renal cell carcinoma. [1,2] Early diagnosis and accurate prognostic assessment remain critical challenges for the treatment of this cancer. Identification of specific biomarkers for this malignancy is crucial for diagnosis, prognosis, and improvement of treatment strategies. [3] Studies have shown that ribosomal proteins (RPs) perform multiple functions, including the regulation of cell growth, cell proliferation and differentiation, immune signalling, DNA repair, and apoptosis. [4,5] Studies have also revealed the role of RPs in development and tumorigenesis. [6]

RPS14, a ribosomal protein, has emerged as a potential prognostic marker of KIRC, particularly in association with specific genetic abnormalities. [7] RPS14 is composed of five exons and four introns, spanning approximately 5.9 kb at chromosome 5q33.[8] The RPS14 protein is composed of 151 amino acids and belongs to the S11P ribosomal protein family, which is necessary for the processing of 18S rRNA and formation of the 40S ribosomal subunit. [9,10] RPS14 is a part of the protein-making machinery of cells and is crucial for ribosome assembly and function, helping to process RNA into proteins. [8] Studies have revealed that RPS14 may play other roles in cell division, programmed cell death (apoptosis), hematopoietic stem cells (HSPCs)[11], and red blood cell development.[12,13]

Recent genomic and proteomic studies have identified RPS14 among the dysregulated genes in various cancers, including KIRC, prompting the investigation of its potential as a cancer biomarker. [14–16] RPS14 plays essential roles in cancer development through the p53 signalling pathway dysregulation glioma [15] , ribosome pathways, and survival associations in breast cancer. [6] Mutations in RPS14 are linked to diseases such as 5q-syndrome[17,18], a type of Myelodysplastic Syndrome (MDS) where deletion of chromosome 5q, including RPS14, impairs red blood cell production, causing anaemia, potentially influencing immune signalling and disease severity, and increasing the risk of acute myeloid leukaemia (AML). [13,14,18–20]

Additionally, alterations in RPS14 expression may affect transcriptional processes and metabolic regulation, such as selective translation programs and nucleolar stress responses causing KIRC. [6] Furthermore, a related congenital disorder known as Diamond–Blackfan anaemia

affects RPs, showing how ribosome defects can cause bone marrow failure. [22,23]

This study aimed to investigate the association between differential RPS14 expression and patients with KIRC and normal controls. Furthermore, gene expression profiling interactive analysis (GEPIA) and other bioinformatics tools were used to analyse RPS14 RNA sequencing expression across KIRC pathological parameters such as stage, sex, age, genetic alteration in RPS14, and immune infiltration changes associated with KIRC development.

Materials and method:

Gene expression profile

RPS14 expression profiles in KIRC were determined using various tools. Initially, the Tumour Immune Estimation Resource (TIMER2.0; https://cistrome.shinyapps.io/timer/ date accessed: 15/12/2025) platform was used to examine the differential gene expression of RPS14 between normal (normal tissues were obtained from non-cancer individuals and represent physiologically healthy tissue), and tumour tissues obtained from the Cancer Genome Atlas (TCGA), which showed significant differential expression in RNA-seq sample analysis using the Wilcoxon test. The main statistical parameter considered during this analysis was $p < 0.05$ (two sides). This step was done by the displayed data obtained upon typing the gene symbol on the DiffExp module on TIMER2.0 was used to identify the types of tumours with upregulated differential expression based on their p-value; $p > 0.05$ not significant; $^{*}p < 0.05$ (weak DiffExp); $^{**}p \leq 0.01$ (moderate DiffExp); $^{***}p \leq 0.001$ (high DiffExp).

Gene Expression Profiling Interactive Analysis (GEPIA) (http://gepia.cancer-pku.cn/ date accessed:17/12/2025) is a web-based tool that performs comprehensive analysis using RNA sequencing data from TCGA and the Genotype-Tissue Expression project (GTEx) databases composed of 8587 normal and 9736 tumour tissues. This study utilised GEPIA to conduct differential expression analysis of KIRC. Gene Expression DIY was used to determine the gene expression profile according to user-defined parameters based on a p-value of < 0.05, log2 Fold Change (log2FC) = 1, and 95% confidence interval.

The University of Alabama at Birmingham Cancer Data Analysis Portal (UALCAN) database (https://ualcan.path.uab.edu/ date accessed:17/12/2025) was used to analyse cancer transcriptomic data. RPS14 expression was assessed across the

TCGA dataset, including sample types and individual cancer stages. We conducted a specific pathological and clinical parameter analysis of RPS14 expression across KIRC samples, including patient race, sex, and age. A p-value of < 0.05 was set as the main statistical parameter.

Genetic alteration analysis

TThe nature of RPS14 mutation and its alteration in KIRC were determined using the cBio Cancer Genomics Portal (cBioPortal) database (https://www.cbioportal.org/; date accessed:17/12/2025). The cBioPortal provides deep computational, genomic, and molecular profiling results obtained from large-scale cancer genomics projects which contribute to a better biological and clinical understanding of genes across different tumours. This comprehensive genomic analysis included a query of 3080 samples from patients to show the types of RPS14 alterations and other types of mutations.

Immune infiltration of RPS14 in KIRC

The correlation between RPS14 expression and immune cell infiltration was analysed using TIMER2.0. The association between high RPS14 expression and infiltration of B cells, CD8+ T cells, CD4+ T cells, neutrophils, macrophages, and dendritic cells was evaluated. A scatterplot displaying the correlation association based on the Spearman's rho value was generated. Immune cell infiltration was statistically determined based on a p-value of < 0.05.

Results:

RPS14 expression profile analysis

The RPS14 differential expression between normal (Represents true physiological baseline tissue expression) and tumour tissues was assessed using TIMER2.0 database. RPS14 showed a highly significant differential expression in KIRC (red bar) ($p=6.1\times10^{-12}$) compared to normal tissue (blue bar) (Fig. 1(A)).

Furthermore, RPS14 differential expression between normal and tumour tissues was assessed using the GEPIA database. The red bar represents tumour tissues, the grey bars represent normal tissues, and the stars indicate the differential significance between the tumour ($p<0.05$*, n=523) and normal samples (n=100).

To validate these findings, TCGA samples, including 533 tumour and 72 normal samples were assessed using the UALCAN database to determine the RPS14 differential expression in KIRC. As shown in Figure 1(C), RPS14 was highly upregulated in tumour samples compared to normal tissues ($p=1.0\times10^{-12}$).

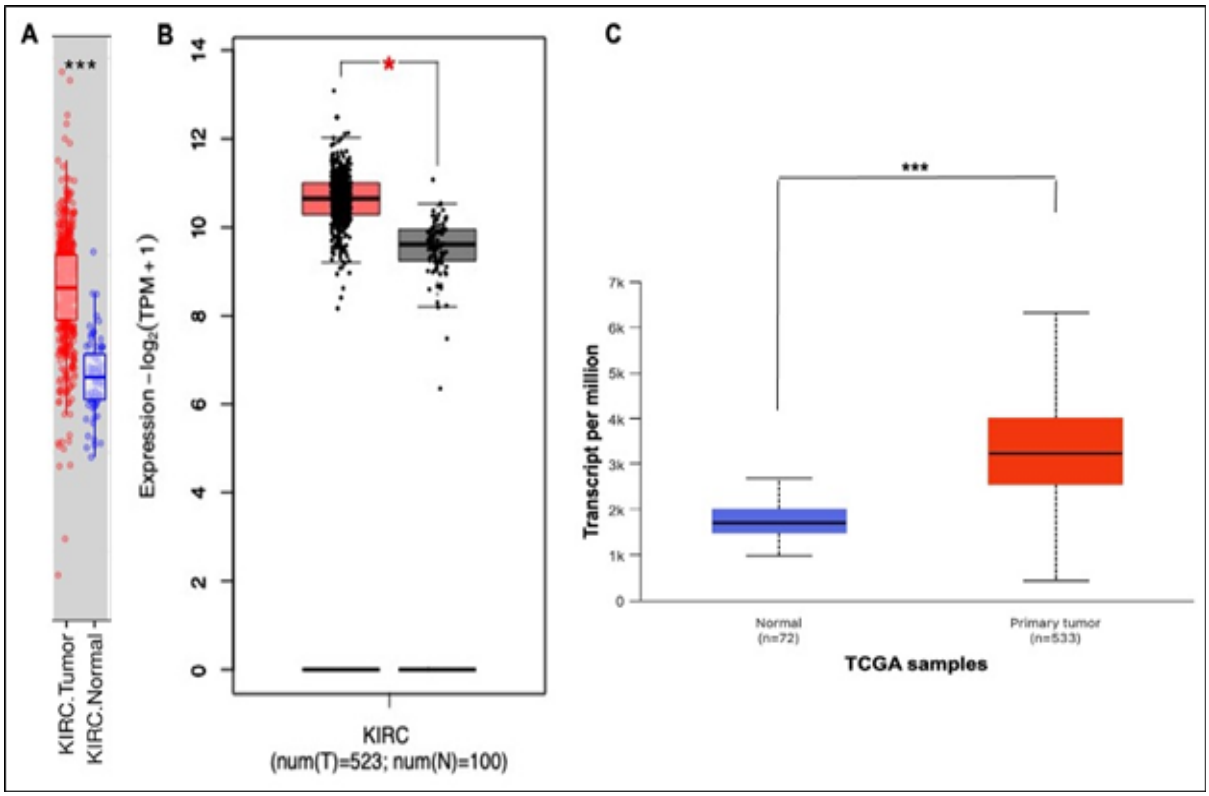


Fig.1: The differential gene expression of RPS14 between normal (blue/gray bar) and tumor (Red bar) kidney clear cell carcinoma (KIRC) tissues

Data obtained from A) Gene Expression Profiling Interactive Analysis (GEPIA), B) Tumor Immune Estimation Resource (TIMER) 2.0 and C) University of Alabama at Birmingham Cancer Data Analysis Portal (UALCAN) database. It was highly upregulated as the p-value was marked ***$p \leq 0.001$. Also, the expression of RPS14 is upregulated as shown in B & C.

RPS14 expression profile analysis based on age, race, and sex

Considering the results obtained from the aforementioned analysis, KIRC was subjected to further analysis using the UALCAN database. The correlation between the clinicopathological parameters (race, sex, and age) and RPS14 expression in KIRC samples was investigated. Sex-specific findings revealed significant upregulation in both males ($p=1.0\times10^{-12}$, n=345) and females ($p=1.0\times10^{-12}$, n=188), compared to normal samples (obtained from non-cancer individuals and represent physiologically healthy tissue) (n= 72) (Fig. 2(A)). Notably, samples from male patients had a significant increase ($p=5.73\times10^{-5}$, compared to those from female patients).

Furthermore, the gene expression profile showed a clear significance based on patient race (Fig. 2(B)), with RPS14 upregulated

in Caucasians ($p=1.62\times10^{-12}$, n=462), African Americans ($p=1.87\times10^{-9}$, n=56), and Asian ($p=4.82\times10^{-3}$, n=8), compared to normal samples. Additionally, the RPS14 expression profile was highly significant in Caucasians ($p=4.39\times10^{-5}$) compared to African Americans (Fig. 2(B)). However, no significant difference in RPS14 expression was noted between Caucasian and Asian populations or between African American and Asian populations.

RPS14 was significantly upregulated in the age groups of 21–40 ($p=3.98\times10^{-4}$, n=26), 41–60 ($p=1.00\times10^{-12}$, n=238), 61–80 ($p=1.62\times10^{-12}$, n=246), and 81–100 years ($p=1.42\times10^{-4}$, n=23) compared to normal samples, as shown in Figure 2(C).

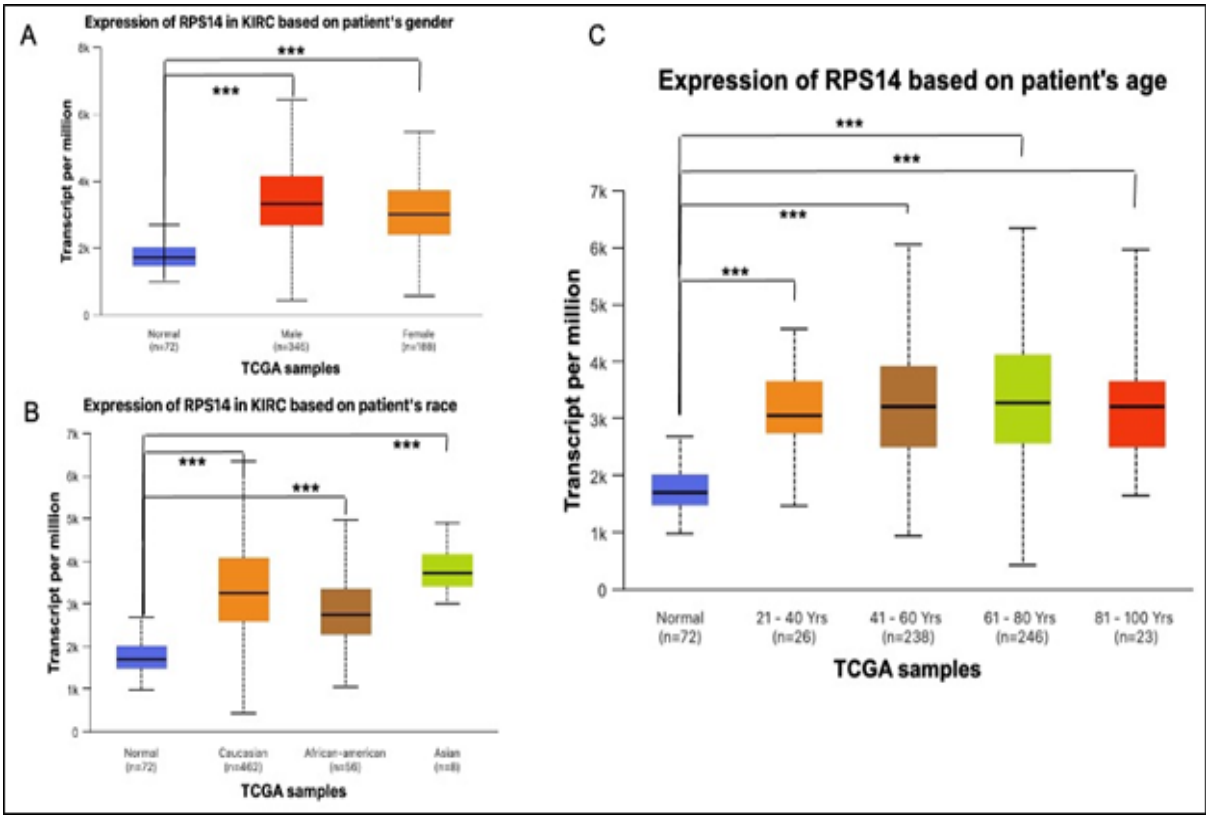


Fig. 2: The differential gene expression of RPS14 between normal (blue) and tumor (Red/orange/green/brown bar) kidney clear cell carcinoma (KIRC) tissues based on patient's gender, race and age.

(A) Both male (n=345) and female (n=188) patients showed increase expression of RPS14 in KIRC compared to normal sample (n=72). (B) All KIRC patients from different races: Caucasian (n=462), Asian American (n=56), and Asian (n=8) showed statistically significant increase expression of RPS14 compared to normal sample (n=72). (C) All age groups showed that the upregulation of RPS14 expression in KIRC compared to normal samples (n=72). It was highly upregulated as the p-vale was

marked *p ≤ 0.05 as shown in A, B & C.

RPS14 expression profile analysis based on cancer stage, grade, subtypes, and nodal metastasis

To evaluate the RPS14 expression in KIRC samples based on the grade of cancer cell differentiation, four grades of KIRC were used. Grades 1 (well differentiated–low grade), 2 (moderately differentiated–intermediate grade), 3 (poorly differentiated–high grade), and 4 (undifferentiated–high grade) were compared to the normal sample (n=72). Figure 2A illustrates that the upregulation of RPS14 in grade 1 samples was statistically higher when compared to that in the normal sample ($p=5.2\times10^{-4}$, n=14). Similarly, grades 2 and 3 showed a significant upregulation of RPS14 ($p=1.0\times10^{-12}$, n=229 and n=206, respectively) compared to that in normal samples. Grade 4 showed an increase in RPS14 expression compared to that in normal samples ($p=1.6\times10^{-12}$, n=76). Notably, analysis of KIRC samples showed that the upregulation of RPS14 expression in grade 1 compared to grades 2 and 3 was significantly increased, whereas no significant changes were observed in the RPS14 expression levels in grade 4 samples (Fig. 2(A)).

Furthermore, the expression of RPS14 among KIRC samples based on cancer stage was assessed using the UALCAN database, which revealed significant alterations in gene expression across all stages of KIRC compared to normal tissue, with highly significant p-values observed in stages 1 ($p=1.62\times10^{-12}$, n=267), 2 ($p=3.08\times10^{-12}$, n=57), 3 ($p=1.0\times10^{-12}$, n=123), and 4 ($p=1.68\times10^{-12,}$ n=84) (Fig. 2(B)).

In terms of nodal metastasis status, gene expression was significantly upregulated in regional lymph node metastasis (N0) ($p=1.62\times10^{-12}$, n=240) compared to that in normal tissue (n=72). Additionally, significant differences were observed between metastases in 1–3 axillary lymph nodes (N1) ($p=1.98\times10^{-3}$, n=16) and normal tissue (n=72) (Fig. 2(C)).

Analysis of the differential expression of RPS14 among KIRC samples based on cancer subtypes A and B (Fig. 2(D)) revealed that RPS14 expression increased in KIRC subtype A significantly ($p=1.00\times10^{-12}$, n=205) compared to that in normal tissue. Similarly, RPS14 expression increased in KIRC subtype B significantly ($p=1.00\times10^{-12}$, n=175) compared to normal tissue. Notable differences in RPS14 expression were detected between KIRC subtypes A and B ($p=5.4\times10^{-4}$).

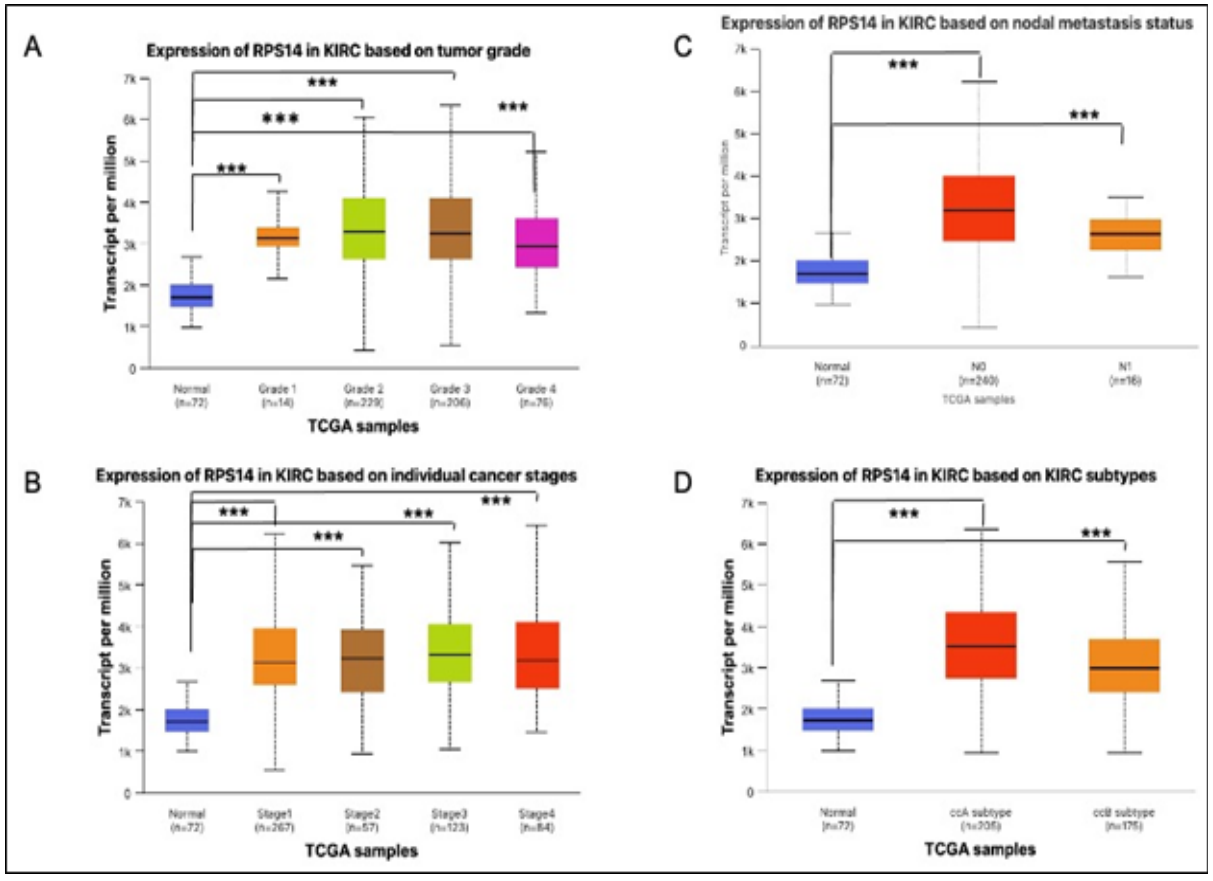


Fig. 3: The differential gene expression of RPS14 between normal (blue) and tumor (Red/orange/green/brown bar) kidney clear cell carcinoma (KIRC) tissues based on tumor grade, cancer stages, nodal metastasis and KIRC subtypes.

(A) In All KIRC grades RPS14 expression was increased significantly (grade 1, n=14), grade 2, n=229), (grade 3, n=206, (grade 4, n=76) compared to normal sample n=72. (B) All KIRC patients from different stages: stage1 (n=287), stage 2 (n=57), and stage 3 (n=123) and stage 4 (n= 84) showed a statistically significant increase expression of RPS14 compared to normal sample (n=72). (C) Both nodal metastasis (N0 and N1) shows that an upregulation of RPS14 expression in KIRC compared to normal samples (n=72). (D) Both KIRC subtypes A & B had increased expression of RPS14 as the p-value was marked *$p \leq 0.05$ as shown in A, B, C & D.

RPS14 expression profile analysis based on genetic alteration analysis

Examining 2281 patients from eight studies, including Dana-Farber Cancer Institute (DFCI, Science 2019); Beijing Genomics Institute (BGI, Nat Genet 2012); International Renal Consensus (IRC, Nat Genet 2014); The Cancer Genome Atlas (TCGA, Firehose Legacy), (TCGA, Nature 2013), (TCGA, PanCancer Atlas), and (TCGA, GDC); and the University of Tokyo (UTokyo, Nat Genet 2013),

revealed RPS14 alteration in 141 (6%) of 2281 queried patients (Fig. 4). Putative copy number alterations from the Genomic Identification of Significant Targets in Cancer (GISTIC) detected two missense somatic mutations. The structural variant in which threonine at position 45 changes to alanine (T45A) is a point mutation that alters the structure and function of the protein. The second mutation in RPS14 is a specific amino acid change from lysine to arginine at position 7 (K7R). Furthermore, RPS14 amplifications were estimated from GISTIC including genetic alterations (gain and/or diploidy) (Fig. 4).

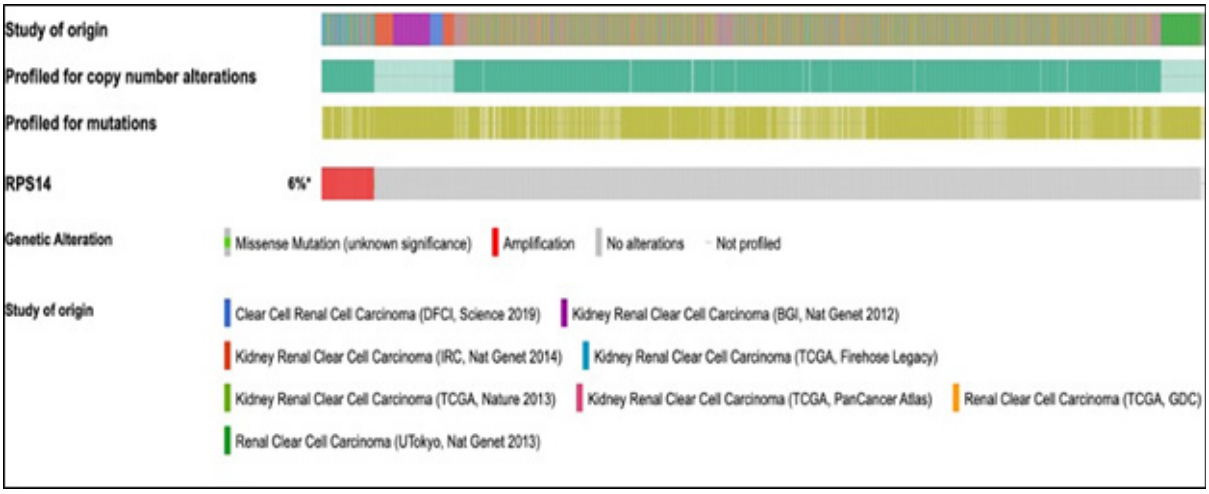


Fig. 4: RPS14 gene mutation's nature and its alteration in KIRC using the cBio Cancer Genomics Portal (cBioPortal database).

Querying 2281 patients in 8 studies showed that RPS14 is altered in 141 (6%) including (amplification and missense mutation) of queried samples.

Correlation analysis of RPS14 expression and immune infiltration of immune cells

TThe tumour purity indicates that the tissue was composed more of cancer cells than of immune cells. The patient samples of KIRC revealed that the correlation between RPS14 expression in KIRC tissues was weak (partial Cor=0.08, p=0.054). The infiltration of immune cells such as CD8+ T cells, CD4+ T cells, B cells, neutrophils, macrophages, and dendritic cells was investigated to explore the prognosis of tumours, which is influenced by the interaction of immune cells. The relationship between RPS14 and KIRC expression was significantly positively correlated with B-cell infiltration (partial. Cor= 0.15, p=0.04), CD4+ T cells (partial. Cor=0.168, p=0.0002), and neutrophils (partial. Cor= 0.138, p=0.003) (Table 1).

Table 1: Correlation between RPS14 expression and immune infiltration of cells using the TIMER database.

Cancer	Variable	Partial correlation (ABS)	p value	Interpretation
KIRC	Purity	0.089661646	0.0541214	not
KIRC	B Cell	0.095174458	0.0415386	Sig*
KIRC	CD8+ T Cell	0.040378053	0.39924017	not
KIRC	CD4+ T Cell	0.168177378	0.00029118	Sig*
KIRC	Macrophage	0.065872085	0.16396295	not
KIRC	Neutrophil	0.138126744	0.00305475	Sig*
KIRC	Dendritic Cell	0.061765477	0.18846471	not

Discussion:

In human ribosomes, RPS14 forms a small 40S subunit at the junction of the 18S pre-RNA. [9,24] RNA polymerase I (RNA Pol I) generates a 45S precursor transcript, which is processed into three rRNA species: 28S, 5.8S, and 18S. RNA polymerase III (RNA Pol III) transcribes 5S rRNA.[4, 24] Genetic alterations in different ribosomal proteins are associated with different errors in pre-RNA processing. [25] In this study, data from normal kidney tissue and KIRC tissue samples from TCGA were investigated to identify RPS14 expression profiles using three different databases. The differential expression of RPS14 in KIRC showed increased expression of RPS14 which could play an essential role in tumour development (Fig. 1). These findings align with earlier studies that identified RPS14 as being upregulated in various tumour types, such as breast cancer,[26] glioma,[15] and hepatocellular carcinoma. [16] Furthermore, analysis of the UALCAN database confirmed significant upregulation of RPS14 expression in KIRC across various clinicopathological parameters, including sex, race, and age groups, when compared to the RPS14 expression in normal tissue. The results also showed a significant increase in RPS14 expression in male patients with KIRC compared to female patients (Fig. 2(A)). Studies have revealed that males have a higher incidence of kidney cancer than females by approximately 2:1 worldwide.[27] This may be related to multiple factors, including hormonal protection in females, increased carcinogenic exposure (e.g., smoking), sexual dimorphism in gene expression, and immune-related pathways in kidney tissues, which could partly explain the risk disparity. [27–29]

In terms of racial groups (Fig. 2(B)), Caucasian and Asian populations were more affected than the African-American population. This may be related to lifestyle and socioeconomic factors that also affect the racial epidemiology and histological

subtypes of renal cell carcinoma (RCC). [30,31] A similar pattern of RPS14 upregulation was observed across the groups, indicating that all ages were at a similar risk for KIRC development (Fig. 2(C)).

Further analysis of other clinicopathological parameters such as disease grade, stage, metastasis status, and KIRC subtypes revealed that RPS14 expression in nodal metastasis N0 was higher than that in N1, and showed higher expression in subtype A than in subtype B. However, all KIRC stages (1–4) showed similar overexpression levels (Fig. 2(D)).

The cBioPortal platform was used to identify genetic alterations in RPS14 across the eight studies. Genetic alterations in RPS14 were reported, such as T45A and K7R, and multiple gene amplifications have been reported in 6% of patients with KIRC (Fig. 4). Earlier studies have shown that K7R and T45A mutations lead to reduced RPS14 levels (haploinsufficiency), directly causing erythroid (red blood cell) failure and anaemia in 5q- syndrome. [18,32,33] Additionally, loss of one copy may affect RPS14 stability, interactions, regulatory roles, and immune signalling. [18] Such mutations, even partial losses, highlight how ribosome dysfunction (ribosomopathy) affects haematopoiesis and can increase susceptibility to leukaemia,[34] breast cancer,[6] hepatocellular carcinoma,[16] and other tumours.

The tumour microenvironment (TME) plays a vital role in tumour progression and effectiveness of immunotherapy. Genes that are highly expressed in the TME are expected to have negative associations with tumour purity, whereas the opposite is expected for genes that are highly expressed in tumour cells. In this study, we examined the relationship between RPS14 expression and immune cell infiltration in KIRC, using the TIMER2.0 database. Weak correlation of RPS14 expression indicates that the upregulation of RPS14 was negatively associated with tumour purity, although this finding was not statistically significant. Furthermore, overexpression of RPS14 has been shown to correlate with the infiltration of B cells, CD4+ T cells, and neutrophils, suggesting that mutations in RPS14 affect immune signalling (Table 1). These results indicated that RPS14 expression is linked to distinct patterns of immune cell infiltration within the TME, which may affect tumour progression and prognosis.

These results highlight the need for further studies to validate RPS14 as a diagnostic and prognostic biomarker and as a predictive indicator of treatment response in KIRC. Additionally, integrated analyses of DNA methylation, survival data, and proteomic profiles are required to substantiate the functional and clinical

significance of RPS14 as a promising therapeutic target in KIRC and other malignancies.

Conclusion:

This study emphasizes that RPS14 was overexpressed, suggesting its potential role in the dysregulation of cell proliferation and its involvement in KIRC development. An investigation of the association between RPS14 overexpression and KIRC showed that males were more likely to develop KIRC than females. Additionally, significant differences were detected between RPS14 overexpression in KIRC subtype A compared to KIRC subtype B. Moreover, genetic amplification of RPS14 gene expression was estimated in 6% of KIRC patients from GISTIC, including genetic alterations (gain and/or diploid). Finally, the upregulation of, observed to be associated with immune infiltration, highlights its potential effect on the tumour microenvironment. Further studies are required to determine the importance of RPS14 as a prognostic biomarker and therapeutic target in cancer.

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