PGGHG and ODF3B as New Tumor Suppressor Genes in Renal Cell Carcinoma

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Abstract

Background

Renal cell carcinoma (RCC) is the most frequent heterogeneous type of kidney cancer. Although the role of many genes in the development of RCC has been shown, its specific molecular mechanism is still unknown. In this research, we studied the expression profile of two poorly studied genes, *PGGHG* and *ODF3B*, with possible role in renal cancers in our RCC patients' tumors and adjacent normal tissues, as well as their clinicopathological features.

Materials and Methods

The *PGGHG* and *ODF3B* expression was determined using the quantitative real-time PCR in 40 tumor samples and their adjacent non-cancerous tissues. Moreover, Enrichr was used to investigate their pathways, ontologies, and possible upstream transcription factors. Furthermore, STRING was applied to search for interaction between *PGGHG* and *ODF3B*.

Results

The expression level of *PGGHG* and *ODF3B* was lower in the tumor tissues compared to their adjacent normal tissues. The expression level of *ODF3B* in the patients who had tumor size > 4cm was significant. Our data found the possible upstream transcription factor of *ODF3B* and *PGGHG* regulating their expression in biological pathways, mainly *PPARG* as the same upstream transcription factor affecting both genes. Moreover, our data revealed connection between protein networks of *PGGHG* and *ODF3B*. We found several main KEGG, biological process (BP), molecular function (MF), and cellular component (CC) for proteins involved in these networks.

Conclusion

PGGHG and *ODF3B* downregulation in RCC tumors suggests their possible tumor-suppressive role in RCC. More research is needed to determine the precise mechanism of their involvement in RCC pathogenesis.

Introduction

Renal cell carcinoma (RCC) is the most common type of kidney malignancies (1) and highly aggressive heterogeneous disease (2). On the basis of data of SEER database, the 5-year relative survival rate of RCC for all SEER stages combined is approximately 75%. The most common histological subtypes of RCC are clear cell, papillary, and chromophobe (3) responsible for 70%, 10–15%, and 5% of the RCC types, respectively (4). So far, many factors have been implicated in the development of ccRCC, including genetic and environmental factors. The majority of ccRCC has shown genetic or epigenetic inactivation of the von Hippel-Lindau (*VHL*) gene. Moreover, dysregulation of several genes has been contributed to the RCC progression and metastasis in different pathways. CcRCC patients respond poorly to radiotherapy and chemotherapy. Therefore, the molecular predictions of disease progression and metastasis are important for therapies. We are looking to explore new molecular biomarkers of disease progression and ccRCC-specific molecular mechanisms that may provide new targeted treatment options.

This study set out to investigate the potential contribution of two genes with unknown roles, including protein-glucosylgalactosylhydroxylysine glucosidase (*PGGHG*) (also known as *ATHL1*) and outer the dense fiber of sperm tails

3B (*ODF3B*) (Also known as *FAP123*, *ODF3L3*), in RCC pathogenesis. The chromosomal positions of *ODF3B* and *PGGHG* genes are 22q13.33 and 11p15.5, respectively.

The pronephros is consisted of two types of epithelial cells, including transportive and multiciliated cells (MCCs). The epithelial MCCs expresses *odf3b* protein (5). Previous study showed that *odf3b* transcripts localize in pronephros cells and labels maturing MCCs in renal progenitors (6). Using STRING database, *ODF3B* showed interaction with proteins involved in main biological pathways and cancers (including renal cancers), for example WAPAL (7), STAG1 (8), RAD21L1(9) and among others, representing its possible role in these pathways and cancers (Fig. 1).

In relation to the *PGGHG*, it was found to act in release of glucose from human placental type IV collagen (10). Data extracted from Enrichr (<https://maayanlab.cloud/Enrichr/>) shows its involvement in hydrolase activity, hydrolyzing O-glycosyl compounds (GO: 0004553). Moreover, previous study conducted by Wei Shi et al. showed higher expression of *hsa-mir-484* correlated with worse prognosis in breast cancer. In their study, they found that one of target gene of *hsa-mir-484* was *PGGHG*(11). In addition to these data, its protein network revealed its interaction with several important proteins involved in biological pathways and cancers (including renal cancers), for instance, COL10A1 (12) (Fig. 1). Furthermore, when we constructed and connected the protein network for *PGGHG* and *ODF3B* using STRING (<https://string-db.org>), these two proteins showed involvement in the same network (Fig. 1), suggesting their possible roles in renal pathways and cancers. Therefore, our aim was to investigate their expression and roles in our available samples from renal cancers.

Materials And Methods

Patient characteristics and tumor samples

To investigate mRNA expression of *PGGHG* and *ODF3B* in renal cancers, we collected 40 tumor tissues and their adjacent normal tissues of RCC patients from Ali-Asghar, Namazi, and Ghadir Mother and Child Hospitals (Shiraz, Iran). In total, RCC patients had not received radiotherapy and chemotherapy before surgery. The samples are immediately immersed in liquid nitrogen and stored at -80°C until use. The clinicopathologic and demographic features of RCC patients are shown in Table 1. After surgery, we followed up patients to find they had all-cause mortality.

Table 1
The association of ODF3B and PGGHG expression levels with demographic and clinicopathological factors in RCC patients.

		ODF3B level					PGGHG level				
		N	Mean	SD	Median	P-value	N	Mean	SD	Median	P-value
Sex	male	29	0.924	0.984	0.620	0.844	29	0.795	1.037	0.309	0.296
	female	11	0.748	0.637	0.660		11	0.971	0.857	0.798	
Tumor size	≤ 4	20	1.054	0.881	0.919	0.060	20	0.758	0.843	0.540	0.745
	> 4	20	0.697	0.899	0.357		20	0.928	1.122	0.333	
Tumor focality	focal	25	1.097	1.031	0.774	0.067	25	0.840	1.039	0.491	0.856
	unifocal	15	0.507	0.434	0.587		15	0.849	0.918	0.309	
Tumor type	clear cell	27	0.959	0.835	0.688	0.004	27	0.925	0.934	0.505	0.017
	papillary	5	1.283	1.330	0.774		5	0.974	0.958	0.644	
	chromophore	6	0.043	0.030	0.031		6	0.069	0.096	0.029	
	oncocytoma	2	1.231	0.864	1.231		2	1.730	2.427	1.730	
Tumor necrosis	seen	20	0.924	1.086	0.601	0.715	20	0.598	0.771	0.331	0.160
	not seen	20	0.827	0.685	0.674		20	1.088	1.124	0.635	
Fuhrman nuclear grade	1	5	0.328	0.308	0.233	0.113	5	0.359	0.573	0.090	0.225
	2	21	0.768	0.920	0.470		21	0.818	0.961	0.399	
	3	9	1.189	0.711	1.603		9	1.268	1.180	0.798	
	4	5	1.310	1.271	1.065		5	0.665	0.979	0.302	
Lymph vascular perineural invasion	No	8	1.057	1.055	0.841	0.467	8	0.801	1.042	0.304	0.866
	Yes	32	0.830	0.866	0.625		32	0.854	0.985	0.445	
Extension	No	30	0.870	0.884	0.625	1.000	30	0.901	1.046	0.445	0.779
	Yes	10	0.892	0.985	0.695		10	0.670	0.787	0.330	
Cancer history	No	19	0.891	0.998	0.587	0.882	19	0.897	1.085	0.399	0.903
	Yes	21	0.861	0.820	0.774		21	0.794	0.906	0.333	
BMI	≤ 25	9	1.224	1.433	0.616	0.918	9	0.762	0.928	0.234	0.784
	25–29	26	0.771	0.689	0.645		26	0.826	1.007	0.445	
	≥ 30	5	0.791	0.650	0.620		5	1.080	1.134	0.798	
Kidney disease	No	29	0.867	0.838	0.629	0.880	29	0.880	1.038	0.357	0.940
	Yes	11	0.897	1.083	0.470		11	0.745	0.860	0.399	

Rna Extraction And Cdna Synthesis

Using the Trizol isolation reagent (Invitrogen, Thermo Fisher) total RNA was extracted from each sample according to the manufacturer’s instructions. Gel electrophoresis and a nanodrop spectrophotometer (BioTek, HTX multi-mode reader) was used to assess RNA quality and quantity, which detected ratios of ≥ 2 and 1.8 for A260/A230 and A260/A280, respectively. Total RNA was reverse-transcribed into first-strand cDNA using the PrimeScript™ RT Reagent Kit (Takara, Cat.No: RR037A) following manufacturer’s recommendations.

Qrt-pcr

QRT-PCR was carried out using Power SYBR® Green PCR Master Mix (ABI, USA) on the 7500 real-time PCR system (ABI, life technology). 7.5 µl BioFACT™ master mix including SYBR Green (Ampliqon, Cat.No: A325402-25), along with 1 µl of cDNA, 0.75 µl of each primer and 5 µl DNase-free dH2O was used for total volume of 15 µl reaction mix. B2M was used as an internal control for normalization of data compared to the studied genes. All reactions were confirmed at least twice and the results were shown to be consistent. The sequences for sense (S) and antisense (AS) primers are shown in Table 2. Relative quantification strategy for quantitative real-time polymerase chain reaction (qPCR) data analysis was calculated by the $2^{-\Delta\Delta CT}$ (fold change) method.

Table 2
Primers sequences

mRNAs and housekeeping gene	Primer	
PGGHG	Forward	TCTACTACCTGCTCAGTG
	Reverse	GTGGAACATCAGGATACT
ODF3B	Forward	GGACCTGGCAGGTACTTC
	Reverse	CGTAGATGGAGCAAGTTG
B2M (housekeeping gene)	Forward	AGATGAGTATGCCTGCCGTG
	Reverse	GCGGCATCTTCAAACCTCCA

Statistical analysis

Statistical analyses were done in IBM SPSS version 26 software (IBM Corp, Armonk, NY). The data were presented as mean and standard deviation for $\Delta\Delta Ct$, or median for fold change. Wilcoxon test was used to compare fold changes between tumors and adjacent normal renal tissues. The relation of PGGHG and ODF3B expression with demographic and clinicopathological features was assessed by Mann–Whitney and Kruskal-Wallis tests. In the next step, fold changes were divided into 2 groups of high and low expressions conforming to median for each gene, and the comparison among these groups were analyzed by chi-square test and independent t test. The level of P-value less than 0.05 was regarded as a statistical significance. After surgery, we followed up patients until all-cause death used and censored alive one. The log rank test is a statistical hypothesis test that may be used to compare two high and low gene expression survival curves.

Bioinformatic Analysis

We used Enrichr database (<https://maayanlab.cloud/Enrichr/>) to look for their pathways, ontologies, and possible upstream transcription factors and among others. Moreover, we used STRING (<https://string-db.org>) to look for interaction between *PGGHG* and *ODF3B* and their partner, helping identify their possible pathways. To evaluate survival analysis our patients, we used the Kaplan–Meier method based on observed survival times. This analysis was performed using IBM SPSS version 26 software (IBM Corp, Armonk, NY).

Result

Downregulation of *PGGHG* and *ODF3B* gene expression in RCC

The expression levels of mRNAs were assessed in 40 tumor samples and their tumor's adjacent normal tissues using qRT-PCR. As shown in the Fig. 2A, the expression of *PGGHG* had significantly lower in tumor tissues (median = 0.378) compared to their adjacent normal tissues (median = 1.120) (Pvalue = 0.034). Also, the expression level of *ODF3B* was significantly lower in tumors tissues (median = 0.625) in comparison to the tumor's adjacent normal tissues (median = 0.977) (Pvalue = 0.042, Fig. 2B).

Association between *PGGHG* and *ODF3B* gene expression and clinicopathological and demographic features of RCC

Next, we investigated the relationship between *PGGHG* and *ODF3B* expression levels and the clinicopathologic status of patients with renal cell carcinoma. Our results demonstrated that *ODF3B* had lower expression in RCC patients who had tumor size > 4cm (Pvalue = 0.060, Fig. 3A). When we divided fold changes into two high and low expressions groups, chi-square test proved the significant relation between tumor size and *ODF3B* expression (Pvalue = 0.011, Table 3). Comparison of *PGGHG* expression level and tumor size did not show a significant difference (Pvalue = 0.744).

Table 3

The association of ODF3B and PGGHG expression levels with demographic and clinicopathological factors in RCC patients, according to categorizing patients in to two groups of high and low expressions.

		ODF3B level					PGGHG level					
		Low		High		P-value	Low		High		P-value	
		N	%	N	%		N	%	N	%		
Tumor size	≤ 4	6	30.0%	14	70.0%	0.011	9	45.0%	11	55.0%	0.527	
	> 4	14	70.0%	6	30.0%		11	55.0%	9	45.0%		
Tumor focality	focal	11	44.0%	14	56.0%	0.327	12	48.0%	13	52.0%	0.744	
	unifocal	9	60.0%	6	40.0%		8	53.3%	7	46.7%		
Fuhrman nuclear grade	1	4	80.0%	1	20.0%	0.157	4	80.0%	1	20.0%	0.093	
	2	12	57.1%	9	42.9%		10	47.6%	11	52.4%		
	3	2	22.2%	7	77.8%		2	22.2%	7	77.8%		
	4	2	40.0%	3	60.0%		4	80.0%	1	20.0%		
Lymph vascular perineural invasion	No	4	50.0%	4	50.0%	1.000	5	62.5%	3	37.5%	0.429	
	Yes	16	50.0%	16	50.0%		15	46.9%	17	53.1%		
BMI	≤ 25	5	55.6%	4	44.4%	0.793	6	66.7%	3	33.3%	0.508	
	25–29	12	46.2%	14	53.8%		12	46.2%	14	53.8%		
	≥ 30	3	60.0%	2	40.0%		2	40.0%	3	60.0%		
Background disease	None	11	52.4%	10	47.6%	0.727	12	57.1%	9	42.9%	0.313	
	High blood pressure	7	58.3%	5	41.7%		6	50.0%	6	50.0%		
	diabetes	1	33.3%	2	66.7%		1	33.3%	2	66.7%		
	High blood pressure and diabetes	1	33.3%	2	66.7%		0	0.0%	3	100.0%		
	prostate problems	0	0.0%	1	100.0%		1	100.0%	0	0.0%		
Kidney disease	No	14	48.3%	15	51.7%	0.723	15	51.7%	14	48.3%	0.723	
	Yes	6	54.5%	5	45.5%		5	45.5%	6	54.5%		
Kidney stone	No	10	47.6%	11	52.4%	0.752	10	47.6%	11	52.4%	0.752	
	Yes	10	52.6%	9	47.4%		10	52.6%	9	47.4%		

Since ccRCC is the most common of RCC and oncocytoma is the least common type in our study community (Table 1), our analysis showed that *PGGHG* expression level was higher in oncocytoma type in comparison to other type of RCC

(Pvalue = 0.017, Fig. 3B). Similarly, the relationship between the *ODF3B* expression level and the tumor type showed significant. The expression level of *ODF3B* was higher in oncocytoma type (Pvalue = 0.004, Fig. 3C).

Bioinformatic Analysis

Functional enrichment analysis

Using Enrichr, we found down-regulation of *ODF3B* upon *RUNX1* Knock-out mouse, *PPARG* deficiency mouse, *SETDB1* Knock-down in THP1 human cells, *IRF9* Knock-down in human cells, *NANOG* over-expressed mouse, and *CREM* Knock-out mouse (Table 4). These data can suggest the possible upstream transcription factors of *ODF3B* which regulate its expression in biological pathways. Among GO ontologies using Enrichr, *ODF3B* was found to be involved in cytoskeleton (GO: 0005856, p-value: 0.03000) in terms of cellular component (CC).

Table 4
Down-regulation of ODF3B mRNA upon transcription perturbations.

Index	Name	P-value	Adjusted p-value	Odds Ratio	Combined score
1	RUNX1 KO MOUSE GSE34292 CREEDSID GENE 2396 DOWN	0.00895	0.01826	19821	93478.06
2	PPARG DEFICIENCY MOUSE GSE23421 CREEDSID GENE 1231 DOWN	0.0103	0.01826	19794	90569.85
3	SETDB1 KD THP1 HUMAN GSE103409 SGRNA6DAY4 RNASEQ DOWN	0.01105	0.01826	19779	89111.01
4	IRF9 KD HUMAN GSE50588 CREEDSID GENE 2809 DOWN	0.0132	0.01826	19736	85408.48
5	NANOG OE MOUSE GSE48370 CREEDSID GENE 2980 DOWN	0.01565	0.01826	19687	81844.62
7	CREM KO MOUSE GSE29593 CREEDSID GENE 2559 DOWN	0.01905	0.01905	19619	77704.89

In relation to the *PGGHG*, Enrichr revealed its down-regulation upon *ZNF503* shRNA in H1 human cells, *NFYC* Knock-out in mouse, *BCL6* Knock-down in human cells, *POU2AF1* over-expression in mouse, *EPAS1* Knock-down in HUVEC human cells, *NFYA* Knock-out in mouse, *FOXP3* activation in human cells, *PPARG* over-expression in mouse, and *GATA4* over-expression in A549 human cells (Table 5).

Table 5
Down-regulation of PGGHG mRNA upon transcription perturbations.

Index	Name	P-value	Adjusted p-value	Odds Ratio	Combined score
1	ZNF503 SHRNA H1 HUMAN GSE69618 DAY2 RNASEQ DOWN	0.00795	0.01847	19841	95923.19
3	NFYC KD MOUSE GSE56838 CREEDSID GENE 1343 DOWN	0.0093	0.01847	19814	92684.96
5	BCL6 KD HUMAN GSE45838 CREEDSID GENE 1815 DOWN	0.0118	0.01847	19764	87745.54
6	POU2AF1 OE MOUSE GSE12421 CREEDSID GENE 2313 DOWN	0.0122	0.01847	19756	87051.43
7	EPAS1 KD HUVEC HUMAN GSE62974 RNASEQ DOWN	0.0136	0.01847	19728	84784.91
8	NFYA KD MOUSE GSE56838 CREEDSID GENE 1341 DOWN	0.0138	0.01847	19724	84479.77
9	FOXP3 ACTIVATION HUMAN GSE41087 CREEDSID GENE 1054 DOWN	0.01385	0.01847	19723	84404.16
10	PPARG OE MOUSE GSE10192 CREEDSID GENE 2733 DOWN	0.0161	0.01932	19678	81249.36
12	GATA4 OE A549 HUMAN GSE85001 RNASEQ DOWN	0.02545	0.02545	19491	71552.36

The interesting result was that while *PPARG* over-expression in mouse resulted in *PGGHG* down-regulation, *PPARG* deficiency mouse showed *ODF3B* down-regulation. These data may indicate *PPARG* as the same upstream transcription factor affecting *ODF3B* and *PGGHG*.

Among GO ontologies using Enrichr, *PGGHG* showed involvement in hydrolase activity, hydrolyzing O-glycosyl compounds (GO: 0004553, p-value: 0.001850, in terms of molecular function (MF)).

As shown in Fig. 1, protein networks of *PGGHG* and *ODF3B* are connected and correlated. Therefore, we investigated the GO terms for all proteins involved in these networks to shed light into the identification of possible pathways of *PGGHG* and *ODF3B*.

Enrichr revealed significant KEGG, biological process (BP), MF, and CC for proteins involved in these networks given in Fig. 4. However, the highest significant terms were as follow: **KEGG**: Starch and sucrose metabolism (p-value: 3.058e-43); **BP**: glucan biosynthetic process (GO:0009250, p-value: 5.259e-24), glycogen biosynthetic process (GO:0005978, p-value: 5.259e-24), and glycogen metabolic process (GO:0005977, p-value: 8.723e-27); **MF**: UDP-glucosyltransferase activity (GO:0035251, p-value: 1.319e-8) and 1,4-alpha-oligoglucan phosphorylase activity (GO:0004645, p-value: 2.305e-7); **CC**: chromosome (GO:0005694, p-value: 1.921e-8). All information and proteins involved in these terms are provided in Supplementary file 1 and 2. These data may propose the possible roles of *PGGHG* and *ODF3B*, directly or indirectly, in these GO terms and KEGG pathways.

Overall Survival

To evaluate survival analysis, we used the Kaplan–Meier method based on observed survival times. However, we were not able to predict the prognosis of renal cell carcinoma patients based on *PGGHG* (*ATHL1*) and *ODF3B* gene expression using this data (Fig. 5A, 5B). The small proportion of participants evaluated, low duration of follow up and undetected exact cause of death could be some of the reasons.

Discussion

Renal cell carcinoma (RCC) is the most common form of kidney cancer and accounts for approximately 3.7% of the total cancer deaths (13). Identification of biomarkers helps in improving the prognosis and early detection of RCC. Despite astonishing molecular advances and their significant impact on the detection of functional mRNAs in malignancies, numerous questions remain regarding molecular interactions and their function and need further attention.

In this study, we compared the *PGGHG* and *ODF3B* expression levels in tumor and their adjacent normal tissues in RCC patients and their association with patients' demographical and clinicopathologic factors. Analysis of these biomarkers in tissue samples indicated that they may have value for discriminating RCC patients from healthy controls.

Our results indicate that the expression level of *ODF3B* is significantly lower in tumor tissues than their normal adjacent ones. There are few investigations about the role of the studied genes in the development and progression of cancers. In the case of the *ODF3B* gene, an experimental study related to cancer has not been reported. Jihye Ryu *et al.* (14) examine promotor activity of *ODF3B* in multiple sclerosis (MS) on large scale by genome-wide association studies (GWAS) signals. In this study, an association of promotor variants with the expression of this gene was reported. In another study, Honglin Zhu *et al.* (15) showed the *ODF3B* as potential methylation-regulated differentially expressed gene (DEG). This gene delineates the abnormal activation of immune regulation in the pathogenesis of Systemic sclerosis (SSc).

Our study also showed significant lower expression of *PGGHG* in RCC tissues in comparison to their adjacent normal specimens. In the past decades, the role of aerobic glycolysis in cancer cells has been discussed, however, data supporting for glycogen biology in RCC are lacking (16). In line with our results, a study demonstrated that reduced *AGL*, a glycogen debranching enzyme, increased tumor growth in patients with bladder cancer by RNA interference screen (17). In another study, *AGL* loss led to the progression of bladder cancer. Details of this metabolomics pathway showed increased glucose metabolism with the help of serine hydroxymethyltransferase in cells (18).

Our data from Enrichr revealed significant KEGG, MF, BP, and CC for proteins involved in *ODF3B* and *PGGHG* networks, suggesting the possible roles of these two proteins, directly or indirectly, in these GO terms and KEGG pathways. Moreover, using Enrichr, we found the down-regulation of *ODF3B* and *PGGHG* upon perturbations of some transcription factors, mainly *PPARG* which affected both genes. *PPARG*, Peroxisome Proliferator Activated Receptor Gamma, is involved in adipocyte differentiation (19) considered a “master regulator” of adipogenesis. Moreover, it represses inflammatory response genes in mouse macrophages (20).

We also identified the positive relationship of tumor size with the *ODF3B* expression level (Table 3). According to a study by Zhi *et al.*, an increase in tumor size has been reported in the progression and high risk of lymph node metastases (LNM) in ccRCC (21).

In summary, we found lower expression of *ODF3B* and *PGGHG* in renal cancers and proposed their possible biological pathways and upstream transcription factors. Our results propose investigation of functional studies for *ODF3B* and

PGGHG genes to identify their exact roles in biological pathways and cancers.

Declarations

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Conflict of Interest: The authors declare that there is no conflict of interest.

Availability of data and material:

Samples were provided from Ali-Asghar, Namazi, and Ghadir Mother and Child hospitals, Shiraz, Iran.

Authors' contributions:

Conceptualization: [Elham Mohammadisoleimani]; Methodology: [Elham Mohammadisoleimani, Zahra Firoozi, and Hassan Dastsooz]; Formal analysis and investigation: [Hassan Dastsooz, Mohammad Mehdi Naghizadeh, and Elham Mohammadisoleimani]; Writing - original draft preparation: [Elham Mohammadisoleimani, Hamed Haghi-Aminjan, Hassan Dastsooz, and Zahra Firoozi]; Writing - review and editing: [Hassan Dastsooz, Abdolreza Daraei, and Yaser Mansoori]; Funding acquisition: [Yaser Mansoori], Resources: [Shahryar Zeighami, Ali Ariafar, Hosein Mansoori]; Supervision: [Yaser Mansoori]. All authors read and approved the manuscript before submission.

Ethics approval:

The local ethical committee at Fasa University of Medical Sciences (ethical code: IR.FUMS.REC.1398.147) approved this study.

Consent to participate:

Signed informed consent form was collected from each individual in the patient.

Consent for publication:

Consent for publication is not required as no identifying personal information is published in this manuscript.

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Figures

Figure 1

Protein-protein interaction network of PGGHG and ODF3B. It shows connection between protein networks of PGGHG and ODF3B, representing their possible roles in the same pathways and cancer pathogenesis

Figure 2

The boxplot of comparison of PGGHG and ODF3B expression between tumor samples and paired adjacent normal tissues

Figure 3

A) The association between the expression of ODF3B and tumor size. B,C) PGGHG and ODF3B expression levels in the different subgroups of tumor types in RCC

Figure 4

Significant KEGG (Kyoto Encyclopedia of Genes and Genomes) human pathways, BP (biological process), MF (molecular function), and CC (cellular component) GO terms for proteins involved in PGHG and ODF3B networks

Figure 5

Kaplan-Meier curves for survival rate. Overall survival of patients by 2-year (A: PGGHG, B: ODF3B)

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