Supplementary Tables with legends

Supplementary Table 1. Comparison of clinical characteristics at diagnosis of ATN and control groups.

	ATN (n=5)	ST (n=4)	P value
Male, n (%)	4 (80.0)	0 (0.0)	0.010
Age (y), mean±SD	52.0±5.5	68.3±3.8	0.056
Serum Cr (μmol/L), mean±SD	346.54±139.24	60.23±4.61	0.059
BUN (mmol/L), median (IQR)	15.40 (12.94, 17.70)	7.73 (4.90, 8.86)	0.014
Uric acid (mmol/L), mean±SD	379.00±51.03	285.00±19.78	0.164
Hemoglobin (g/dL), mean±SD	102.80±13.82	123.50±4.84	0.243
TG (mmol/L), mean±SD	5.20±3.71	2.75±2.09	0.610
TC (mmol/L), mean±SD	8.80±3.02	4.50±1.66	0.286
Lymphocyte (%), median (IQR)	0.98 (0.39, 1.07)	1.39 (1.18, 2.86)	0.014
Neutrophil (%),mean±SD	4.22±0.95	3.75±1.32	0.773

Abbreviations: Cr, creatinine; BUN, blood urea nitrogen; IQR, interquartile range; n, number; SD, standard deviation; TC, total cholesterol; TG, triglyceride; y, year.

Supplementary table 2. Spearman analysis for correlation about serum SRP14.

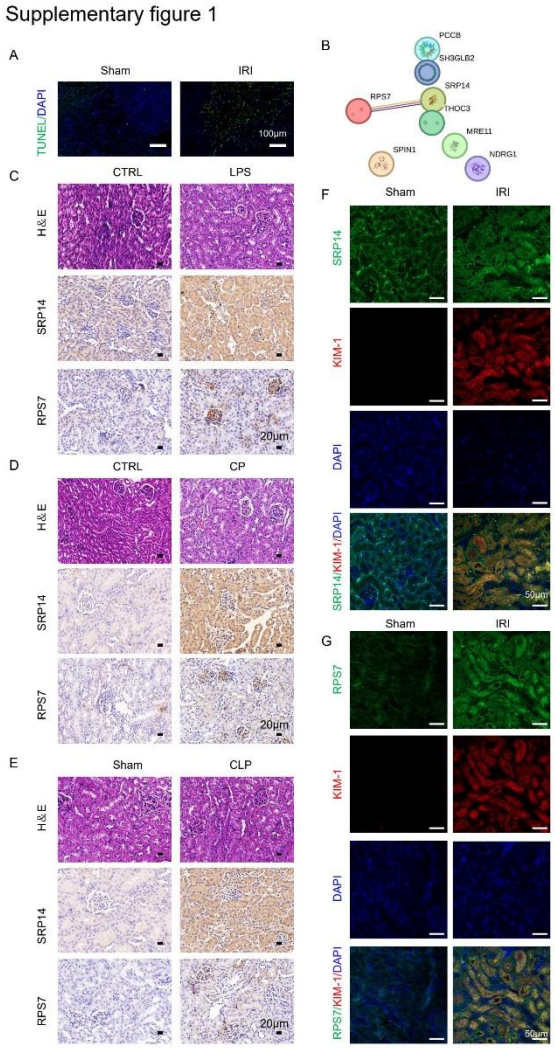
Spearman analysis	ρ	p	95 % CI
Serum creatinine-SRP14	0.31	0.038*	0.0088 ± 0.56
BUN-SRP14	0.49	0.00054#	0.23 ± 0.69
eGFR-SRP14	-0.30	0.043*	-0.55 ± -0.0019
Uric acid-SRP14	0.39	0.0076#	0.10 ± 0.62
Total cholesterol -SRP14	-0.19	0.22	-0.46 ± 0.12
TG-SRP14	0.16	0.28	-0.15 ± 0.44
HCT-SRP14	-0.06	0.72	-0.35 ± 0.25
Lymphocyte-SRP14	-0.26	0.081	-0.52 ± 0.04
Neutrophilicgranulocyte-SRP14	0.51	0.00032#	0.25 ± 0.70
Platelet -SRP14	-0.0089	0.95	-0.31 ± 0.29
KIM-1-SRP14	0.55	0.00010#	0.29 ± 0.73
TIMP-2-SRP14	0.15	0.31	-0.15 ± 0.44
Age- SRP14	0.32	0.030*	0.025 ± 0.57
HB- SRP14	-0.061	0.69	-0.36 ± 0.25

"*" represents "p <0.05"; "#" represents "p <0.01".

Supplementary table 3. The primers for genotyping of *SRP14*$^{-/-}$, Ggt-Cre mice.

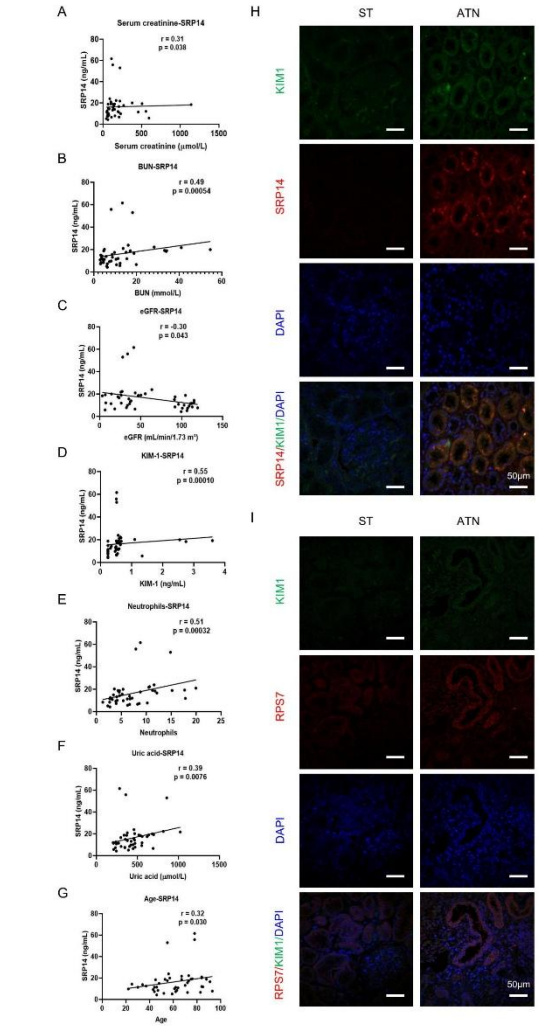
Species	Gene	Forward sequence (5' to 3')	Reverse sequence (5' to 3')
Mouse	Srp14	CTGTCAAGGTAAAGCAAGGCTACA	GGACCTGTCTCATGAAACTCTATCT
Mouse	Ggt1	CATCACATCAGGCACCCCAGAA-3	GAACATCTTCAGGTTCTGCGGGA

Supplementary figures with legends



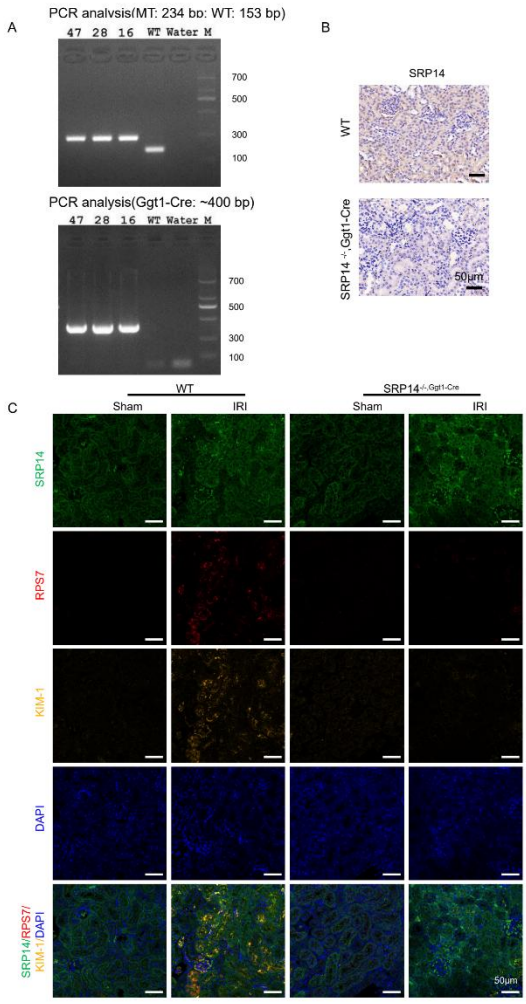
Supplementary figure 1. A. TUNEL staining revealed obvious apoptosis in kidney of IRI mice. Scale bar: white = 100 μm. **B.** PPI analysis predicted an interaction between SRP14 and RPS7. **C.** H&E staining and immunohistochemistry staining of SRP14 and RPS7 in renal tissues from including LPS injection (n = 6). Scale bar: black = 20 μm. **D.** H&E staining and immunohistochemistry staining of SRP14 and RPS7 in renal tissues from cisplatin injection (n = 6). Scale bar: black = 20 μm. **E.** H&E staining and immunohistochemistry staining of SRP14 and RPS7 in renal tissues from CLP (n = 6). **F.** Immunofluorescent staining of SRP14 and KIM-1 in murine kidneys. **G.** Immunofluorescent staining of RPS7 and KIM-1 in murine kidneys. Scale bar: white = 50 μm. "IRI" represents "ischemia/reperfusion injury". "CP" represents "Cisplatin injection to induce AKI"; "CLP" represents "Cecum ligation and puncture to induce AKI".

Supplementary figure 2



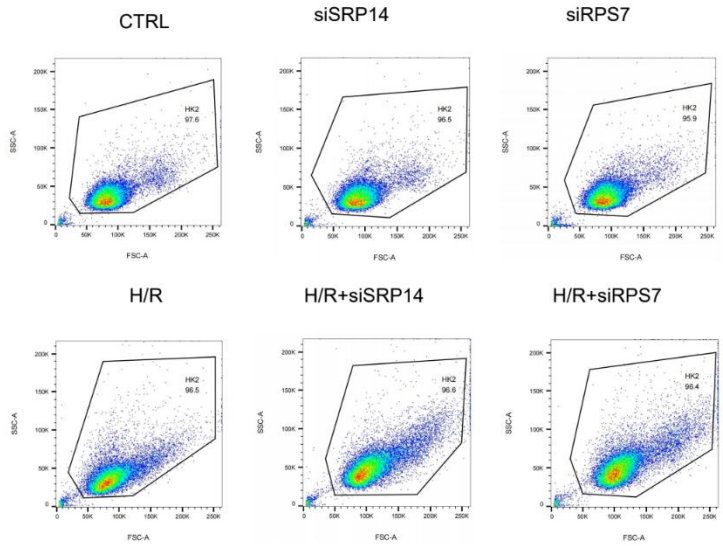
Supplementary figure 2. Serum SRP14 was tested in 45 candidates, among which 29 candidates were AKI patients. AKI stages were defined by the KDIGO definition. **A.** The correlation analysis of these constant with SRP14 included the correlation between SRP14 and serum creatinine (Spearman's ρ=0.31, p=0.038). **B.** The correlation analysis of these constant with SRP14 included the correlation between SRP14 and BUN (Spearman's ρ=0.49, p=0.00054). **C.** The correlation analysis of these constant with SRP14 included the correlation between SRP14 and eGFR (Spearman's ρ=-0.30, p=0.043). **D.** The correlation analysis of these constant with SRP14 included the correlation between SRP14 and KIM-1 (Spearman's ρ=0.55, p=0.00010). **E.** The correlation analysis of these constant with SRP14 included the correlation between SRP14 and Neutrophils (Spearman's ρ=0.51, p=0.0032). **F.** The correlation analysis of these constant with SRP14 included the correlation between SRP14 and Uric acid (Spearman's ρ=0.39, p=0.0076). **G.** The correlation analysis of these constant with SRP14 included the correlation between SRP14 and Age (Spearman's ρ=0.32, p=0.030). **H.** Immunofluorescent staining of SRP14 and KIM-1 in human kidneys. **I.** Immunofluorescent staining of RPS7 and KIM-1 in human kidneys. Renal tissue specimens from patients with acute tubular necrosis (ATN) (n = 5) and Normal renal tissue surrounding the tumor (ST) (n = 4) from patients with renal carcinoma were collected. Scale bar: white = 50 μm.

Supplementary figure 3

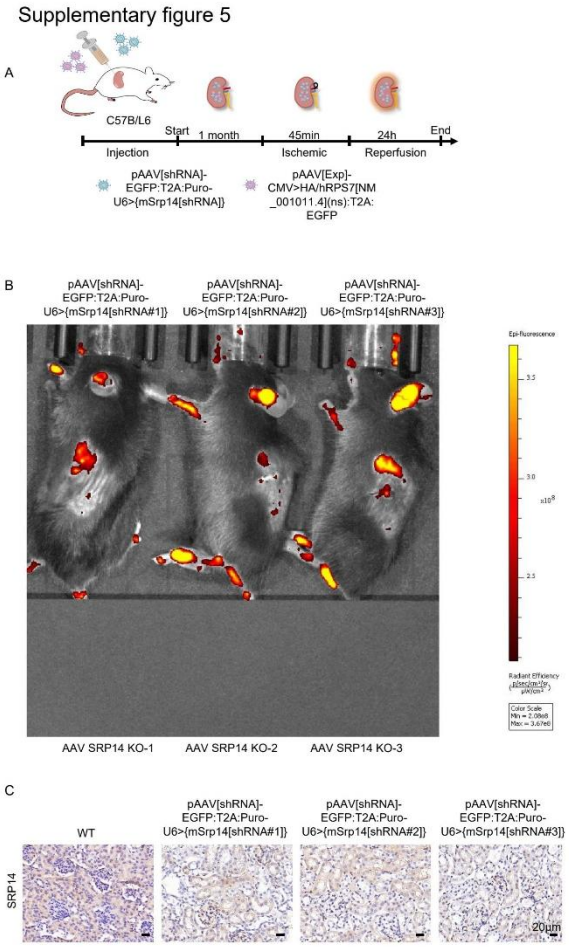


Supplementary figure 3. To explore the role of SRP14 in renal tubules, tubule specific-SRP14 knockout (*SRP14*$^{-/-}$, *Ggt-Cre*) mice were generated by a *Cre-LoxP* recombination system. **A.** Tail genotyping measurement of *SRP14*$^{-/-}$, Ggt-Cre mice. **B.** Immunohistochemistry staining of SRP14 in renal tissues of *SRP14*$^{-/-}$, Ggt-Cre mice. Scale bar: black = 50 μm. **C.** Immunofluorescent staining of SRP14, RPS7 and KIM-1 in murine kidneys. Scale bar: white = 50 μm. "IRI" represents "ischemia/reperfusion injury".

Supplementary figure 4

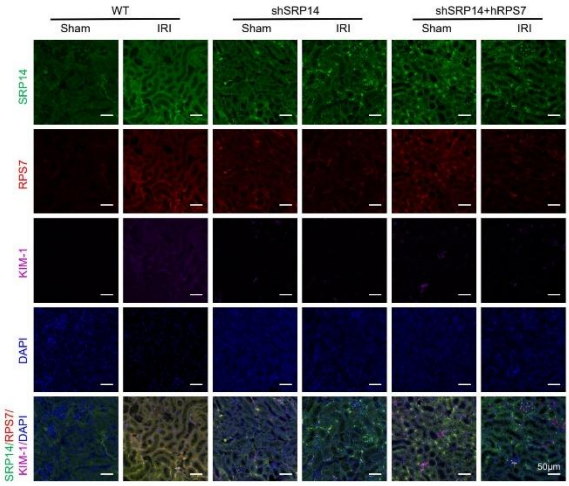


Supplementary figure 4. The gating strategy for Annexin V FITC/PI staining. “H/R” represents “hypoxia/reoxygenation”.



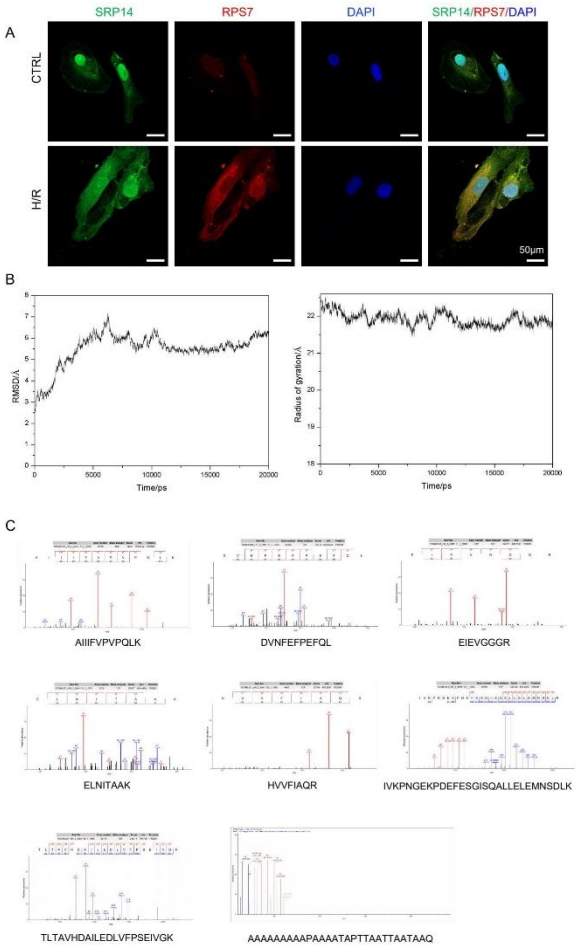
Supplementary figure 5. A. Schematic diagram shows the experimental procedure. **B.** The optical *in vivo* imaging system has confirmed the successful delivery for the recombinant adeno-associated virus vector harboring shRNA-SRP14. **C.** Immunohistochemistry measurement of SRP14 have further verified the reduction of SRP14 expression in murine kidney by the recombinant adeno-associated virus vector harboring shRNA-SRP14. Scale bar: black = 50 μm.

Supplementary figure 6



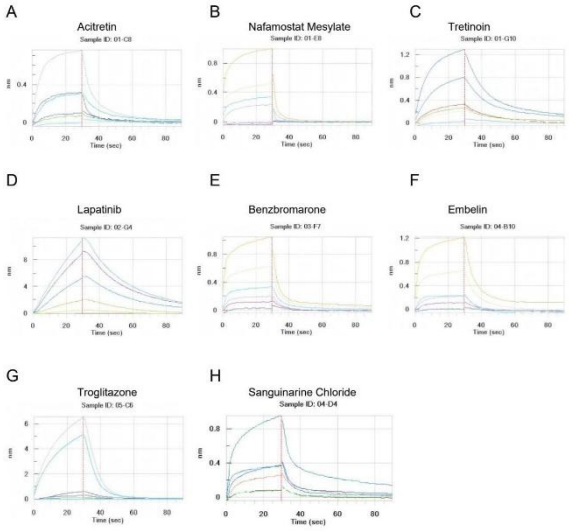
Supplementary figure 6. Immunofluorescent staining of SRP14, RPS7 and KIM-1 in murine kidneys (n = 6). Scale bar: white = 50 μm. “shSRP14” represents “The recombinant adeno-associated virus vector harboring shRNA-SRP14 was delivered into the kidney of C57 BL/6 male mice by intrarenal injection”. “hRPS7” represents “The mice accept intrarenal injection of the hRPS7 fusion gene recombinant adeno-associated virus vector injection”. “IRI” represents “ischemia/reperfusion injury”.

Supplementary figure 7



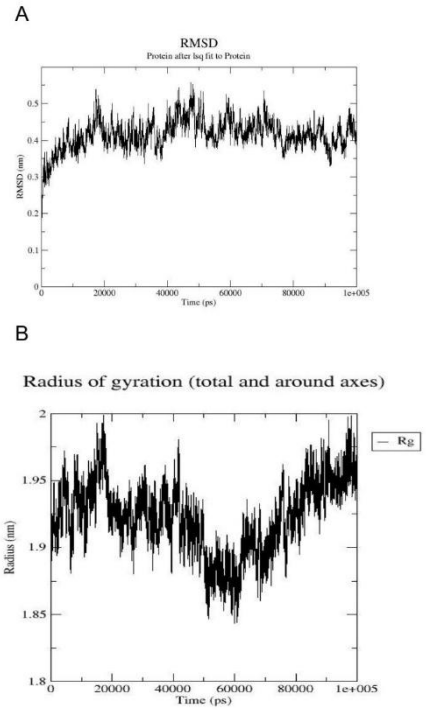
Supplementary figure 7. A. Confocal microcopy showed significant colocalization of SRP14 and RPS7 in renal tubular epithelial cells after hypoxia and reoxygenation treatment. **B.** The molecular simulation for the SRP14-RPS7 complex. **C.** The potential binding domain of SRP14 and RPS7 tested by mass spectra. “H/R” represents “hypoxia/reoxygenation”.

Supplementary figure 8



Supplementary figure 8. The results of biolayer interferometry about screened 8 small molecule compounds. **A.** The results of biolayer interferometry about Acitretin. **B.** The results of biolayer interferometry about Nafamostat Mesylate. **C.** The results of biolayer interferometry about Tretinoin. **D.** The results of biolayer interferometry about Lapatinib. **E.** The results of biolayer interferometry about Benzbromarone. **F.** The results of biolayer interferometry about Embelin. **G.** The results of biolayer interferometry about Troglitazone. **H.** The results of biolayer interferometry about Sanguinarine Chloride.

Supplementary figure 9



Supplementary figure 9. The molecular simulation for the RPS7 and Nafamostat mesilate complex.

Supplemental complete materials and methods

Subjects

Patients who admitted in Nephrology Department of Sichuan Provincial People's Hospital during June to December, 2018 were included in this study. Patients with confirmed diagnosis of end stage kidney disease (defined as eGFR<15 mL/min/1.73 m^2) at admission were excluded. Demographic information of patients was collected, including age and sex. Fast serum samples were taken 48 hours after admission or earlier if needed. The samples were stored at -20 ℃ before measurements. Kidney biopsies of acute tubular necrosis (ATN) patients were collected with the clinical information showed in Supplementary table 2. Normal renal tissue surrounding tumor (ST) from patients with renal carcinoma were collected as the normal control. Written informed consents were obtained from patients before conducting any study procedure. All the procedure followed the Declaration of Helsinki and approved by the ethics committee of Sichuan Provincial People's Hospital (No.2018-176 and No. 2018-284).

Measurement and AKI definition

AKI was defined by the Kidney Disease: Improving Global Outcomes (KDIGO) definition as an increase in serum creatinine by > 26.5 μmol/L within 48 h or > 1.5 times baseline within 7 days [1]. At confirmation of AKI, AKI stages were further defined as KDIGO definition: Stage 1: increase in serum creatinine by ≥ 26.5 μmol/L within 48 hours or > 1.5 times baseline within 7 days; Stage 2: increase in serum creatinine by 2 to 2.9 times baseline within 7 days; Stage 3: increase in serum creatinine by ≥ 3.0 times baseline or increase in serum creatinine by > 353.6 μmol/L

or initiation of renal replacement therapy (RRT). The baseline creatinine was defined as creatinine level measured at admission. The level of SRP14 in the fast serum samples at diagnosis of AKI was determined using ELISA kit (#ZC-54503, Zhuochai biology, Shanghai, China).

Routine indexes were also measured concurrently with the determination of occurrence of AKI, including uric acid, serum albumin, hemoglobin, triglyceride, and total cholesterol. eGFR was calculated using CKD-EPI equation [2]. Patients who developed AKI within 48 hours after admission were included in the AKI group, whereas patients without AKI were included in the control group.

Animal studies

Male C57 BL/6 mice aged from 6 to 8 weeks and weighed from 18 to 22 g were purchased from Chengdu Dossy experimental animals co., LTD (Chengdu, China). Male tubule specific SRP14 knockout ($SRP14^{-/-}$, Ggt-Cre) mice were obtained by Cyagen biosciences (Suzhou, China). All animals were housed in the animal center of Sichuan provincial people's hospital under a specific pathogen free condition with a 12-hour light/dark cycle, humidity from 40 % to 70 %, and ambient temperature from 18 to 22 ℃. The mice were allowed to freely get food and water. All the animal studies were approved by the ethics committee of Sichuan provincial people's hospital (No.2018-176 and No. 2020-215).

After anesthetized the mice with 50 mg/kg pentobarbital sodium (Merk, Germany) by intraperitoneal injection, we clamped the bilateral renal arteries with artery clamps (#RS-5420, ROBOZ, USA) at 37 ℃ for 45 min to establish the murine AKI model

induced by IRI. Mice in the sham group accept the same procedure without clamp. As describe as Daniel Rittirsch et al [3], we established the sepsis AKI model by the cecal ligation and puncture. Mice in the sham group accept the same procedure without the cecal ligation and puncture. We also respectively intraperitoneally injected mice with 10 mg/kg LPS (#L2630, Sigma-Aldrich, USA) and 15 mg/kg Cisplatin (#479306, Sigma-Aldrich, USA) to induce AKI. As the normal control, mice injected with equal volume of saline. Nafamostat mesylate (at dosages of 20 mg/kg.d, 25 mg/kg.d, and 50 mg/kg.d) and Tretinoin (at a dosage of 20 mg/kg.d) were respectively orally treated to mice with renal IRI.

The genotyping of SRP14$^{-/-}$, Ggt-Cre mice

The SRP14 $^{lox\ P/lox\ P}$ mice were hybridized with the Ggt-Cre transgenic mice (#012841, The Jackson Laboratory, USA) to produce heterozygous offspring for SRP14 $^{lox\ P/+}$, Ggt-Cre mice. Then hybridized the SRP14 $^{lox\ P/+}$, Ggt-Cre mice and Ggt-Cre transgenic mice to produce SRP14$^{-/-}$, Ggt-Cre mice. The primers for genotyping are as below: SRP14 F: 5'-CTGTCAAGGTAAAGCAAGGCTACA-3', R: 5'-GGACCTGTCTCATGAAACTCTATCT-3'; Cre: F: 5'-GAACGCACTGATTTCGACCA-3', R: 5'-GCTAACCAGCGTTTTCGTTC-3'. And the PCR reaction was performed as the instruction for Trelief mouse direct PCR kit for Genotyping (#TSE014, Tsingke, China).

AAV vectors and luciferase imaging in live animals

pAAV[shRNA]-EGFP:T2A:Puro-U6>{mSrp14[shRNA]} and pAAV[Exp]-CMV>HA/hRPS7[NM_001011.4](ns):T2A:EGFP were purchased from

VectorBuilder Inc.(Guangzhou, China). Mice were injected in situ with AAV vector, at a dose of 7×10^{10} GC per gram of body weight. After four weeks, mice were anesthetized and images were recorded using IVIS Lumina XRMS Series III (PerkinElmer, Waltham, MA, US).

Assessment of renal function

Blood samples were collected from mice and centrifuged at 3000 rpm for 15mins. The supernatant was used to analyze the level of creatine (#C011-2, Nanjing Jiancheng Biotech., Nanjing, China) and urea nitrogen (#C013-2, Nanjing Jiancheng Biotech., Nanjing, China) by assay kits.

Routine histology and immunohistochemistry

The kidney tissues of human and mice were fixed by paraffin and then cut into 2 μm thick sections. Routine histological measurements such as Hematoxylin-Eosin (HE) (#sC231202, Baso, China) staining and Periodic acid-Schiff (PAS) (#C241001, Baso, China) staining were performed on the renal sections. The renal tissue slices were respectively incubated with anti-SRP14 antibody (1:100, #NBP2-94184, Novus, USA) and anti-RPS7 antibody (1:50, #SC-100834, Santa Cruz, USA) at 4 ℃ overnight. After cleaning, the sections were incubated with appropriate horseradish peroxidase labeled secondary antibody (#K5007, Dako Producs, Denmark) at 37 ℃ for one hour.

Multiplex immunofluorescence staining and multi-spectral imaging

The multiplex immunofluorescence staining was performed on 2 μm renal tissue paraffin sections from human and mice by Opal 4 color Manual IHC staining Kit

(Akoya Bioscience, USA). Primary antibodies including anti-SRP14 antibody (1:100, #NBP2-94184, NOVUS, USA), anti-RPS7 antibody (1:50, #SC-100834, Santa Cruz, USA), and anti-KIM-1 antibody (1:400, #NBP-43761, NOVUS, USA) were used for multiplex immunofluorescence staining. Then sections were observed by the Confocal microscopy (#LSM900, ZEISS, Germany).

TdT-mediated dUTP Nick-End Labeling (TUNEL)

Murine renal tissues were fixed by paraffin and then cut into 2 μm thick sections. *In situ* apoptosis measurement was performed on the renal tissue slices by the DeadEnd Fluorometric TUNEL System (#G3250, Promega, USA). Confocal microscopy (#LSM900, ZEISS, Germany) observed the production of fluorescein-12-dUTP-labeled DNA.

Cell culture and treatments

Human renal cortex proximal tubular epithelial cell line HK2 cells were bought from National Collection of Authenticated Cell Cultures (#GNHu47, National Collection of Authenticated Cell Cultures, Shanghai, China). The HK2 cells were cultured by Dulbecco's Modified Eagle's Medium/F-12 (DMEM/F-12) (#C11330500BT, Gibco, USA) containing 10 % fetal bovine serum (#10099-141c, Gibco, USA). The cells were hypoxic with 0 % Oxygen and 95 % Nitrogen with 5 % Carbon dioxide by the Cell hypoxia/hyperoxia workstation (#MiniStation Plus-MPS230418047, Gene Science, Chongqing, USA). For regeneration, the cells were cultured in 21 % Oxygen and 74 % Nitrogen with 5 % Carbon dioxide. HK2 cells were respectively transfected with SRP14 siRNA (#SIGS0008000-4, RIBOBIO, Guangzhou, China) or RPS7

siRNA (#SIGS0001928-4, RIBOBIO, Guangzhou, China) for 48 h using Lipofectamine 3000 Transfection Reagent (#L3000015, Thermo fihser, USA), and then treated by hypoxia for 8 h and reoxygenation for 24 h. Cells were washed 3 times by PBS, then harvested and prepared for other experiments.

Protein sample preparation for cells

The frozen sample was taken out to a 1.5 ml centrifuge tube and 300 μl of a lysate containing a protease inhibitor was added. Ultrasonic disruption on ice, centrifugation to remove the supernatant, and repeat the operation once. Acetone was added to the supernatant, and the precipitate was collected by centrifugation at -20 ℃ overnight. After drying at room temperature, it is dissolved in SDS, and the mixture is centrifuged at room temperature, and the supernatant is centrifuged, and the operation is repeated once. The supernatant is the total protein solution of the cells, and the protein is quantified by BCA, and the enzymatic hydrolysis is carried out by the FASP method. After enzymatic hydrolysis, the peptide was dissolved in 0.1% TFA and desalted using a RP-C18 solid phase extraction column.

Liquid chromatography-mass spectrometry

The hydrolyzed products were analyzed by LC-MS/MS, and each sample was analyzed three times. Separation was carried out using a nanoliter flow rate HPLC liquid phase system (EASY-nLC1000). The digested product was separated and analyzed using an LTQ Orbitrap Velos Pro mass spectrometer. The first- and second-stage mass spectra were collected using profile and centroid methods to reduce the data file size.

Maxquant Analysis

The original file of the library LC-MS/MS was searched using Maxquan software for analysis. The uniprot database and the protein information were further analyzed by MaxQuant software, and the identification ratio was calculated from all the identified peptides. Further use the OmicsBean cloud platform to screen the differential proteins for gene function annotation (GO), analyze the subcellular localization of differential proteins, molecular functions and specific functions of biology; then perform differential protein analysis by KEGG to analyze protein expression. The signal path that may be involved in the function.

CCK-8 assessment

The apoptosis compound library (#Cherry Pick Library(96-well)-L2000-Z439858, Shanghai, Selleck, USA) including 356 FDA approved compounds were purchased from the Selleck Company. 1×10^4 HK2 cells were seeded into 96-well plates. After hypoixa and reoxygen, 10 μl CCK-8 solution (#CK04-500, Dojindo, Tokyo, Japan) was added into each well. After incubating for 4 h, the optical density was detected at 450 nm using a measured microplate reader (#680, BIORAD, USA).

Flowcytometry

2×10^5 HK2 cells were seeded into six well plates and respectively transfected with SRP14 siRNA and RPS7 siRNA. After hypoxia/reoxygenation, the cells were collected to perform flowcytometry with a Becton Dickinson FAC Sort cytometer (For Annexin V, excitation: 633 nm; emission, 660 nm. For Propidium Iodide, excitation: 488 nm; emission, 580 nm.) by the Annexin V Alexa Fluor 488 &

Propidium Iodide cell apoptosis detection kit from DOJINDO Laboratories (#AD11, DOJINDO, Japan).

The purification of protein

According to requirements for the study, prepared the LB liquid and solid media. Respectively constructed the RSP7 and SRP14 expression plasmid. And proceeded with the expression and transformation of the proteins. Utilized the HisTrap HP affinity chromatography pre-packed column (GE, USA) and the AKTA protein purification system (GE, USA). Collected purified protein samples with a 280 nm UV absorption peak using an automatic collector. Identified the target protein in the elution peak by SDS-PAGE gel electrophoresis, to ensure a purity of over 95 %.

Biolayer Interferometry

To analysis the interaction between SRP14 and RPS7, we respectively synthesized and expressed SRP14 recombinant protein and RPS7 recombinant protein by pET21b (+) prokaryotic expression plasmid. RPS7 recombinant protein was biotinylated by kit (G-MM-IGT, Genemore, China) following the manufacturer's protocol. The SA sensor (Fortebio, Pall Life Sciences, Menlo Park, USA) was loaded with biotinylated RPS7 for 600 seconds. After being washed with PBS, the sensors were dipped into SRP14 recombinant protein at the concentration from 88.8 nM to 355 nM for 60 seconds association followed by 180 seconds disassociation in PBS. Kinetics were recorded using Octet K2 (Fortebio, Pall Life Sciences, Menlo Park, USA) at 1,000 rpm vibration and analysed by Octet Data Analysis HT 11.1 software.

The apoptosis compound library including 356 FDA approved compounds were

purchased from the Selleck Company (USA). For compound screening about RPS7, The SSA sensor (Fortebio, Pall Life Sciences, Menlo Park, USA) was loaded with biotinylated RPS7 for 600 seconds. After being washed with PBS, the sensors were dipped into solutions containing the compounds at the corresponding concentration for 60 seconds association followed by 180 seconds disassociation in PBS. Kinetics were recorded using Octet K2 (Fortebio, Pall Life Sciences, Menlo Park, USA) at 1,000 rpm vibration and analysed by Octet Data Analysis HT 11.1 software.

Molecular Docking and Dynamics Simulation

To analysis the interaction between SRP14 and RPS7, the 3D structures of SRP14 and RPS7 were respectively constructed by homologous modeling techniques (modeler 9.18) with crystal structures 1E8O (PDB ID) and 5k0y (PDB ID) as templates. Utilized Zdock online server (http://zdock.umassmed.edu/) to integrate SRP14 and RPS7. All docking parameters were default. And the top 1 conformation was selected as the docking conformation for subsequent molecular dynamics simulations. A 20000 ps molecular dynamics simulation was conducted on the SRP14-RPS7 complex. The simulation process shows that the complex tends to equilibrium after 8000ps. Therefore, the binding free energy of SRP14-RPS7 was calculated using the dynamic trajectory after 8000ps.

To analysis the interaction between Nafamostat mesilate and RPS7, the 3D structures of RPS7 was constructed by homologous modeling techniques (modeler 9.18) with crystal structures 5k0y (PDB ID) as the template. Utilized Zdock online server (http://zdock.umassmed.edu/) to integrate Nafamostat mesilate and RPS7. All docking

parameters were default. And the top 1 conformation was selected as the docking conformation for subsequent molecular dynamics simulations.

Immunoprecipitation and immunoblotting

Total proteins from murine renal tissues and cells were extracted by a lysis buffer (#P0013B, Beyotime, China) with a protease inhibitor (#ST506-2, Beyotime, China). For immunoprecipitation, 20 μL protein A/G agarose was added to each sample and incubated for 1 h at room temperature. The total cell lysates with 500 μg were immunoprecipitated with RPS7 antibody (#SC-100834, Santa Cruz, USA) or SRP14 antibody (#SC-377012, Santa Cruz, USA) overnight at 4 ℃. The immunoprecipitants were separated by12.5 % SDS-PAGE. Western blot analysis was performed using specific antibodies for SRP14 and RPS7.

For western blot analysis, proteins were denatured by addition of 5 × loading buffer and boiled at 100 ℃ for 5 mins. Proteins were separated by SDS-PAGE and electrotransferred to PVDF membrane (#ISEQ00010, Merck Millipore, Germany), which were then blocked in TBS buffer containing 5 % BSA and 0.1 % Tween 20 at room temperature for 1 h. The membranes were incubated with primary antibodies at 4 ℃ overnight and then with the corresponding secondary antibodies conjugated to horseradish peroxidase at room temperature for 1 h. The protein bands were detected using the β-actin (1:1000, #6008, Proteintech, China) protein levels were used as a loading control. Primary antibodies included SRP14 (1:1000, #NBP2-94184, NOVUS, USA), RPS7 (1:1000, #PA5-77005, Invitrogen, USA), Bax (1:1000, #ab182733, Abcam, Britain), Cleaved Caspase3 (1:1000, #9664s, Cell signaling

technology, USA), p53(1:1000, #ab131442, Abcam, Britain), MDM2(1:1000, #ab259265, Abcam). The secondary antibodies included HRP-labeled Goat Anti-Rabbit (1:5000, #511203, ZENBIO, China) and HRP-labeled Goat Anti-Mouse (1:10000, #511103, ZENBIO, China). The signals were detected by the Immobilon western chemilum HRP substrate (#WBKLS-638173, Millipore, Merck, USA). The signals were visualized and analyzed by a chemiluminescence image analysis system (#5200, Tanon, Shanghai, China).

Statistical analysis

Continuous variables were expressed as mean ± standard deviation (SD) under normally distribution or median and interquartile range (IQR) under skewed distribution. The differences of variables between AKI and control groups were compared by unpaired Student's t test, Mann-Whitney U test, or chi square test. Correlation analyses were done for variables that had significant difference between AKI and control groups. The levels of SRP14 among groups of different AKI stages were compared using one-way analysis of variance (ANOVA) analysis. $P<0.05$ was considered statistically significant. All data analysis was conducted using SPSS for Windows version 29.0 (IBM Corporation, Chicago, IL, USA) and GraphPad Prism version 8.0.1 (GraphPad Software, La Jolla, CA, USA).

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