



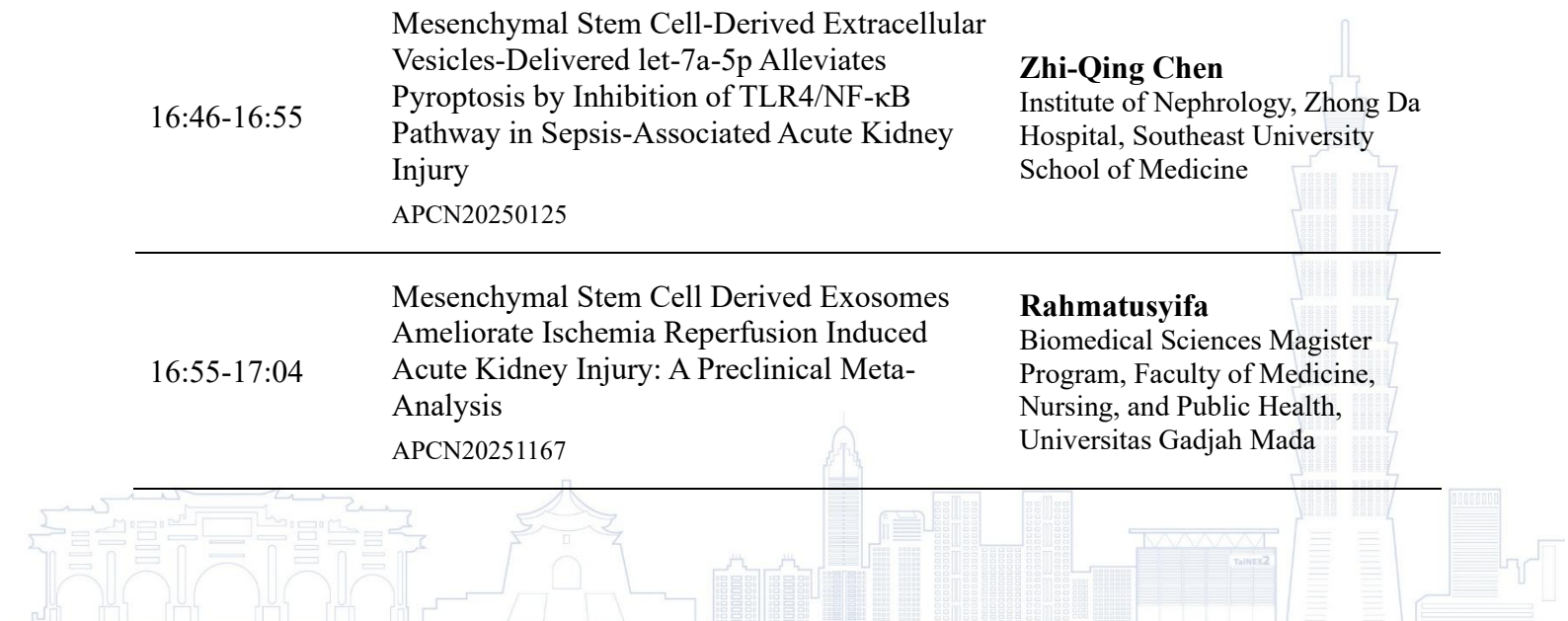
Oral Communications 1

Acute Kidney Injury (AKI)

December 5, 2025 (Friday) 16:10~17:40

Venue : Room 7 (703)

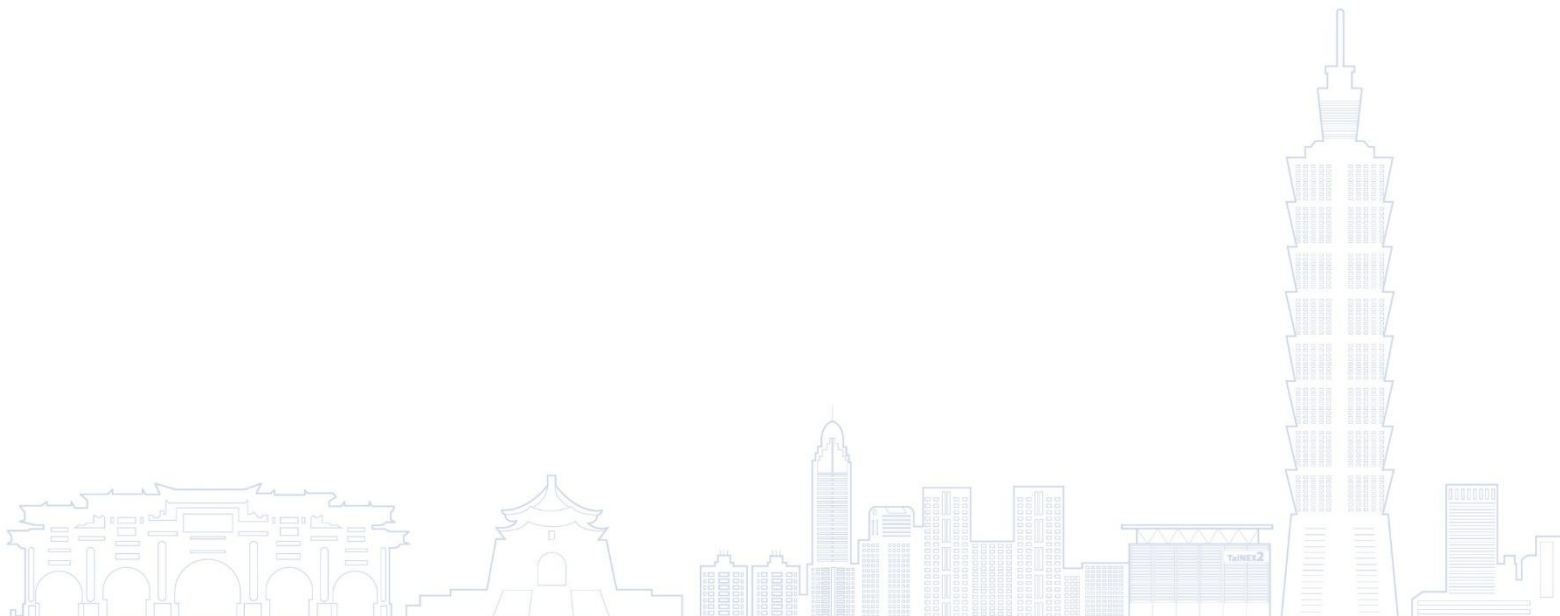
Chair(s)	Saruultuvshin Adiya, Chun-Te Huang	
16:10-16:19	MSC-EVs Ameliorates Septic AKI by Delivering miR-125a-5p Targeting TNFR2/NLRP3 axis to Block Pyroptosis APCN20250144	Chen Feng Institute of Nephrology, Zhong Da Hospital, Southeast University School of Medicine
16:19-16:28	IP3R2 Orchestrates Calcium-Independent ER-Mitochondrial Signalling to Protect Proximal Tubular Cells from Anoxia-Reoxygenation APCN20250796	Midori Sakashita Division of CKD Pathophysiology, Graduate School of Medicine, The University of Tokyo
16:28-16:37	Low One-Week Nrf2 Identifies Septic AKI Patients Unlikely to Recover Renal Function Despite Adequate Antioxidant Enzymes APCN20251057	Tsai-Jung, Wang Division of Nephrology, Department of Internal Medicine, Taichung Veterans General Hospital
16:37-16:46	Silver Nanoparticles of Resveratrol Attenuate Renal Damage in Acute Kidney Injury Induced by Glycerol Via Apoptosis and Anti-Inflammatory Effect APCN20250763	Ekta Yadav Department of Pharmaceutical Sciences, SHUATS
16:46-16:55	Mesenchymal Stem Cell-Derived Extracellular Vesicles-Delivered let-7a-5p Alleviates Pyroptosis by Inhibition of TLR4/NF-κB Pathway in Sepsis-Associated Acute Kidney Injury APCN20250125	Zhi-Qing Chen Institute of Nephrology, Zhong Da Hospital, Southeast University School of Medicine
16:55-17:04	Mesenchymal Stem Cell Derived Exosomes Ameliorate Ischemia Reperfusion Induced Acute Kidney Injury: A Preclinical Meta-Analysis APCN20251167	Rahmatusyifa Biomedical Sciences Magister Program, Faculty of Medicine, Nursing, and Public Health, Universitas Gadjah Mada





APCN x TSN 2025
23rd Asian Pacific Congress of Nephrology
Dec. 5 Fri. ▶ Dec. 7 Sun., 2025 TaiNEX 2, Taipei, Taiwan

17:04-17:13	Comparative Effectiveness of SGLT2 Inhibitors Versus GLP-1 Receptor Agonists on incident Dementia in Type 2 Diabetes with Acute Kidney Disease: A Target Trial Emulation Study APCN20251153	Chen, Ying Ru National Taiwan University Hospital
17:13-17:22	Renoprotective Effect of Intravenous Amino Acid Infusion in Adult Patients Undergoing Cardiac Surgery: A Systematic Review and Meta-Analysis APCN20250016	Ramon Jr Larrazabal Section of Nephrology, Department of Internal Medicine, Cebu Doctors' University Hospital
17:22-17:31	Exploration of the Renal Autonomous Cholinergic System via Single-Cell RNA Sequencing APCN20251118	Chia-Hsien Wu Department of Physiology of Visceral Function and Body Fluid, Graduate School of Biomedical Sciences, Nagasaki University
17:31-17:40	Effect of a Multidisciplinary Team Approach on Major Adverse Kidney Events in Patients with Acute Kidney Disease: A Randomized Clinical Trial (ISACC Trial) APCN20251231	Duong Toan Trung Division of Nephrology, Department of Internal Medicine, Shuang Ho Hospital, Taipei Medical University



Oral Communications : AKI

Abstract Submission No. : APCN20250144

MSC-EVs ameliorates septic AKI by delivering miR-125a-5p targeting TNFR2/NLRP3 axis to block pyroptosis

陈丰^{1,2}; Tao-Tao Tang¹; Zhi-Qing Chen¹; Bi-Cheng Liu¹

¹ Institute of Nephrology, Zhong Da Hospital, Southeast University School of Medicine, Nanjing, China

² Department of Nephrology, Second Affiliated Hospital of Guangzhou Medical University, Guangzhou, China

Abstract

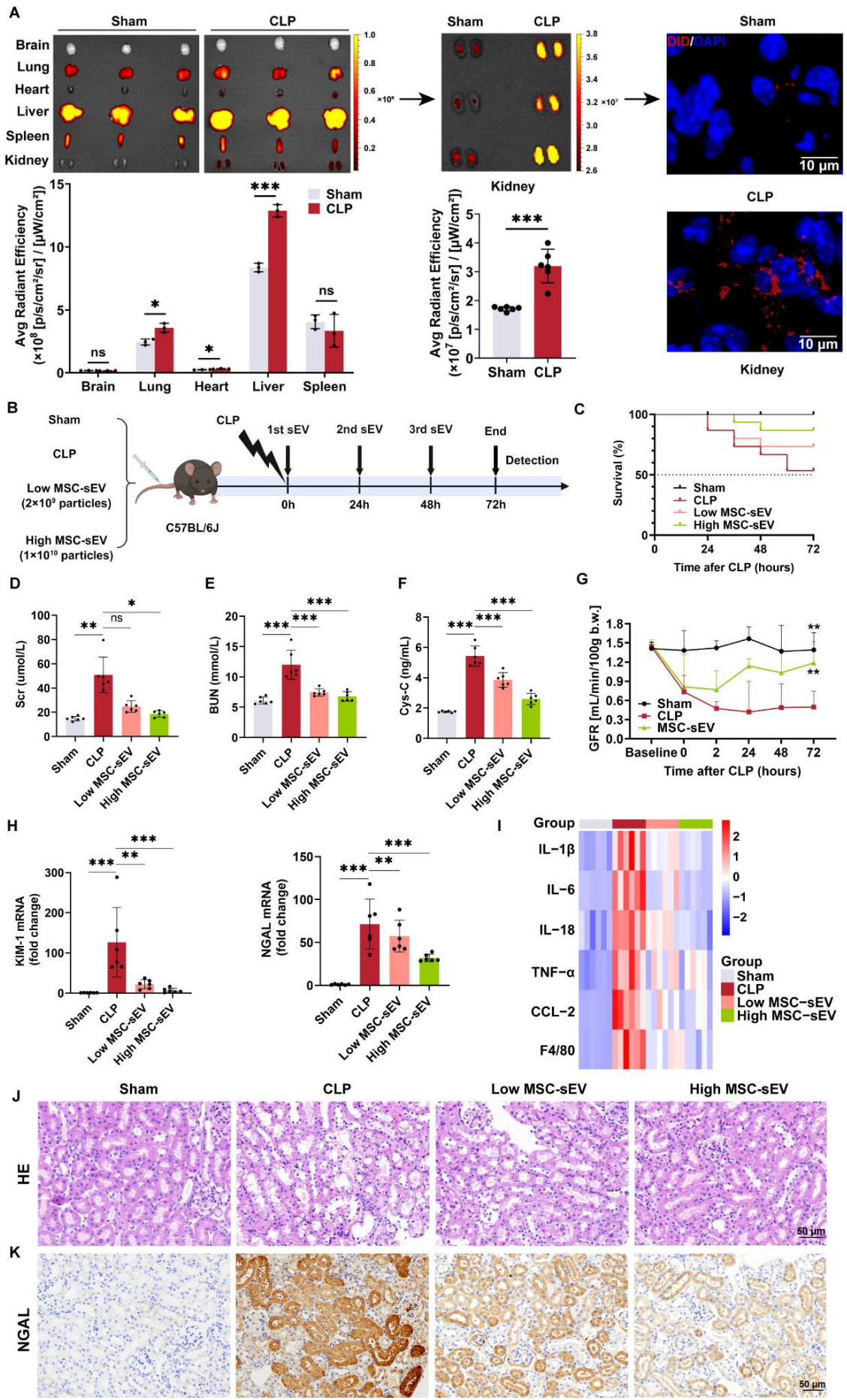
Background: Acute kidney injury (AKI) is an acute renal dysfunction that can be caused by a variety of etiologies. AKI occurs in approximately 10%-15% of hospitalized patients and more than 50% of patients in the intensive care unit. Notably, sepsis accounts for more than 50% of AKI cases, with a mortality rate of up to 40%. However, there is no specific effective treatment other than supportive care. Extracellular vesicles derived from human umbilical cord mesenchymal stem cells (hucMSC-EVs) have the capacity for intercellular signaling communication, making them a novel therapeutic strategy for various diseases. The aim of this study was to investigate the renal protective effects and mechanisms of hucMSC-EVs in sepsis.

Methods: The hucMSC-EVs were obtained and characterized. In vivo and in vitro models of S-AKI were established by cecal ligation and puncture (CLP) and LPS, respectively. The distribution of hucMSC-EVs was observed using the IVIS imaging system and confocal microscopy. The renal function, inflammatory response and activation of related signal pathways were evaluated by RNA sequencing, ELISA, Western blot, qRT-PCR, and immunohistochemistry/fluorescence etc.

Results: In vivo, IVIS showed that hucMSC-EVs homed significantly after CLP-induced renal injury, and hucMSC-EVs intervention decreased Scr, BUN and cys-C, and alleviated renal pathological damage in CLP mice. In addition, the mRNA levels of KIM-1, IL-1 β , IL-6, TNF- α reduced. Similarly, in vitro, confocal microscopy and flow cytometry showed that the uptake of hucMSC-EVs by LPS-stimulated HK-2 cells was significantly increased, the inflammatory response of LPS-stimulated HK-2 cells was inhibited, and ROS levels were reduced. Mechanically, RNA sequencing revealed that CLP induced upregulated genes were mainly associated with inflammatory pathways, while hucMSC-EVs induced upregulation was reversed. Similarly, pyroptosis-related genes, particularly GSDMD, were significantly altered. Combined with exosomal miRNA sequencing and bioinformatics analysis and qRT-PCR validation, miR-125a-5p, which is highly enriched in hucMSC-EVs, was demonstrated to ameliorate renal injury by reducing pyroptosis by targeting TNFR2/NLRP3 signaling pathway. In contrast, inhibition of miR-125a-5p in hucMSCs attenuated the protective effect of hucMSC-EVs against S-AKI.

Conclusion: Collectively, we reveal a novel potential therapeutic mechanism for S-AKI in which hucMSC-EVs may modulate TNFR2/NLRP3 signaling by miR-125a-5p to inhibit pyroptosis, alleviating kidney damage and thereby improving survival.

Keywords : Mesenchymal stem cell; Extracellular vesicles; Acute kidney injury; microRNAs; Pyroptosis



Oral Communications : AKI

Abstract Submission No. : APCN20250796

IP3R2 Orchestrates Calcium-Independent ER-Mitochondrial Signalling to Protect Proximal Tubular Cells from Anoxia–Reoxygenation

Midori Sakashita^{1,2}; Qi Li^{1,2}; Yuto Takenaka^{1,2}; Madina Saipidin; Kenzo Hirose³; Shigeyuki Namiki³; Masaomi Nangaku²; Reiko Inagi¹

¹ Division of CKD Pathophysiology, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan

² Department of Nephrology and Endocrinology, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan

³ Department of Pharmacology, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan

Abstract

Background: Acute kidney injury (AKI) after ischemia–reperfusion centers on proximal-tubule (PT) dysfunction and often progresses to chronic disease. Type-2 inositol-1,4,5-trisphosphate receptor (IP3R2) is an endoplasmic-reticulum (ER) Ca²⁺ channel enriched at mitochondria-associated ER membranes (MAMs) that has been linked to aging and cellular senescence, yet its influence on tubular stress responses remains unknown. This study aimed to elucidate a calcium-independent role for IP3R2 in regulating PT cell fate during anoxia–reoxygenation (AR) and ER-mitochondrial crosstalk.

Methods: HK-2 and primary RPTEC cultures were first transfected with selective siRNA to silence IP3R2. Cells were subjected to 24-hour anoxia followed by 4-hour reoxygenation. Cell viability was assessed by CCK-8 and LDH assays, whereas apoptosis was evaluated by Annexin-V/7-AAD staining and JC-1 monitoring of mitochondrial membrane potential. Cell-cycle distribution was assessed by flow cytometry and immunoblotting of p27 and phosphorylated CDK2. Mitochondrial morphology was visualized with MitoTracker, and oxidative phosphorylation was profiled by Seahorse extracellular-flux analysis of basal and maximal oxygen-consumption rate (OCR) and ATP production. Finally, intracellular Ca²⁺ dynamics were recorded with Fluo-4-AM and Fura-2-AM imaging.

Results: Our data demonstrate that specific knockdown of IP3R2 significantly improved HK-2 and RPTEC cell viability after AR injury. Annexin-V and JC-1 assays showed that AR-induced early and late apoptosis were attenuated by silencing IP3R2. AR injury prominently induced a G1 phase arrest, accompanied by a significant upregulation of the cell cycle inhibitor p27. Crucially, IP3R2 knockdown effectively alleviated this AR-induced G1 phase arrest and robustly suppressed the upregulation of p27, leading to a recovery of phosphorylated CDK2 levels and reduced apoptotic cell death. Furthermore, IP3R2 knockdown preserved mitochondrial morphology and maintained bioenergetic competence despite modestly lower maximal respiration and ATP-linked OCR, indicating adaptive energy conservation rather than failure. The induction of senescence-associated secretory phenotype (SASP: TGF- β 1 and IL-6) by AR was also significantly ameliorated by IP3R2 knockdown. Intriguingly, these protective effects occurred independently of significant changes in intracellular calcium flux.

Conclusion: Our findings unveil a novel calcium flux-independent role for IP3R2 in preserving renal proximal tubular cell survival after AR injury. IP3R2 appears to modulate cell cycle progression by interacting with the cell cycle inhibitor p27, thereby preventing AR-induced G1 arrest and subsequent apoptosis. These results provide new insights into calcium-independent mechanisms of ER-mitochondrial crosstalk in tubular injury.

Keywords : AKI, MAM, mitochondria, cell cycle

Oral Communications : AKI

Abstract Submission No. : APCN20251057

Low One-Week Nrf2 Identifies Septic AKI Patients Unlikely to Recover Renal Function Despite Adequate Antioxidant Enzymes

Tsai-Jung, Wang^{1,2,3}

¹ Division of Nephrology, Department of Internal Medicine, Taichung Veterans General Hospital, Taichung, Taiwan

² Department of Critical Care Medicine, Taichung Veterans General Hospital, Taichung, Taiwan

³ Department of Nutrition, Chung Shan Medical University, Taichung, Taiwan

Abstract

Introduction: Sepsis-associated acute kidney injury (AKI) has high mortality but lacks robust early prognostic biomarkers. Nuclear factor erythroid 2-related factor 2 (Nrf2) has recently emerged as a promising indicator in kidney disease and a potential target for therapeutic intervention. While Nrf2 activators have shown efficacy in chronic kidney disease and diabetic nephropathy, their relevance in the context of AKI has been less extensively studied. In this study, we examined whether peripheral blood Nrf2 expression could predict 28-day renal outcomes in septic patients with AKI.

Methods: We prospectively enrolled 124 ICU adults with sepsis. Nrf2 mRNA was measured from buffy coat leukocytes by qPCR on days 0 and 7 of ICU admission ($\Delta\text{Ct}\%$, normalized to GAPDH). In parallel, we measured serum concentrations of downstream antioxidant markers, including reduced glutathione (GSH), oxidized glutathione (GSSG), the GSH/GSSG ratio, and enzymatic activities of glutathione peroxidase (GPx) and glutathione reductase (GR). AKI was defined according to the KDIGO criteria and occurred in 88 patients (71%). The primary outcome was major adverse kidney events by day 28 (MAKE28), defined as initiation of dialysis, a doubling of serum creatinine, or death. Associations between Nrf2 expression and MAKE28 were evaluated using multivariable logistic regression models adjusted for baseline clinical covariates.

Results: Admission (day 1) Nrf2 expression did not differ between patients who did and did not experience MAKE-28. Furthermore, Nrf2 levels at both day 1 and day 7 showed no cross-sectional correlation with serum antioxidant markers. However, by day 7, Nrf2 levels were significantly lower in AKI patients who developed MAKE-28 compared to those who recovered (median 6.0% vs. 9.8%, $p = 0.009$). In multivariable logistic regression restricted to the AKI subgroup, each 1% increase in day-7 Nrf2 was independently associated with a reduced risk of MAKE-28 (adjusted OR 0.899; 95 % CI 0.809–0.998; $p = 0.046$).

Conclusion: Blunted Nrf2 response by day 7 predicted poor renal outcomes in sepsis, independent of antioxidant enzyme levels. Nrf2 may reflect broader redox dysregulation or delayed regulatory dynamics beyond the first week, and merits further evaluation as both a prognostic and therapeutic target in septic AKI.

Keywords : acute kidney injury, critical care, Nrf2, antioxidant, oxidative stress

Oral Communications : AKI

Abstract Submission No. : APCN20250763

Silver nanoparticles of resveratrol attenuate renal damage in acute kidney injury induced by glycerol via apoptosis and anti-inflammatory effect

Ekta Yadav¹

¹ Department of Pharmaceutical Sciences, SHUATS, Prayagraj

Abstract

Introduction: Acute kidney injury (AKI) is the most common severe clinical complication of kidney dysfunction, leading to chronic kidney injury. Microbial infection, surgeries, and exposure to heavy metals are the causative factors for AKI. Besides the availability of chemical medications having inevitable toxic effects, there is a strict need to develop natural-based treatments with high safety against AKI. Nowadays, silver nanoparticles have drawn the attraction in biomedical applications due to their unique physical, chemical, and biological characteristics. Considering this, the study was designed to explore the protective effect of biofabricated silver nanoparticles of resveratrol (AgR) against AKI model.

Methods: Wistar rats were used for the study, and they were randomly divided into different groups: control, negative control treated with glycerol (Gly), Gly+(AgNO₃), Gly+AgR (5 mg/kg), and Gly+AgR (10 mg/kg). All the groups were administered with respective treatments for 14 days, and analyzed for different biomarker levels at the end of treatment to estimate the efficacy of AgR against AKI.

Results: Group treated with Gly exhibited significant elevation in different rhabdomyolysis-related markers, including creatine kinase and lactate dehydrogenase, along with other parameters such as kidney injury molecule-1, neutrophil gelatinase-associated lipocalin, relative kidney weight, level of creatinine, and serum urea. A decline in antioxidant enzyme levels, as well as Nfe2l2 and Hmox-1 expressions, was observed in the Gly-treated group, accompanied by an increase in renal MDA and NO concentrations. Pretreatment with AgR significantly restored the damaging effects induced by Gly. Results also evidenced the downregulation of renal inflammation by reducing the levels of TNF- α , IL-6, IL-1 β , and Nos2 expression. Alleviation in Bax, caspase-3, and cytochrome-c, as well as Bcl-2 and Pipk3 expression (renal damage sign), were significantly modulated by AgR in a dose-dependent manner. Co-administration of AgR significantly reversed the AKI alterations in terms of molecular, biochemical, and histological structure.

Conclusion: AgR treatment could be a beneficial tool against AKI-associated renal damage by regulating oxidative stress, inflammation, and apoptosis.

Keywords : nanoparticles, resveratrol, AKI, oxidative stress, inflammation

Oral Communications : AKI

Abstract Submission No. : APCN20250125

Mesenchymal Stem Cell-Derived Extracellular Vesicles-Delivered let-7a-5p Alleviates Pyroptosis by Inhibition of TLR4/NF- κ B Pathway in Sepsis-Associated Acute Kidney Injury

Zhi-Qing Chen¹; Tao-Tao Tang¹; Feng Chen²; Ri-Ning Tang^{1,3}; Bi-Cheng Liu¹

¹ Institute of Nephrology, Zhong Da Hospital, Southeast University School of Medicine, Nanjing, China.

² School of Clinical Medicine, Tsinghua University, Beijing, China.

³ Department of Nephrology, Nanjing Drum Tower Hospital, Affiliated Hospital of Medical School, Nanjing University, Nanjing, China

Abstract

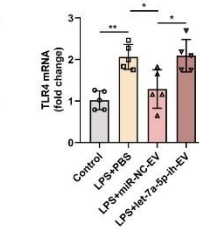
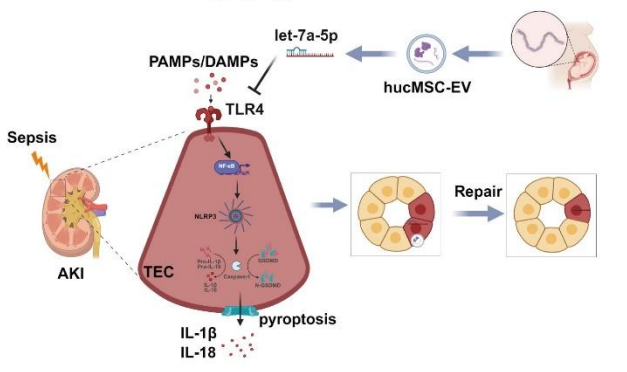
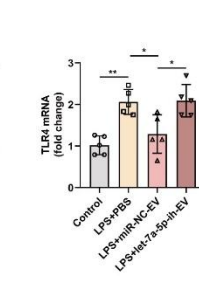
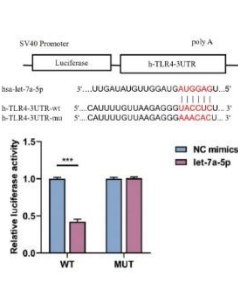
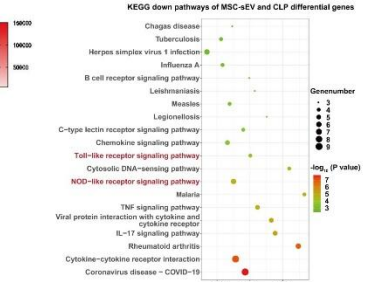
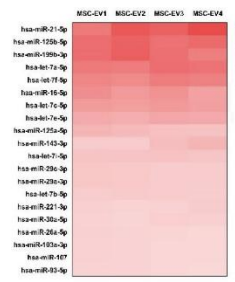
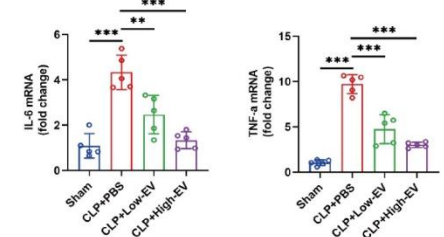
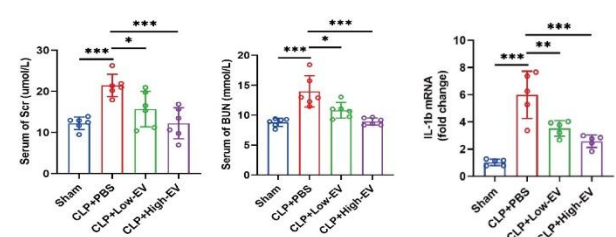
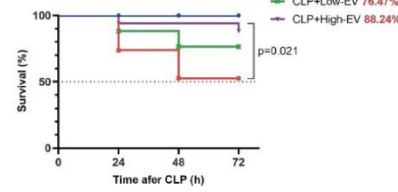
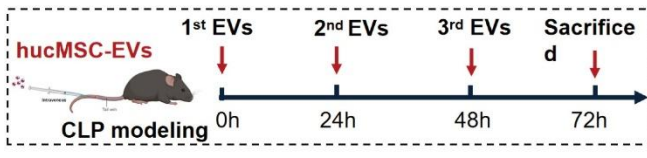
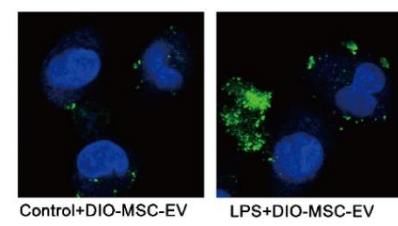
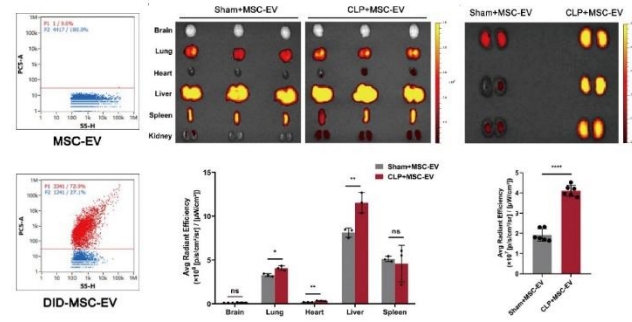
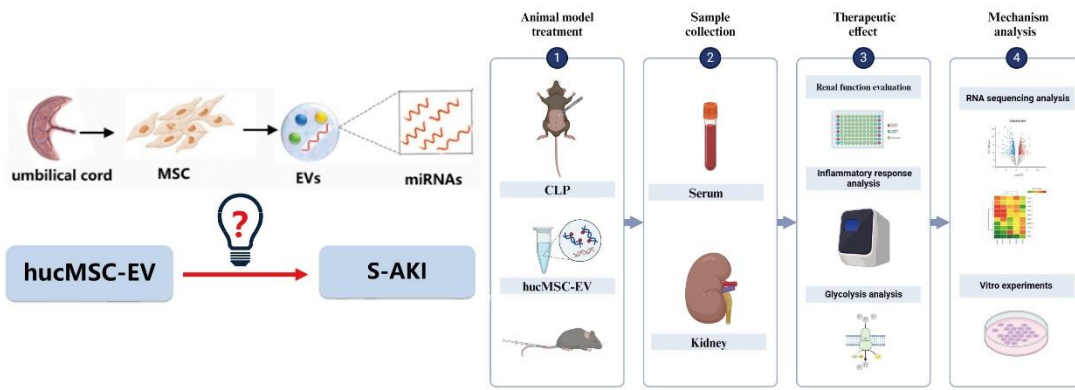
Introduction: Human umbilical cord mesenchymal stem cell-derived extracellular vesicles (hucMSC-EVs) have shown great therapeutic potential as a cell-free therapy in various kidney diseases. However, the role and underlying mechanisms of hucMSC-EVs in sepsis-associated acute kidney injury (SA-AKI) remain poorly understood.

Methods: hucMSC-EVs were purified and characterized. Animal and cell models of SA-AKI were established using cecal ligation and puncture (CLP) and LPS, respectively. The biodistribution of hucMSC-EVs in mice and cellular uptake in HK-2 cells were detected by the IVIS imaging system and confocal microscopy, respectively. The effects of hucMSC-EVs on survival rate, renal function, and histopathological changes in SA-AKI mice were evaluated. Subsequently, mRNA and protein expression levels of inflammation- and pyroptosis-related genes were detected in both renal tissues and HK-2 cells following hucMSC-EVs administration. miRNA profiles in hucMSC-EVs were analyzed by high-throughput miRNA sequencing. Differentially expressed genes after hucMSC-EVs treatment were identified by tissue RNA-seq and the potential regulatory mechanisms were further validated using a series of experiments.

Results: hucMSC-EVs homed to the injured kidneys in a dose-dependent manner, alleviated body weight loss, increased survival and improved renal function in SA-AKI mice. Similarly, confocal microscopy further revealed that LPS-induced injury in HK-2 cells increased the uptake of hucMSC-EVs. Furthermore, hucMSC-EVs significantly inhibited inflammation and pyroptosis both in vivo and in vitro. Mechanistically, let-7a-5p, which is highly enriched in hucMSC-EVs, directly targeted and inhibited the TLR4/NF- κ B pathway in tubular epithelial cells. Finally, inhibition of let-7a-5p attenuated the renoprotective effect of hucMSC-EVs against SA-AKI.

Conclusion: We demonstrate that let-7a-5p delivered by hucMSC-EVs ameliorates SA-AKI by alleviating inflammation and pyroptosis through targeted inhibition of the TLR4/NF- κ B pathway. This study provides a new theoretical and experimental foundation for the clinical application of hucMSC-EVs in SA-AKI treatment.

Keywords : Keywords: Mesenchymal stem cell; Extracellular vesicles; Sepsis; Acute kidney injury



Oral Communications : AKI

Abstract Submission No. : APCN20251167

Mesenchymal Stem Cell Derived Exosomes Ameliorate Ischemia Reperfusion Induced Acute Kidney Injury: A Preclinical Meta-Analysis

Rahmatusyifa¹; Astia Anelia¹; Wimbi Kartika Ratnasari¹

¹ Biomedical Sciences Magister Program, Faculty of Medicine, Nursing, and Public Health, Universitas Gadjah Mada, Indonesia

Abstract

Background:

Acute kidney injury (AKI) affects 10–15% of hospitalized patients and over 50% of ICU cases. Exosomes derived from mesenchymal stem cells (MSCs) have shown renoprotective effects in animal models mimicking AKI through ischemia-reperfusion, yet their efficacy has not been systematically quantified. This meta-analysis evaluates MSC-derived exosomes ameliorate renal ischemia-reperfusion injury (IRI) in preclinical studies.

Methods:

A systematic review was conducted by screening 362 studies retrieved from five databases: PubMed, Scopus, Web of Science, Elsevier, and Google Scholar, focusing on exosomes derived from mesenchymal stem cell therapy for IRI. Seven outcome parameters were measured: serum creatinine (SCr), blood urea nitrogen (BUN), tubular damage, apoptotic cells, inflammatory markers interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), and the apoptotic marker caspase-3. Data were analyzed using Review Manager (RevMan version 5.4), and study quality was assessed using the ROBINS-I (Risk Of Bias In Non-randomized Studies - of Interventions) tool.

Results:

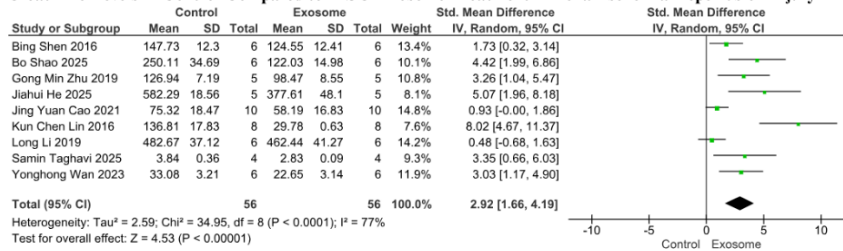
A total of 11 in vivo studies published between 2016 and 2025, involving 132 animals, were included. Exosome doses ranged from 50 to 250 μ g, with diameters of 120 - 140 nm. At 24 hours post-treatment, exosomes significantly reduced SCr (SMD = 2.92; 95% CI: 1.66–4.19; $P < 0.00001$, $I^2 = 77\%$) BUN (SMD = 2.76; 95% CI: 1.34–4.17; $P = 0.0001$, $I^2 = 74\%$), tubular damage (SMD = 1.28; 95% CI: 0.70–1.86; $P < 0.0001$, $I^2 = 44\%$), and apoptotic cell counts (SMD = 4.61; 95% CI: 1.65–7.57; $P = 0.002$, $I^2 = 88\%$) compared to IRI model controls. Secondary analyses demonstrated attenuation of inflammatory markers, including IL-6 (SMD = 3.45; 95% CI: -0.24–7.15; $P = 0.07$, $I^2 = 88\%$), TNF- α (SMD = 5.27; 95% CI: 1.72–8.81; $P = 0.004$, $I^2 = 76\%$), as well as the apoptotic marker caspase-3 (SMD = 5.20; 95% CI: 2.18–8.22; $P = 0.0007$, $I^2 = 77\%$). No exosome-related adverse effects were reported in any of the included studies.

Conclusions:

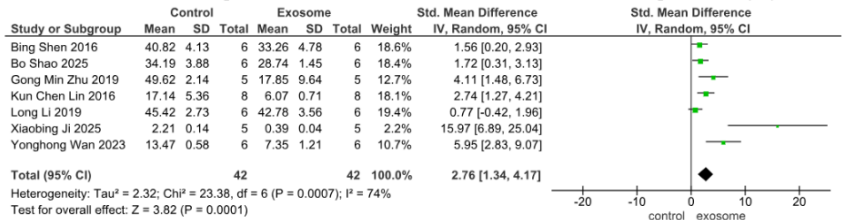
MSC-derived exosomes significantly improve renal function and attenuate tissue damage, apoptosis, and inflammation in preclinical models of ischemia reperfusion induced acute kidney injury.

Keywords : Acute Kidney Injury, Exosomes, In Vivo, Ischemia-Reperfusion Injury, Mesenchymal Stem Cells

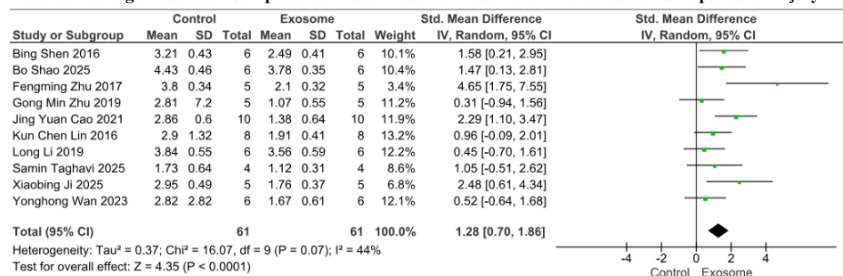
Creatinine Levels in Control Compared to MSC-Exosome Treatment in Renal Ischemia-Reperfusion Injury



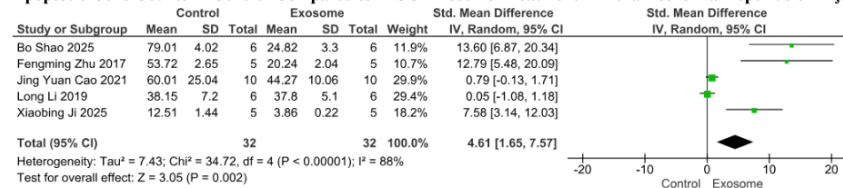
BUN Levels in Control Compared to MSC-Exosome Treatment in Renal Ischemia-Reperfusion Injury



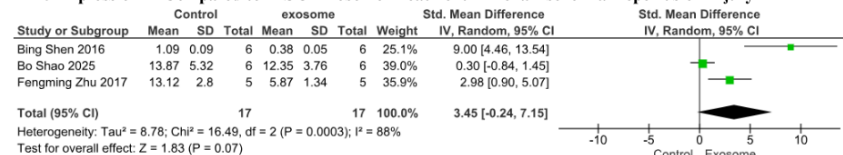
Tubular Damage in Control Compared to MSC-Exosome Treatment in Renal Ischemia-Reperfusion Injury



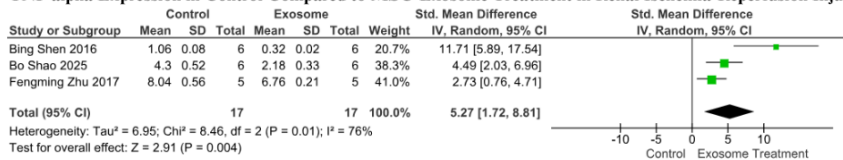
Apoptotic Cells Counts in Control Compared to MSC-Exosome Treatment in Renal Ischemia-Reperfusion Injury



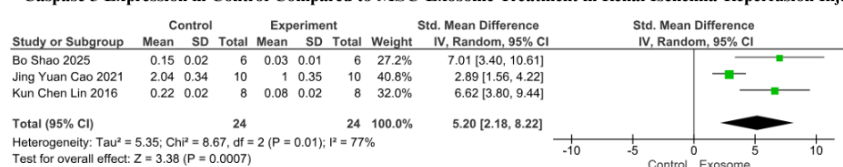
IL 6 Expression in Control Compared to MSC-Exosome Treatment in Renal Ischemia-Reperfusion Injury



TNF alpha Expression in Control Compared to MSC-Exosome Treatment in Renal Ischemia-Reperfusion Injury



Caspase 3 Expression in Control Compared to MSC-Exosome Treatment in Renal Ischemia-Reperfusion Injury



Oral Communications : AKI

Abstract Submission No. : APCN20251153

Comparative Effectiveness of SGLT2 Inhibitors Versus GLP-1 Receptor Agonists on incident Dementia in Type 2 Diabetes with Acute Kidney Disease: A Target Trial Emulation Study

chen, ying ru¹; Jiang, Zheng-Hong²; Jui-Yi Chen³; Vin-Cent Wu²

¹ National Taiwan University Hospital, Taipei City, Taiwan

² Division of Nephrology, Internal Medicine, National Taiwan University Hospital, Taipei, Taiwan

³ Division of Nephrology, Department of Internal Medicine, Chi-Mei Medical Center, Tainan, Taiwan

Abstract

Background :

Previous studies have demonstrated an increased risk of dementia following acute kidney injury (AKI). There are beneficial effects of newer glucose-lowering drugs (GLDs), including glucagon-like peptide-1 receptor agonists (GLP-1RAs) and sodium-glucose co-transporter-2 inhibitors (SGLT2i), in reducing the risk of all-cause dementia in individuals with type 2 diabetes (T2D). Our study aims to evaluate the association between the use of GLP-1RAs and SGLT2i and the risk of dementia in patients who have experienced AKI.

Methods :

This cohort study utilized data from the TriNetX Research Network. The study included patients without dementia, aged 18 or older, who had been detached from temporary dialysis and survived for at least 90 days following discharge from the hospital. Participants were grouped based on their use of either SGLT2i or GLP-1RAs, with data collection spanning from January 1, 2015, and January 1, 2024. Developing dementia, all-cause mortality, major adverse cardiovascular events (MACE), and major adverse kidney events (MAKE), were assessed over a period of three months to five years post-discharge.

Results :

Among 14460 individuals who could withdraw from acute dialysis, 7707 patients were prescribed with SGLT2i, while another 6753 were GLP-1RAs users. After performing 1:1 propensity score matching, there were 4773 patients in each group. Patients prescribed SGLT2i demonstrated a significantly lower risk of dementia (aHR of 0.78 (95% CI: 0.625–0.973, p = 0.0282) and MAKE (aHR = 0.821, 95% CI: 0.713–0.945, p = 0.0063) compared to GLP-1RAs users. However, there was no significant difference in all-cause mortality (aHR= 0.961, 95% CI 0.832-1.109) and MACE(aHR= 1.145, 95% CI 0.996-1.317) between the two groups.

Conclusions :

SGLT2 inhibitors significantly reduced the risk of dementia and major kidney events compared to GLP-1 agonists in type 2 diabetes with acute kidney disease, highlighting their superior protective effects.

Keywords : sodium-glucose cotransporter 2 inhibitors (SGLT2i), glucagon-like peptide-1 receptor agonists (GLP-1RAs), acute kidney disease, dementia

Oral Communications : AKI

Abstract Submission No. : APCN20250016

Renoprotective Effect of Intravenous Amino Acid Infusion in Adult Patients Undergoing Cardiac Surgery: A Systematic Review and Meta-Analysis

Ramon Jr Larrazabal¹; Radjuli, Laila²; Tan, Alexander Jr. U.¹

¹ Section of Nephrology, Department of Internal Medicine, Cebu Doctors' University Hospital, Cebu City, Philippines

² Department of Internal Medicine, Cebu Doctors' University Hospital, Cebu City, Philippines

Abstract

Introduction: Acute kidney injury (AKI) complicates 5–42% of cardiac surgeries, with severe cases requiring renal replacement therapy (RRT) (1–5%) linked to mortality rates up to 63%. Despite extensive research, no pharmacologic intervention has proven definitively effective for prevention. This study evaluates whether amino acid infusion reduces AKI incidence, RRT requirement, and 30-day mortality post-cardiac surgery.

Methods: A systematic search (PubMed/MEDLINE, CENTRAL, Embase, Web of Science) up to April 1, 2025, identified randomized controlled trials (RCTs) comparing perioperative amino acid infusion with standard care in adults undergoing cardiac surgery. Outcomes included AKI incidence, RRT initiation, and 30-day mortality. Fixed-effects meta-analyses (Review Manager 5.4) calculated pooled odds ratios (OR) with 95% confidence intervals (CI).

Results: Three studies (3646 participants) were included. There was no significant heterogeneity among the studies. Amino acid infusion had decreased incidence of AKI (overall) by 22% (OR 0.78, 95% CI: 0.68, 0.90; P = 0.0008) and reduced the number of patients needed to be initiated on RRT by 45% (OR 0.55, 95% CI: 0.33, 0.93; P = 0.02) post-cardiac surgery. Lastly, our analysis did not show a significant effect on 30-Day mortality. However, there was a trend towards benefit favoring the amino acid group.

Conclusion: Perioperative amino acid infusion significantly lowers AKI risk and RRT need in cardiac surgery patients, supporting its potential as a preventive strategy. While mortality effects remain inconclusive, these findings highlight its clinical relevance for mitigating postoperative kidney injury in high-risk populations.

Keywords : Amino Acid, Acute Kidney Injury, Critical Care Nephrology, Cardiac Surgery

Study or Subgroup	Amino Acid		Placebo		Weight	Odds Ratio M-H, Fixed, 95% CI	Odds Ratio M-H, Fixed, 95% CI
	Events	Total	Events	Total			
Kazawa 2024	9	33	14	32	2.4%	0.48 [0.17, 1.36]	
Landoni 2024	474	1759	555	1752	96.1%	0.80 [0.69, 0.92]	
Pu 2019	3	33	7	35	1.5%	0.40 [0.09, 1.70]	
Total (95% CI)		1825		1819	100.0%	0.78 [0.68, 0.90]	
Total events	486		576				
Heterogeneity: Chi ² = 1.71, df = 2 (P = 0.42); I ² = 0%							
Test for overall effect: Z = 3.36 (P = 0.0008)							

Oral Communications : AKI

Abstract Submission No. : APCN20251118

Exploration of the Renal Autonomous Cholinergic System via Single-Cell RNA Sequencing

Chia-Hsien Wu¹; Yasuna Nakamura¹; Umene Ryusuke¹; Yuri Yamada¹; Tsuyoshi Inoue¹

¹ Department of Physiology of Visceral Function and Body Fluid, Graduate School of Biomedical Sciences, Nagasaki University, Nagasaki, Japan

Abstract

Introduction

Vagus nerve stimulation (VNS) has been reported to exert anti-inflammatory and organ-protective effects, including attenuation of acute kidney injury (AKI). These effects are believed to be mediated through acetylcholine (ACh) and nicotinic acetylcholine receptors (nAChRs), particularly the $\alpha 7$ subtype. However, whether the kidney possesses an intrinsic cholinergic system capable of local ACh production and signaling has remained unclear. This study aimed to identify ACh-secreting renal cells and elucidate how this system contributes to kidney function and protection, using single-cell RNA sequencing (scRNA-seq).

Methods

To investigate renal ACh-producing cells, we utilized ChAT-Cre:R26GRR reporter mice, in which cholinergic cells express fluorescence, enabling isolation by FACS for scRNA-seq analysis. Separately, wild-type mice received electrical VNS, followed 24 hours later by bilateral renal ischemia-reperfusion injury (IRI) to induce AKI. Kidneys were harvested 24 hours post-IRI for scRNA-seq profiling. In addition, $\alpha 4$ nicotinic receptor ($\alpha 4$ nAChR) knockout mice were generated using CRISPR/Cas9 to investigate receptor-specific roles in cholinergic signaling.

Results

scRNA-seq analysis revealed that ACh-producing cells are broadly distributed in the kidney, including proximal tubules, the loop of Henle, vascular endothelial cells, and immune cells. Among these, proximal tubule cells exhibited strong expression of choline acetyltransferase (ChAT), suggesting active ACh synthesis. VNS significantly upregulated the expression of phosphoenolpyruvate carboxykinase (Pck1), a key gluconeogenic enzyme, specifically in proximal tubules. This upregulation peaked at 4 hours post-stimulation, as confirmed by qPCR. Furthermore, $\alpha 4$ nAChR was selectively expressed in Slc5a12-positive proximal tubule cells. Notably, VNS-induced Pck1 expression was abolished in $\alpha 4$ nAChR-deficient mice, and these mice exhibited more severe tubular injury after AKI, indicating a protective role of $\alpha 4$ nAChR-mediated signaling.

Conclusion

These findings support the existence of a renal autonomous cholinergic system, independent of central parasympathetic input, capable of regulating tubular metabolism and contributing to kidney protection. Specifically, ACh derived from proximal tubular cells may act in an autocrine or paracrine manner via $\alpha 4$ nAChR to promote Pck1 expression and metabolic resilience during injury. This previously unrecognized mechanism offers new insight into renal physiology and potential therapeutic strategies for AKI.

Keywords : Vagus nerve stimulation, scRNA-seq, acetylcholine

Oral Communications : AKI

Abstract Submission No. : APCN20251231

Effect of a Multidisciplinary Team Approach on Major Adverse Kidney Events in Patients with Acute Kidney Disease: A Randomized Clinical Trial (ISACC Trial)

DUONG TOAN TRUNG^{3,6}; Yu-Wei Chen^{1,2}; Alex PA Nguyen^{4,5}; Cai-Mei Zheng^{1,2}; Mei-Yi Wu^{1,2}; Chia-Te Liao^{1,2}; Mai-Szu Wu^{1,2}

¹ Division of Nephrology, Department of Internal Medicine, Shuang Ho Hospital, Taipei Medical University, New Taipei City, Taiwan

² Division of Nephrology, Department of Internal Medicine, College of Medicine, School of Medicine, Taipei Medical University, Taipei, Taiwan

³ College of Medicine, International Graduate Program in Medicine, Taipei Medical University

⁴ Institute of Data Science, College of Management, Taipei Medical University, New Taipei City, Taiwan

⁵ TMU Research Center of Urology and Kidney, Taipei Medical University, Taipei, Taiwan

⁶ Hemodialysis Department, Cho Ray Hospital, Ministry of Health, Ho Chi Minh City, Viet Nam

Abstract

Background: Acute kidney disease (AKD) represents a critical transitional phase between acute kidney injury (AKI) and chronic kidney disease (CKD). Multidisciplinary team (MDT) care has been proposed as a strategy to improve patient outcomes. The ISACC trial investigated the efficacy of an MDT intervention in reducing Major Adverse Kidney Events (MAKE) in patients with AKD.

Methods: This prospective, open-label, randomized controlled trial, conducted at a single center, enrolled patients with AKD and randomly assigned them to either an MDT intervention or standard care. The primary outcome was MAKE, a composite of renal progression to CKD, chronic dialysis, or death, assessed at 90 days. Secondary outcomes were evaluated at various milestones up to 3 years. Statistical analyses included Kaplan-Meier estimates and Cox proportional hazards models.

Results: The MDT intervention did not significantly decrease the overall likelihood of MAKE compared to standard care at 90 days (20% versus 18%) or 1 year (45% versus 50%; $p = 0.79$). However, in AKD stage 3 patients only, the MDT intervention improved MAKE outcomes at 90 days (28% versus 46%, $p=0.03$). All-cause mortality also showed no significant difference at 90 days (18% versus 15%) or 1 year (33% versus 30%; $p = 0.47$; Hazard Ratio 0.95, 95% CI 0.60-1.50). For CKD progression, although the overall difference was not statistically significant ($p = 0.56$), the cumulative probability at 1 year was 23% in the MDT group compared to 43% in the standard care group. Results related to Renal Replacement Therapy (RRT) were inconsistent. Two sensitivity analyses indicated a significantly higher or trending higher probability of RRT in the MDT group at 1 year (38% versus 20%, $p = 0.045$) among AKD stage 3 patients. The overall HR for RRT was 1.74 (95% CI 0.89-3.4). Conversely, one analysis demonstrated a trend toward lower RRT probability in the MDT group at 1 year (13% versus 22%, $p = 0.078$).

Conclusion: In this randomized trial, an MDT intervention did not significantly reduce the incidence of MAKE or mortality when compared to standard care. While a lower rate of CKD progression was observed at 1 year in the MDT group, this difference was not statistically significant overall. The conflicting results for RRT initiation across different analyses underscore the complexity of this outcome and necessitate further investigation to understand the varied impact of the MDT intervention.

Keywords : Acute Kidney Disease, Acute Kidney Injury, Chronic Kidney Disease, Multidisciplinary Team Care, Major Adverse Kidney Events, Renal Replacement Therapy, Randomized Clinical Trial.