Poster Presentation：Chronic Kidney Disease
Poster No.：B0120
Abstract Submission No.：APCN20250048

Three-Dimensional Kidney Structure Reveals Tubular Obstruction as the Initiating Event in ADPKD

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Abstract

Introduction:

Autosomal dominant polycystic kidney disease (ADPKD) is a progressive genetic kidney disorder and a leading cause of end-stage renal disease. Despite the identification of causative genes, the precise mechanisms underlying disease progression remain unclear, limiting the development of effective treatments. This study aims to elucidate the three-dimensional structural characteristics of kidneys in polycystic kidney disease (PKD) mouse models and explore their implications for disease pathogenesis.

Methods:

Histological evaluations were performed on kidneys from rapid-onset and chronic-onset PKD mouse models. In addition, the kidneys were subjected to tissue clearing techniques for three-dimensional imaging. Two-dimensional pathological sections were analyzed in parallel. Comparative Ki67 immunostaining was conducted between PKD models and wild type mice across various developmental stages to assess cell proliferation activity.

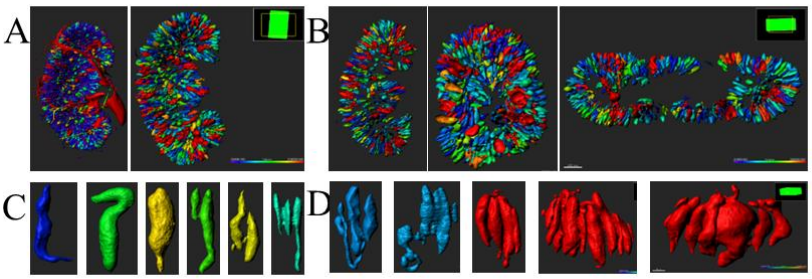
Results:

Three-dimensional imaging revealed that renal cysts formed a cord-like structure resembling a cluster of bananas rather than a bunch of grapes, suggesting that tubular obstruction may serve as the initiating event. Epithelial cell bridges within renal tubules were frequently observed in both three-dimensional and two-dimensional analyses and were identified as potential contributors to luminal obstruction. Ki67 staining demonstrated no significant increase in proliferative activity between PKD models and wild type mice, indicating that abnormal cell proliferation is unlikely to be a primary driver of cyst formation.

Conclusion:

These findings provide strong support for the obstruction hypothesis and challenge the traditional proliferation hypothesis in the pathogenesis of ADPKD. A better understanding of tubular obstruction mechanisms may pave the way for the development of novel therapeutic strategies for ADPKD.

Keywords：ADPKD, Three-Dimensional Imaging , Tubular Obstruction, Pathogenesis, Mouse Model



3D reconstruction of cysts from rapid onset PKD mouse model kidneys.
A: Panoramic 3D images.
B: 3D images in axial, coronal and sagittal viewpoints.
C: Individual tubular cysts.
D: Cluster tubular cysts.

Poster Presentation：Chronic Kidney Disease
Poster No.：B0121
Abstract Submission No.：APCN20250072

Antihypertensive and renoprotective effects of mineralocorticoid receptor antagonist on salt-loaded SDT fatty rat with diabetic nephropathy

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Abstract

Objective: The Spontaneously Diabetic Torii (SDT) fatty rat, an animal model for type 2 diabetes and obesity, develops diabetic nephropathy by salt loading. SDT fatty exhibited hyporeninemic hypertension and showed reduced response to angiotensin-converting enzyme inhibitors as first-line agents. Serum aldosterone, downstream of the renin-angiotensin system, remained in the normal range in SDT fatty. We focused on hypertension and nephropathy via mineralocorticoid receptor (MR) and aimed to study the effect of MR antagonist (MRA) in salt-loaded SDT fatty rats.

Methods: Ten-week-old male SDT fatty rats (n = 15) were divided into three groups: untreated (Cont), treated with 0.3% NaCl (Salt), and 0.3% NaCl and MRA, Kerendia (KER; 10 mg/kg/day). Body weight, water intake and diet intake were measured weekly. Systolic (SBP) and diastolic blood pressure (DBP), and blood glucose level (BGL) were measured 2 weeks post-treatment. Urine albumin-to-creatinine ratio (uACR), urine liver-type fatty acid binding protein-to-creatinine ratio (uL-FABPCR), and blood urine nitrogen (BUN) were assayed 3 weeks post-treatment. Kidney RNA was analyzed by quantitative RT-PCR for relative expression of Hepatitis A virus cellular receptor 1 (Havcr1), Lipocalin-2 (Lcn2), fibrinogen alpha chain (Fga), fibrinogen beta chain (Fgb), fibrinogen gamma chain (Fgg), TIMP metallopeptidase inhibitor 1 (Timp1) and alpha-2-macroglobulin (A2m) using Glyceraldehyde 3-phosphate dehydrogenase (Gapdh) as internal control.

Results: SBP, DBP, uACR, uL-FABPCR, and renal expression of Havcr1, Fgb, Fgg, Timp1 and A2m were significantly increased in Salt, indicating salt-induced hypertension and nephropathy. KER significantly suppressed increases in SBP, DBP, uACR, and renal expression of Havcr1, Fgb, Fgg, Timp1 and A2m. No significant suppression was observed in uL-FABPCR. No significant differences were found in body weight, diet intake, BGL, BUN, or expression of Lcn2 and Fga among groups, nor in water intake between Salt and Salt+KER.

Conclusions: MRA exerts antihypertensive and renoprotective effects in salt-loaded SDT fatty rats with hyporeninemic hypertension.

Keywords：SDT fatty rat, Diabetic nephropathy, Hyporeninemic hypertension, Mineralocorticoid receptor antagonist

Poster Presentation：Chronic Kidney Disease
Poster No.：B0122
Abstract Submission No.：APCN20250107

Mapping the Burden: Multimorbidity Profiles Among Chronic Kidney Disease Patients in Indonesia

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Abstract
Background: Globally, over 10% of the population suffers from chronic kidney disease (CKD), which is often accompanied by other chronic conditions. This coexistence of multiple diseases, known as multimorbidity, significantly reduces quality of life by causing functional impairment, psychological distress, and increased medical burden. Despite the increasing burden of CKD in Indonesia, the patterns and impact of multimorbidity remain underexplored.
Aims : This study aims to identify multimorbidity patterns among individuals with CKD in Indonesia.
Methods : A cross-sectional study was conducted using Indonesian National Health Insurance data from 2020 to 2022, including 234,333 individuals. Multiple Correspondence Analysis (MCA) was then employed to identify common multimorbidity patterns by determining the most frequently co-occurring conditions with CKD.
Results : Among Indonesian referred to hospitals, the prevalence of CKD in 2020 until 2022 was 1.11 (95% CI=0.95-1.28), with 42.82% having multimorbidities. Of these, 30.92% had one comorbidity, 10.22% had two comorbidities, and 1.42% had three comorbidities. Complex multimorbidity (i.e. ≥ 4 long-term conditions) was identified in 0.26% of the sample. MCA identified hypertension (n=1,937), type 2 diabetes mellitus/ T2DM (n=1,470), and ischemic heart disease (n=872) as the most frequently co-occurring condition with CKD (stage 1-4).In addition, a total of 575 patients with CKD were simultaneously diagnosed with both hypertension and T2DM. These findings suggest that hypertension may accelerate CKD progression through mechanisms such as glomerular hyperfiltration, increased intraglomerular pressure, and vascular remodeling.
Conclusions : Nearly half of Indonesians with CKD live with multimorbidity, with hypertension frequently co-occurring with CKD. Conducting routine screenings is essential to identify and prevent potential multimorbidity. Moreover, ensuring appropriate pharmacological management is imperative to minimize the side effects of polypharmacy.

Keywords：Chronic disease, Indonesia National Health Insurance, Chronic Kidney Disease, Multimorbidity

Poster Presentation：Chronic Kidney Disease

Poster No.：B0123

Abstract Submission No.：APCN20250119

Glycogen Synthase Kinase 3β Is Associated With Pericyte Senescence and Renal Fibrosis

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Abstract

Background and Aims

The transition of renal pericytes into myofibroblasts is a key driver of interstitial fibrosis during the progression of chronic kidney disease (CKD). While senescent pericytes are known to mediate fibrosis in other organs, their role in renal fibrosis remains unclear. Glycogen synthase kinase 3β (GSK3β), a critical regulator of fibroblast function, has been implicated in fibrogenesis, cellular senescence, and secretory activity; however, its contribution to renal aging and disease is poorly understood. Investigating the role of GSK3β in pericyte senescence may uncover novel therapeutic targets for kidney diseases.

Methods

To model pericyte behavior, NIH-3T3 fibroblasts were treated with etoposide to induce DNA damage–associated senescence. Senescence was confirmed by senescence-associated β-galactosidase (SA-β-gal) staining, expression of cyclin-dependent kinase inhibitors (CDKIs), and upregulation of senescence-associated secretory phenotype (SASP)-related genes. Profibrotic gene expression and protein production in senescent pericytes were analyzed. Proliferation and apoptosis of senescent pericytes were also evaluated. In vivo, PdgfrbCreERT2/+;Gsk3βF/F mice were used to specifically delete Gsk3β in pericytes in a unilateral ureteral obstruction (UUO) model. Renal fibrosis was subsequently assessed.

Results

Etoposide treatment effectively induced senescence in NIH-3T3 cells, as evidenced by increased expression of CDKIs and SASP-related cytokines, particularly interleukin-6 and monocyte chemoattractant protein-1. These findings indicate the establishment of a stable senescent phenotype with potential immunomodulatory capacity. In vivo, pericyte-specific deletion of Gsk3β significantly attenuated renal fibrosis in the UUO model, highlighting GSK3β as a key mediator of pericyte-driven fibrogenesis.

Conclusion

These findings suggest that pericyte GSK3β promotes renal fibrosis and that senescent pericytes may acquire immunomodulatory properties. Ongoing studies aim to determine whether GSK3β serves as a molecular link between fibrosis and senescence through shared regulatory mechanisms. Collectively, these insights may guide the development of pericyte-targeted GSK3β interventions for the treatment of renal fibrosis and aging-associated kidney diseases.

Keywords：Chronic kidney disease, renal fibrosis, pericyte, aging, GSK3β

Poster Presentation：Chronic Kidney Disease
Poster No.：B0124
Abstract Submission No.：APCN20250130

Neutrophil role in atherosclerosis in chronic kidney disease

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Abstract
Background and Aims
Chronic Kidney Disease (CKD) is associated with high mortality, primarily due to cardiovascular disease (CVD). In pre-dialysis patients, arteriosclerosis, particularly intimal calcification, is a common underlying cause of CVD. While elevated phosphate levels in CKD are recognized as a risk factor for calcification and cardiovascular events, the mechanisms underlying intimal calcification in CKD remain unclear. Recent studies have identified Fibroblast Growth Factor Receptor 1 (FGFR1) in osteocytes as a key sensor of elevated serum phosphate levels, regulating phosphate homeostasis. Among immune cells, neutrophils specifically express FGFR1, leading us to hypothesize that the response of neutrophils to elevated phosphate levels in CKD patients may contribute to the pathogenesis of intimal calcification.
Methods
Neutrophils were isolated from the peripheral blood of healthy controls (HC) and CKD patients using density gradient centrifugation with Polymorphprep. Neutrophils were then stimulated with varying phosphate concentrations. Neutrophil activation was assessed by measuring NETs formation using Sytox Green staining and immunostaining for Myeloperoxidase (MPO) and Citrullinated Histone H3 (CitH3). To investigate the involvement of the phosphate-FGFR1 signaling pathway, neutrophils treated with high phosphate concentrations were pre-treated with FGFR1 and Peptidylarginine deiminase 4 (PAD4) inhibitors. Further assessments of NETs signaling were conducted through inhibitory experiments and immunostaining.
Results
Neutrophils from both HC and CKD patients responded to high phosphate concentrations by forming Sytox- and CitH3-positive NETs. Notably, neutrophils from CKD patients exhibited a more pronounced induction of NETs, suggesting a hyperreactive response to elevated phosphate levels. Pretreatment with FGFR1 or PAD4 inhibitors effectively prevented the formation of CitH3-positive NETs in neutrophils exposed to high phosphate levels. Additionally, during this process, Fetuin-A, a regulator of calcium-phosphate crystal formation, was degraded by the neutrophil proteasome pathway.
Discussion and Conclusion
Neutrophils respond to elevated serum phosphate levels by inducing NETs formation with histone citrullination in a dose-dependent manner, with this response being more pronounced in CKD patients. This response was found to be ROS-PAD4 dependent and was attenuated by FGFR1 and PAD4 inhibitors. The observed vascular inflammation is hypothesized to contribute to the development of intimal calcification in CKD. These findings suggest that neutrophils may play a role in the pathogenesis of atherosclerosis in CKD through their hyperreactive response to elevated phosphate levels.

Keywords：atherosclerosis, calcification, neutrophil, ckd

Poster Presentation：Chronic Kidney Disease
Poster No.：B0125
Abstract Submission No.：APCN20250145

The Effects of T Cell-Specific Hypoxia-Inducible Factor-1 Activation in Murine Kidney Disease Models

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Abstract

Introduction

Activation of hypoxia-inducible factor-2 (HIF-2) with HIF-prolyl hydroxylase inhibitor (HIF-PHI) is approved for the treatment of anemia in patients with chronic kidney disease (CKD). However, pre-clinical studies reported that T cell-specific HIF-1 activation may augment kidney inflammation and injury in nephritis models. We aim to study the effects of T cell-specific HIF-1 activation in a preclinical murine CKD model.

Methods

Tg(Cd4-Cre);Vhl1F/F;Epas1F/F mice were used to achieve T cell-specific activation of HIF-1. The adenine nephropathy model was used to characterize kidney injury. Blood urea nitrogen and serum creatinine levels were used to estimate renal function. In the kidney, the severity of injury was measured by the mRNA level of Havcr1, encoding kidney injury molecule-1 (KIM-1). Kidney inflammation was estimated by the mRNA levels of Ptprc (encoding CD45) and Adgre1 (encoding F4/80). Kidney fibrosis was assessed by the mRNA levels of Col1a1 and Acta2 (encoding α-smooth muscle actin, α-SMA).

Results

Compared with littermate Vhl1F/F;Epas1F/F mice, Tg(Cd4-Cre);Vhl1F/F;Epas1F/F mice had no changes in kidney injury in the adenine nephropathy model. The levels of blood urea nitrogen and serum creatinine were statistically indistinguishable between Vhl1F/F;Epas1F/F and Tg(Cd4-Cre);Vhl1F/F;Epas1F/F mice. In addition, no changes in mRNA levels of Havcr1, Ptprc, Adgre1, Col1a1, or Acta2 could be observed.

Conclusion

T cell-specific HIF-1 activation does not have remarkable impacts on kidney injury in a preclinical CKD model.

Keywords：hypoxia-inducible factor

Poster Presentation：Chronic Kidney Disease
Poster No.：B0126
Abstract Submission No.：APCN20250148

Real-World Effects of Continued SGLT2 Inhibitor Use on Renal Function in Stage 3 CKD Patients

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Abstract
Introduction
Sodium-glucose co-transporter 2 inhibitors (SGLT2i) are known to provide renal protection in clinical trials. However, their real-world effectiveness in patients with stage 3 chronic kidney disease (CKD) remains unclear.
Methods
We conducted a retrospective cohort study using a regional database to analyze adult patients diagnosed with stage 3 CKD in 2023–2024. Patients were grouped by continued SGLT2i use versus non-use throughout 2023. Those with irregular SGLT2i status or insufficient serum creatinine data were excluded. Renal outcomes were measured using changes in estimated glomerular filtration rate (eGFR) and urinary albumin-to-creatinine ratio (UACR) across quarterly intervals. Statistical adjustments accounted for age, sex, diabetes, hypertension and renin angiotensin system inhibitor use.
Results
Among 626 eligible patients in 2023, 227 continued SGLT2i use, and 399 did not. In 2024, of the 302 who continued follow-up and had valid data, including 127 with continued SGLT2i use and 175 did not use. The baseline characteristics were similar, though diabetes prevalence was higher in the SGLT2i group. From Q1 2023 to Q2 2024, no significant difference in eGFR decline was observed ($p=0.589$), but the SGLT2i group showed a more stable trajectory. UACR declined significantly in SGLT2i users from Q1 2023 to Q1 2024 ($p<0.05$), while the non-user group showed no improvement.
Conclusion
In real-world settings, continued SGLT2i use in stage 3 CKD patients was associated with stabilized eGFR and reduced albuminuria. The treatment effect was less pronounced compared to other large-scale studies, but the findings support a protective renal role for SGLT2i in routine care.

Keywords：Sodium-glucose co-transporter 2 inhibitors (SGLT2i),chronic kidney disease (CKD)

Poster Presentation：Chronic Kidney Disease
Poster No.：B0127
Abstract Submission No.：APCN20250155

Association Of Frailty Severity To Anxiety, Depression, And Incidence Of Fall Among Chronic Kidney Disease Patients Undergoing Maintenance Hemodialysis At East Avenue Medical Center: A Cross-Sectional Study

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Abstract

Background:

Patients undergoing hemodialysis often experience multiple health challenges, including frailty, anxiety, depression, and falls. Frailty, a syndrome marked by reduced physiological reserves and heightened vulnerability to stress, is particularly prevalent in individuals with chronic kidney disease (CKD) and is known to predict adverse outcomes.

Objectives:

This study aimed to determine the prevalence of frailty and its complications, assess the presence of anxiety and depression, and identify the incidence of falls among maintenance hemodialysis patients receiving treatment at the East Avenue Medical Center Outpatient Hemodialysis Unit.

Methodology:

The study included 57 patients aged 19 to 80 years with chronic renal disease undergoing maintenance hemodialysis. Data were gathered through a self-administered questionnaire that captured demographic information and evaluated frailty using the Modified Fried Frailty Phenotype. Anthropometric measurements such as height, weight, and BMI were taken. Frailty was assessed based on five criteria: unintentional weight loss, fatigue, reduced walking speed, weak handgrip strength, and low physical activity. Walking speed and grip strength were evaluated, and laboratory data were reviewed. Falls were self-reported using the same questionnaire.

Results:

This study indicates a complex relationship between frailty markers, anxiety, and depression in CKD patients on hemodialysis. Hemodialysis access differed significantly by frailty, with arteriovenous fistulas more common in the pre-frail group (89.47%).

Physical Activity: A significantly higher proportion of frail patients reported change in physical activity (100%) compared to pre-frail patients (89.47%) ($p=0.042$).

Involuntary Weight Loss: More frail patients experienced involuntary weight loss (50%) compared to pre-frail patients (5.26%) ($p=0.001$).

Exhaustion and Walk Test: Both were significantly higher in frail patients (47.37%) than in pre-frail patients (0%) ($p<0.001$ for both).

Although most biochemical and frailty-related measures did not show significant differences across anxiety and depression groups, involuntary weight loss emerged as an essential factor with a higher prevalence of involuntary weight loss among patients with depression (70%) compared to those without (27.66%) ($p=0.011$).

Patients with involuntary weight loss are 6.1026 times more likely to have abnormal anxiety.

Albumin levels showed a slight decrease in anxious and depressed groups, with significant differences noted between anxiety groups ($p = 0.007$ for the non-anxious group).

Female patients are 7.75 times more likely to have abnormal anxiety compared to males.

BMI shows a protective effect against falls. Involuntary weight loss is strongly associated with an increased likelihood of falls.

Conclusion:
Frailty is a critical predictor of health outcomes in hemodialysis patients. Integrating frailty assessments into routine clinical care may help optimize management and improve the quality of life in this vulnerable population.

Keywords：Frailty; Chronic Kidney Disease (CKD); Hemodialysis; Anxiety; Depression; Falls; Modified Fried Frailty Phenotype; Clinical Outcomes; Psychological Health; CKD Complications

Poster Presentation：Chronic Kidney Disease
Poster No.：B0128
Abstract Submission No.：APCN20250161

Effect of Urea-Independent Osmotic Diuresis by An Inhibition of Urea Transporter in Mice

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Abstract

Introduction:

Chronic kidney disease (CKD) induces volume overload, which is a cause of congestive heart failure and mortality. Urea transporter (UT) -A1 absorbs urea in renal collecting duct and adjusts urine osmolality and volume. An inhibition of UT-A1 plays a role of osmotic diuresis depended on urine urea concentration. Here, we investigated an effect of an inhibition of UT for volume overload and a change of heart function in 5/6 nephrectomy mice.

Methods:

We prepared two models of CKD-mice and salt-loaded-mice. CKD mice underwent 5/6 nephrectomy and drank with 1% NaCl water for eight weeks. Salt-loaded mice fed with diet included 8% NaCl for two weeks. We used dimethylthiourea (DMTU) as an inhibitor of UT-A1. DMTU was administrated by intraperitoneal injection (100 mg/kg/day) after surgery. Daily urine was collected by a metabolic cage. For assessment of heart function, we measured mRNA expression associated with fibrosis in heart tissue.

Results:

In normal mice, 24-hr urine volume was 0.023 mL/g (vehicle) and 0.058 mL/g (DMTU). In the study of CKD mice, 24-hr urine volume was 0.15 mL/g (vehicle) and 0.17 mL/g (DMTU) in CKD mice ($p=0.004$). DMTU significantly increased urine volume, although kidney function declined. In CKD mice, the in-out valance (calculated by [drinking water volume] – [urine volume]) in DMTU group was significantly less compared with vehicle group (0.185 mL/g vs 0.114 mL/g, $P<0.05$). Heart weight was 4.2 mg/g (CKD-vehicle) and 3.4 mg/g (CKD-DMTU) ($P=0.018$). Thus, DMTU ameliorated heart hypertrophy in CKD mice. On assessment for heart fibrosis, mRNA gene expressions of collagen I and α-SMA in CKD-vehicle increase 1.47- and 2.94-fold compared with CKD-DMTU, respectively ($P<0.001$). In the study of salt-loaded mice, 24-hr urine volume was 0.25 mL/g (Na-vehicle) and 0.29 mL/g (Na-DMTU) ($p=0.59$). The volume of in-out valance was similar between vehicle- and DMTU-group. Heart weight was 4.2 mg/g (Na-vehicle) and 4.0 mg/g (Na-DMTU) ($P=0.34$). DMTU trend to reduce volume retention, but results were not significantly in salt-loaded model.

Conclusion:

An inhibition of UT ameliorated volume retention in CKD mice. In addition, UT inhibitor may contribute to prevent heart hypertrophy and heart fibrosis.

Keywords：urea, diuresis, volume retention

Poster Presentation：Chronic Kidney Disease
Poster No.：B0129
Abstract Submission No.：APCN20250181

Pharmacoinformatic Studies of Ternary Thiazole–Coumarin–Azomethine Derivatives as Dual TLR4/MD-2 Inhibitors and AMPK Activators: Toward Mechanism-Based Immunometabolic Therapy in Chronic Kidney Disease

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Introduction: Chronic kidney disease (CKD) progression is driven by the synergistic dysregulation of immune and metabolic pathways. Toll-like receptor 4 (TLR4) and its co-receptor MD-2 orchestrate renal inflammation through macrophage M1 polarization and the upregulation of pro-inflammatory mediators such as TNF-α, IL-6, and COX-2, leading to tissue injury and fibrosis. Simultaneously, suppression of AMP-activated protein kinase (AMPK), a central regulator of energy homeostasis, exacerbates oxidative stress, mitochondrial dysfunction, and vascular damage. Dual targeting of TLR4/MD-2 and AMPK may thus represent a promising therapeutic strategy to mitigate inflammation and metabolic dysfunction in CKD. This study aimed to computationally evaluate a series of novel ternary thiazole-coumarin-azomethine derivatives as potential dual modulators of TLR4/MD-2 and AMPK, with emphasis on pharmacokinetics, drug-likeness, toxicity, and solubility enhancement via β-cyclodextrin inclusion.

Methods: Molecular docking was performed using AutoDock Vina against TLR4/MD-2 (PDB ID: 3FXI) and AMPK (PDB ID: 5EZV) to evaluate binding affinity. pkCSM was employed for in silico ADMET predictions, including intestinal absorption, systemic clearance, CNS penetration, and toxicity risk. β-Cyclodextrin inclusion complex modeling was performed to predict improvements in aqueous solubility and bioavailability.

Results: Among the tested derivatives, compound 6j demonstrated the most favorable dual-target binding affinity (TLR4/MD-2: –9.1 kcal/mol; AMPK: –7.4 kcal/mol), followed by 6i (–8.6 kcal/mol for both) and 6h (–8.2 and –6.8 kcal/mol, respectively). All compounds were expected to be easily absorbed in the intestines (6i and 6j: 100%; 6h: 90.6%) and had low rates of being cleared from the body (6j: 0.066, 6i: 0.03 log mL/min/kg), which is good for how they work in the body. None were predicted to inhibit CYP1A2, minimizing the potential for metabolic interactions. Despite low inherent solubility (log S: –3.34 to –4.70), β-cyclodextrin complexation improved solubility through hydrogen bonding and stable host–guest interactions. Predicted blood-brain barrier permeability was low (log BB < –1.0), indicating minimal CNS exposure. All compounds met Lipinski's criteria (one violation each), with bioavailability scores of 0.55 and favorable synthetic accessibility (4.2–4.37). Toxicity profiling identified 6j as the safest candidate (LD_{50}: 3.376 mol/kg). These findings support a dual-target approach wherein TLR4/MD-2 inhibition suppresses inflammation and AMPK activation restores metabolic homeostasis, potentially offering a synergistic therapeutic strategy to halt CKD progression.

Conclusion: Compound 6j emerged as a promising dual TLR4/MD-2 and AMPK modulator with favorable ADMET, safety, and drug-likeness profiles. Its dual-action potential to regulate inflammation and metabolism supports further experimental validation for CKD therapy.

Keywords：Chronic kidney disease (CKD), TLR4/MD-2 inhibition, AMPK activation, Thiazole-coumarin-azomethine derivatives, β-Cyclodextrin inclusion complex

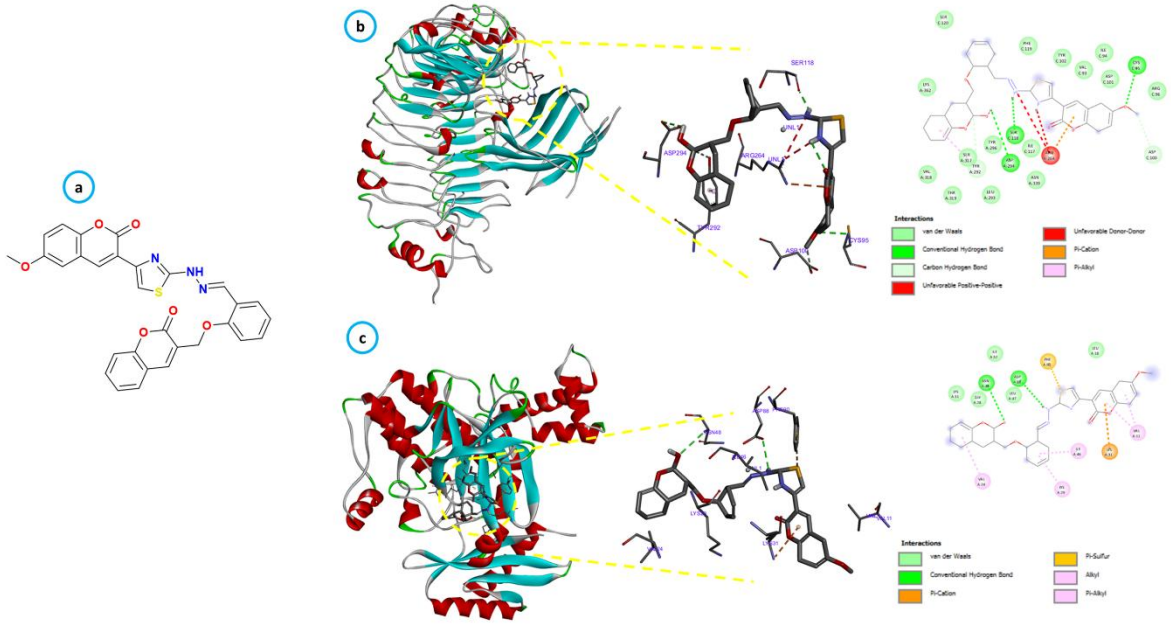

Figure 1. a) Molecule structure of 6j (ternary thiazole coumarin azomethine-based derivative), b) 3D and 2D binding interaction between 6j toward TLR4/MD-2, c) 3D and 2D binding interaction between 6j toward AMPK

Poster Presentation：Chronic Kidney Disease
Poster No.：B0132
Abstract Submission No.：APCN20250244

Integrated Bioinformatic Analysis on the Association between Air Pollution and Chronic Kidney Disease

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Abstract

Background

The World Health Organization (WHO) has stated that 99% of the global population is currently breathing air pollution higher than the normal limits, this is more prevalent in developing countries such as Indonesia and India. Chronic kidney disease (CKD) is a progressive condition that affects more than 10% of the global population and continues to increase each year. The exposure to air pollution can lead to the exacerbation or development of CKD. The association between these two diseases have not been studied further, hence this study aims to identify the distinct molecular mechanisms leading to the progression or development of CKD caused by air pollution.

Methods

Two microarray and RNA-sequencing datasetes (GSE126440 and GSE66494) were identified from the Gene Expression Omnibus (GEO) database with patients of CKD and ex vivo skin samples exposed to air pollution. Following that, the GEO2R analysis was done to analyze the differentially expressed genes (DEGs) contained in each dataset. The common DEGs from each dataset was identified and merged using the interactivenn tool. Then, the Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes was done with Enrichr and ShinyGo tool. Furthermore, the String program contained in Cytoscape was used to create a protein-protein interaction network.

Results

From these two datasets 27 common DEGs were identified, from which 18 genes were downregulated and 9 genes were upregulated. The results of the enrichment analysis of the upregulated common DEGs were linked to negative regulation of ferroptosis, vitamin K metabolism process, oxidoreductase acitvity, and cytochrom B5 reductase, ubiquinon biosynthesis, and NFE2L2 production. The enrichment analysis of the downregulated common DEGs were associated with the cytoskelekton, metal ion binding, apoptosis, G2 Phase, and signal transduction.

Conclusion

This study has shown the molecular mechanism of air pollution causing CKD. The pathways and the common DEGs identified can potentially be used as drug targets for CKD caused by air pollution.

Keywords：Bioinformatics, Chronic Kidney Disease, Air Pollution

Poster Presentation：Chronic Kidney Disease
Poster No.：B0133
Abstract Submission No.：APCN20250254

Rasgrp4-Aloxe3 Pathway Exacerbates Diabetic Kidney Fibrosis Via Scar-Associate macrophage Transformation

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Abstract

Diabetic kidney disease (DKD) is a leading cause of end-stage renal disease, with renal fibrosis being a key pathological feature. While inflammation and oxidative stress drive DKD progression, the role of macrophage phenotypic plasticity—particularly scar-associated macrophage (SAMac) transformation—remains incompletely understood. This study focuses on Ras guanine nucleotide-releasing protein 4 (RasGRP4) and its downstream mediator arachidonate lipoxygenase 3 (ALOXE3) in regulating SAMac-driven fibrosis.

RasGRP4: A Key Regulator in DKD Fibrosis

RasGRP4, a Ca^{2+}- and diacylglycerol-sensitive guanine nucleotide exchange factor, is predominantly expressed in immune cells. In DKD patients, RasGRP4 levels are elevated in peripheral blood mononuclear cells (PBMCs) and renal tissues(fig.1), positively correlating with $F4/80^{+}$ macrophage infiltration(fig.2). Genetic ablation of RasGRP4 in mice attenuates diabetic renal injury, reducing proteinuria, glomerular basement membrane thickening, and interstitial collagen deposition (Masson/PAS/HE-staining)(fig.3). Mechanistically, RasGRP4 deficiency suppresses renal expression of fibrosis markers (α-SMA, TGF-β1, collagen I)(fig.4-5) and SAMac markers (TREM2, Spp1)(fig.6), indicating its role in linking macrophage activation to fibrosis.

ALOXE3 as a Critical Downstream Mediator

Transcriptomic analysis of RasGRP4 wild-type(WT)/knockout(KO) diabetic mice identified ALOXE3(fig.7), a key enzyme in lipid peroxidation, as a downstream target. ALOXE3 expression is significantly reduced in RasGRP4 KO-mice renal tissues and correlates with RasGRP4 levels in human DKD biopsies(fig.8). In vitro, high glucose induces ALOXE3 expression in RAW264.7 macrophages, promoting lipid peroxidation (MDA accumulation)(fig.9) and suppressing the Xc^{-}/GSH/GPX4 antioxidant axis(fig.10). Knockdown of ALOXE3 mimics RasGRP4 deficiency, reducing SAMac phenotypes, and fibroblast-myofibroblast transition(fig.11), Conversely, ALOXE3 overexpression exacerbates oxidative stress and TGF-β/Smad3 signaling(fig.12-13), confirming its role in mediating RasGRP4-driven fibrosis.

The RasGRP4-ALOXE3 Axis in SAMac Transformation

High glucose triggers RasGRP4-dependent ALOXE3 upregulation, which inhibits cystine/glutamate antiport (Xc^{-}), depletes glutathione (GSH), and impairs GPX4 activity—key steps in oxidative stress and SAMac polarization. This process is accompanied by TGF-β1 secretion, activating Smad3 in fibroblasts to promote collagen synthesis. Blocking RasGRP4 or ALOXE3 disrupts this pathway, reducing both macrophage scarring and fibrotic responses(fig.14-15).

These findings establish the RasGRP4-ALOXE3 axis as a critical regulator of SAMac transformation and renal fibrosis in DKD. Targeting this pathway could disrupt the vicious cycle of oxidative stress and macrophage-fibroblast crosstalk. Future studies may explore RasGRP4 inhibitors or ALOXE3 antagonists as novel therapeutic strategies to mitigate DKD progression.

Conclusion

RasGRP4 promotes DKD fibrosis by inducing ALOXE3-mediated oxidative stress and SAMac

transformation. The interplay between RasGRP4, ALOXE3, and the Xc^-/GSH/GPX4 axis provides mechanistic insights into macrophage-driven fibrosis(fig.16), offering potential targets for antifibrotic therapy in DKD.

Keywords：Diabetic kidney disease; RasGRP4; Aloxe3; Scar-associated macrophages; Oxidative stress; Renal fibrosis

Poster Presentation：Chronic Kidney Disease
Poster No.：B0134
Abstract Submission No.：APCN20250275

Histone deacetylase 6 inhibition reverses renal fibrosis in a mouse model of unilateral ureteral obstruction.

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Abstract

Introduction

Histone acetylation plays a key role in kidney development and disease, prompting interest in histone deacetylase inhibitors (HDACi) as potential therapies. However, current pan-HDACi like SAHA and TSA lack selectivity and cause side effects, highlighting the need for targeted compounds. Focusing on HDAC6, we used a unilateral ureteral obstruction (UUO) mouse model to evaluate its role in chronic kidney disease (CKD) and the impact of its inhibition on renal fibrosis.

Methods

UUO mouse models were used to induce renal fibrosis. HDAC expression was analyzed by qPCR and Western blot. Proximal tubular epithelial cells (PTECs) were treated with TGF-β1 and HDAC6 inhibitor. Fibrotic kidney was used to analysis after in vivo treatment.

Results

We analyzed the gene expression of HDAC isoforms in the kidneys at first. Interestingly, the expression of all studied HDAC genes was increased in the fibrotic kidneys induced by UUO or unilateral ischemia-reperfusion injury (UIRI). UUO surgery induced pericyte-to-myofibroblast transition and progressive renal fibrosis in mice. As expected, α-SMA increased, and HDAC6 protein levels were markedly elevated over time in UUO kidneys. Immunoblotting showed reduced acetyl-H3 levels from day 0 to day 10, suggesting that HDAC6 plays a key role in early-stage fibrosis by repressing acetyl-H3, especially at days 7 and 10. The absence of acetyl-H3 reduction at day 14 implies HDAC6 acts mainly in early fibrosis. Its expression increased in tubular epithelial cells, but not in Col1a1-GFP+ myofibroblasts or pericytes. To verify HDAC6 expression in tubular epithelial cells, Translating Ribosome Affinity Purification (TRAP) was performed on mRNA from Slc34a1-expressing proximal tubular epithelial cell. The isolated mRNA confirmed increased Hdac6 expression following UUO surgery. We tested different concentrations HDAC6 inhibitor (HDAC6i) treatment in PTECs, despite not reducing HDAC6 protein levels, increased acetylation of H3 and α-tubulin. This suggests HDAC6i can restore tubular cell acetylation at certain concentrations. UUO mice received oral administration HDAC6i #60 twice daily for 10 days to conquer its short half-life. Mice treated with HDAC6i #60 showed reduced Acta2 and Pdgfrb expression in UUO kidney, with other fibrotic genes trending downward. α-SMA was also significantly reduced. Picrosirius red staining and quantification confirmed a marked reduction in fibrosis after HDAC6i #60 treatment.

Conclusion

HDAC6 inhibitor compound #60 effectively inhibited HDAC6 activity and reduced renal fibrosis in both in vitro and in vivo models.

Keywords：Histone Deacetylase 6, Renal Fibrosis, Unilateral Ureteral Obstruction

Poster Presentation：Chronic Kidney Disease
Poster No.：B0135
Abstract Submission No.：APCN20250293

Epigenetic Modulation of Acta2 Repressors Mitigates Kidney Fibrosis

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Abstract

Introduction

Kidney injury induces the transition of pericytes into myofibroblasts—the main collagen-producing cells in fibrosis—accompanied by increased α-SMA expression. This shift is associated with reduced expression of Acta2 repressors, including Rasal1, Pura, Tead1, Ybx1, and Ybx2, leading to elevated α-SMA levels. SUV39H2, a lysine methyltransferase, promotes H3K9me3, silencing gene transcription. We hypothesized that SUV39H2-mediated H3K9me3 suppresses Acta2 repressors, enhancing α-SMA expression. This study examines whether chaetocin, an H3K9me3 inhibitor, can reverse these epigenetic changes and reduce kidney fibrosis.

Methods

A kidney fibrosis model was established in C57BL/6 mice using unilateral ureteral obstruction (UUO), with gene expression analyzed at sham, and 7, 10, and 14 days post-surgery. Mice received either vehicle or chaetocin treatment. qPCR assessed fibrosis-related genes, histone methyltransferases, and Acta2 repressors. Western blotting examined key fibrotic proteins, and picrosirius red staining quantified fibrosis. In vitro, C3H10T1/2 cells were treated with TGF-β1 (5 ng/mL) alone or with chaetocin (10 nM), and gene expression was analyzed to investigate chaetocin's molecular effects.

Results

With prolonged UUO, expression of pro-fibrotic genes (Acta2, Col1a1, Col3a1) progressively increased. Suv39h2 was also significantly upregulated, while Suv39h1 remained unchanged. Conversely, Acta2 repressors like Ybx2 showed a gradual decline over time.

In vitro, TGF-β1 stimulation of C3H10T1/2 cells promoted a pro-fibrotic phenotype, marked by increased Acta2, Col1a1, Tgfb1, and Suv39h2 expression, while Suv39h1 remained unchanged. Chaetocin treatment reversed Acta2 and Col1a1 expression, indicating anti-fibrotic effects, without altering Suv39h2 levels—suggesting its action occurs at the functional rather than transcriptional level. Additionally, chaetocin restored Ybx2 expression suppressed by TGF-β1, counteracting its inhibitory effect.

To assess the anti-fibrotic effect of chaetocin via H3K9me3 inhibition in vivo, we used the UUO kidney fibrosis model. Chaetocin administered intraperitoneally significantly reduced fibrotic gene expression compared to vehicle-treated UUO mice.

Western blot analysis revealed increased pro-Col1a1 and α-SMA levels in UUO kidneys on day 10 compared to the contralateral side. Chaetocin treatment significantly reduced both markers, indicating anti-fibrotic activity. Additionally, chaetocin restored Ybx2 expression, which was downregulated in UUO kidneys.

Picrosirius red staining showed marked fibrosis in UUO kidneys at day 10, which was significantly reduced by chaetocin treatment. These results suggest that chaetocin alleviates fibrosis by

regulating pro-fibrotic genes and reversing related epigenetic alterations.
Conclusion
Chaetocin reduces kidney fibrosis by regulating pro-fibrotic genes and reversing fibrosis-related epigenetic changes. By restoring Acta2 repressors and blocking TGF-β1-driven pericyte-to-myofibroblast transition, it shows promise as a therapy for epigenetic fibrosis.

Keywords：unilateral ureteral obstruction, chaetocin

Poster Presentation：Chronic Kidney Disease
Poster No.：B0136
Abstract Submission No.：APCN20250298

Novel Perspectives on Leptospirosis Kidney Disease: Insights from Single-Nucleus RNA Sequencing and Spatial Transcriptomics

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Background: Leptospirosis, a neglected zoonotic disease caused by Leptospira infection, poses a major public health challenge. Clinical and basic research have shown that leptospiral infections are associated with both acute and chronic kidney disease. However, studies on the pathophysiology of leptospirosis kidney disease remain limited. To elucidate the underlying mechanisms of the transition from acute to chronic kidney disease due to Leptospira infection, we performed single-nucleus RNA sequencing and spatial transcriptomics on kidney tissues from infected mice.

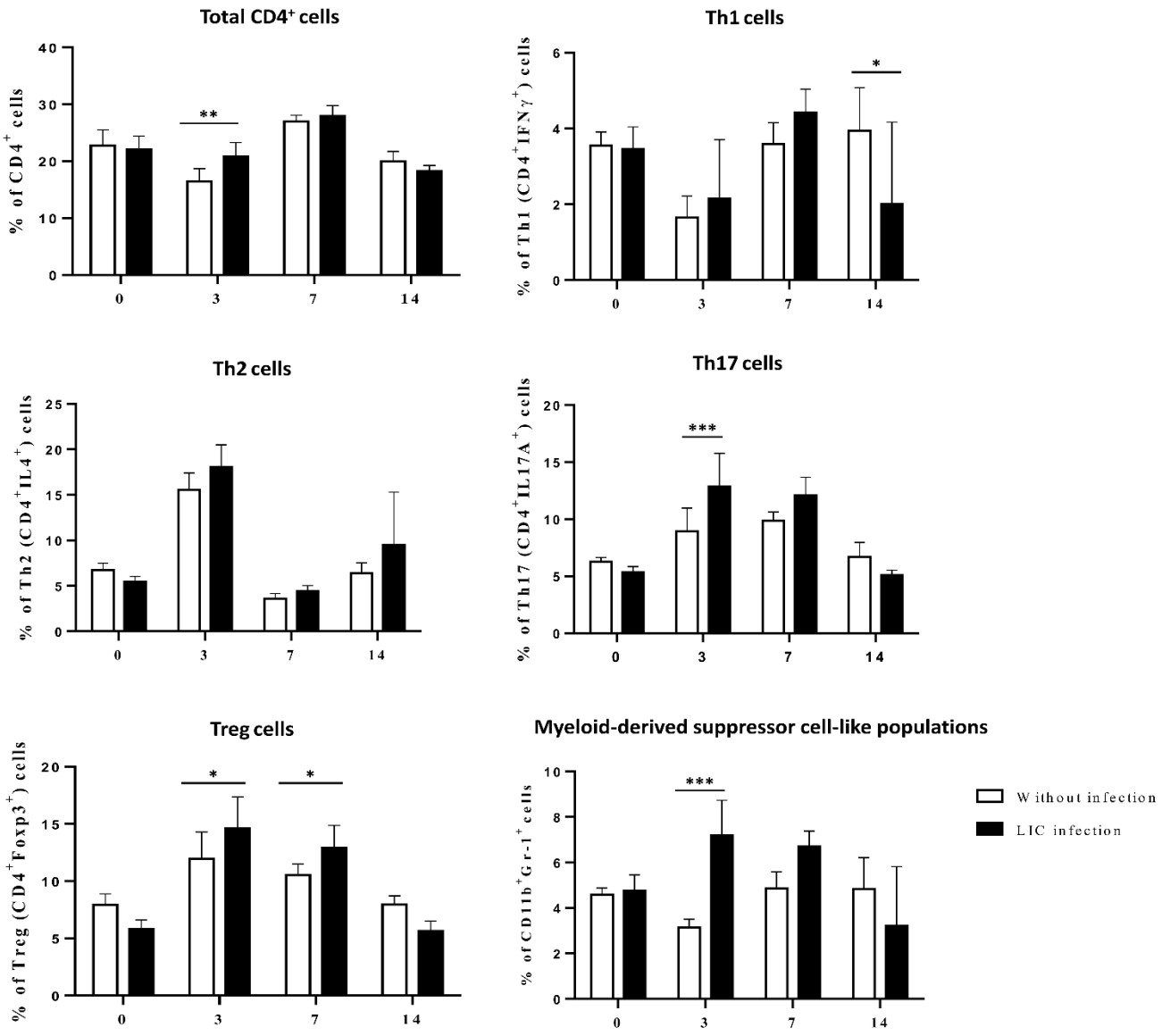
Methods

1. Mouse model for leptospiral kidney disease
2. High-dimensional flow cytometry for analyzed leukocyte subpopulations in peripheral blood at early infection stages
3. Single-nucleus RNA sequencing for profiling changes in kidney cell populations
4. Spatial transcriptomics for mapping of the spatial distribution and transcriptional profiling

Results: SnRNA-seq analysis revealed significant changes in kidney cell populations, with a notable impact on proximal tubule cells, which exhibited marked injury, followed by immune cells infiltration, indicating an active immune response. We further explored systemic immune response by applying high-dimensional flow cytometry to analyze leukocyte subpopulations in peripheral blood of infected mice at early and late stages of infection. The result showed significant changes in CD4+ T cell subsets, including increases in both Th17 and regulatory T cells during early infection. There was an observed expansion of CD11b+Gr-1+ myeloid-derived suppressor cell-like populations, suggesting the activation of immunosuppressive mechanisms that may facilitate persistent infection and chronic inflammation. Moreover, spatial transcriptomics of formalin-fixed, paraffin-embedded kidney sections from infected mice was performed using the 10X Genomics Visium CytAssist platform. This approach enabled comprehensive mapping of the spatial distribution and transcriptional profiling of injured proximal tubules and infiltrating immune cells. The analysis revealed a profibrotic and inflammatory microenvironment, characterized by interactions among injured tubules, fibroblasts, immune cells, and endothelial cells, which may contribute to the transition from acute kidney injury to chronic kidney disease.

Conclusion: Our study highlights the dynamic interplay between kidney injury, immune responses, and fibrosis in leptospirosis kidney diseases. The insights gained offer potential therapeutic targets to slow or prevent disease progression. Future research is needed to further clarify the cellular and molecular mechanisms of chronic kidney injury in leptospirosis and to develop effective strategies aimed at preventing from acute to chronic kidney disease following Leptospira infection.

Keywords：Leptospirosis kidney disease; acute kidney injury; chronic kidney disease; single-nucleus RNA sequencing; Spatial transcriptomics


Total CD4+ cells
% of CD4+ cells
Th1 cells
% of Th1 (CD4+IFNγ+) cells
Th2 cells
% of Th2 (CD4+IL4+) cells
Th17 cells
% of Th17 (CD4+IL17A+) cells
Treg cells
% of Treg (CD4+Foxp3+) cells
Myeloid-derived suppressor cell-like populations
% of CD11b+Gr-1+ cells
Without infection
LIC infection

Poster Presentation：Chronic Kidney Disease
Poster No.：B0137
Abstract Submission No.：APCN20250302

AhR Antagonism Restores H_2S Signaling and Renal Function in 5/6 Nephrectomy Rats

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Abstract

Indoxyl sulfate (IS), a major protein-bound uremic toxin, is known to impair the cytoprotective effects of hydrogen sulfide (H_2S) in renal tubular cells, but its in vivo impact on the renal H_2S-generating system remains unclear. Using a rat model of chronic kidney disease (CKD) induced by 5/6 nephrectomy (Nx), we observed that poor renal clearance was associated with elevated serum levels of IS and its precursor, homocysteine. Compared to controls, the expression of H_2S-producing enzymes—cystathionine β-synthase (CBS), cystathionine γ-lyase (CSE), and 3-mercaptopyruvate sulfurtransferase (3-MST)—was markedly reduced at both the mRNA and protein levels in Nx kidneys. This was accompanied by a decline in the transcription factor specificity protein 1 (Sp1), a key upstream regulator of these enzymes. To examine the role of IS signaling, we administered CH-223191, an antagonist of the aryl hydrocarbon receptor (AhR), the primary receptor for IS. CH-223191 treatment restored Sp1 expression and activity, increased the levels of CBS, CSE, and 3-MST, and enhanced H_2S concentrations in both renal tissue and plasma. Functionally, this intervention mitigated homocysteine accumulation, improved renal perfusion and excretory function, and attenuated histologic tubular injury. Additionally, markers of oxidative stress—including superoxide generation, malondialdehyde (MDA) accumulation, and reduced glutathione depletion—were all significantly improved by AhR blockade. Notably, CH-223191 had no effect on circulating IS levels, suggesting that blockade of downstream IS signaling rather than toxin removal is key to therapeutic benefit. Our findings highlight that IS disrupts Sp1-mediated H_2S biosynthesis in vivo and that targeting AhR may represent a novel strategy to protect against CKD progression.

Keywords：Hydrogen sulfide, indoxyl sulfate, aryl hydrocarbon receptor, specificity protein 1, chronic kidney disease, oxidative stress

Poster Presentation：Chronic Kidney Disease
Poster No.：B0138
Abstract Submission No.：APCN20250303

Redox Gene Dysregulation and CCL5 Suppression in Metabolic Syndrome-Induced Kidney Injury.

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Abstract

Metabolic syndrome (MetS) is a growing public health concern associated with increased risk of chronic kidney disease. It is characterized by obesity, insulin resistance, and dyslipidemia, all of which contribute to renal injury through oxidative stress (OxS). However, the molecular basis linking systemic OxS to kidney damage remains poorly understood. In this study, we developed a rat model of MetS using a high-fat diet combined with a low-dose streptozotocin injection. MetS rats exhibited features of systemic OxS, including elevated free oxygen radicals, diminished antioxidant capacity, and increased blood pressure. Renal evaluation revealed lipid peroxidation, glomerular hyperfiltration, and tubular damage. Transcriptomic analysis of renal tissue identified six significantly downregulated redox-related genes: C-C motif chemokine ligand 5 (CCL5), glutamate-cysteine ligase catalytic subunit (GCLC), glutathione peroxidase 6 (GPX6), recombination activating gene 2 (RAG2), NAD(P)H quinone oxidoreductase 1 (NQO1), and selenoprotein P1 (SEPP1). Among these, CCL5 was notably suppressed across multiple levels: its mRNA, protein expression, and serum levels were consistently reduced in MetS rats. Given CCL5's known functions in immune regulation and redox balance, its suppression may exacerbate oxidative damage in the kidney. Immunohistochemical staining confirmed reduced CCL5 in key renal subregions, including the cortex, medulla, and papilla. These findings suggest that disruption of redox gene expression, particularly CCL5 repression, plays a key role in the pathogenesis of MetS-associated kidney injury. CCL5 may serve as both a mechanistic link and a potential biomarker for oxidative renal damage in metabolic syndrome, offering future therapeutic and diagnostic value.

Keywords：Metabolic syndrome; oxidative stress; kidney injury; CCL5; redox gene expression

Poster Presentation：Chronic Kidney Disease
Poster No.：B0139
Abstract Submission No.：APCN20250365

Catalpol improves high-salt induced renal injury by reducing the production of intestinal metabolite TMAO.

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Abstract
Objective
The study aims to explore high-salt diet induced aging kidney injury whether by influencing the metabolites of the intestinal flora, and treatment with catalpol could improve aging kidney damage induced by high-salt diet.
Method
This study included 24 male C57BL/6J mice which with the age of 24-month-old, they were randomly divided into 3 groups: normal salt diet (NSD, 0.3% NaCl) group, high salt diet (HSD, 8% NaCl) group, and HSD + Catalpol treatment group. Catalpol dissolved in phosphate-buffered saline (PBS); administered intragastrically once a week (10 mg/kg) at weeks 7-10 and weeks 17-20 respectively. Collect the urine, serum, kidney tissue and feces of mice. Use 16S rRNA sequencing, ELISA, LC-MS/MS quantification and other experimental methods to analyze the corresponding indicators.
Results
After 10-week high-salt diet, we observed significant kidney damage, including the assessment of glomerular injury index and tubulointerstitial injury index through PAS staining. The treatment group with catalpol showed a significant reduction in the corresponding damage. By detecting the 16SrRNA sequencing of feces in each group, the α-diversity analysis describing species richness showed that the Chao1 index in the HSD group was lower than that in the NSD group, but not significantly (P=0.206). After catalpol treatment, the Chao1 index increased, indicating that HSD treatment could reduce the abundance of intestinal flora species in mice. Catalpol treatment can alleviate the reduction of intestinal flora abundance. Shannon index analysis revealed that HSD significantly reduced the diversity of the intestinal microbiota in mice (P=0.003), and catalpol treatment could significantly increase the diversity of the intestinal microbiota in mice. In order to further explore the bacterial genera with significant differences in the intestinal microbiota among different groups, we analyzed the differences of the top 10 bacterial genera in terms of microbiota abundance. The results showed that the abundance of Lachnospiraceae NK4A136 in the HSD group was significantly decreased, and the abundance was significantly increased after catalpol treatment. Metabolites were detected by the targeted LC/MS method. It was found that the expression of TMAO in the blood of the high-salt diet group increased, and the expression of TMAO could be reduced by catalpol treatment.
Conclusion
High-salt diet in aged mice leads to metabolic abnormalities in the intestinal microbiota, which in turn causes kidney injury. The treatment with catalpol can improve these metabolic abnormalities and thereby alleviate kidney injury. This study provides new therapeutic targets and methods for improving high-salt-induced kidney injury.

Keywords：High salt diet, catalpol, kidney injury,intestinal flora

Poster Presentation：Chronic Kidney Disease
Poster No.：B0140
Abstract Submission No.：APCN20250398

Association Between Genetic Polymorphisms and Chronic Kidney Disease: A Systematic Review

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Abstract
Background:
Chronic Kidney Disease (CKD) is a multifactorial condition influenced by both environmental and genetic factors. Numerous studies have investigated the role of genetic polymorphisms in CKD susceptibility and progression, but results have been inconsistent. This systematic review aims to synthesize existing evidence on the association between specific genetic polymorphisms and CKD risk.
Methods:
A comprehensive literature search was conducted across PubMed, EMBASE, Scopus, Web of Science, and Cochrane Library databases from 2010 to April 2025. Studies were included if they assessed the association between genetic polymorphisms and CKD in human populations and provided sufficient data for calculating ORs and 95% CIs. Data extraction and quality assessment were performed independently by two reviewers.
Results:
A total of 8 studies met the inclusion criteria, encompassing 1326 participants. Significant associations were identified between CKD and polymorphisms in genes such as APOL1, UMOD, ACE, eNOS, SLC22A2, IL-6, TNF-α, and MTHFR. For instance, the APOL1 G1 and G2 variants were strongly associated with increased CKD risk among individuals of African descent. The UMOD rs12917707 polymorphism was linked to salt-sensitive hypertension and CKD susceptibility . Subgroup analyses revealed variations in associations based on ethnicity and CKD etiology.
Conclusions:
The review highlights the significant role of specific genetic polymorphisms in CKD susceptibility and progression. Understanding these genetic associations can enhance risk stratification and inform personalized interventions in CKD management. Further large-scale, multi-ethnic studies are warranted to validate these findings and explore underlying mechanisms.

Keywords： Genetic Polymorphism, Chronic Kidney Disease, Systematic Review , CKD, Genetics

Poster Presentation：Chronic Kidney Disease
Poster No.：B0141
Abstract Submission No.：APCN20250443

Quality of Life Among Family Members of Chronic Kidney Disease Patients in Odisha, India: A Cross-Sectional Study

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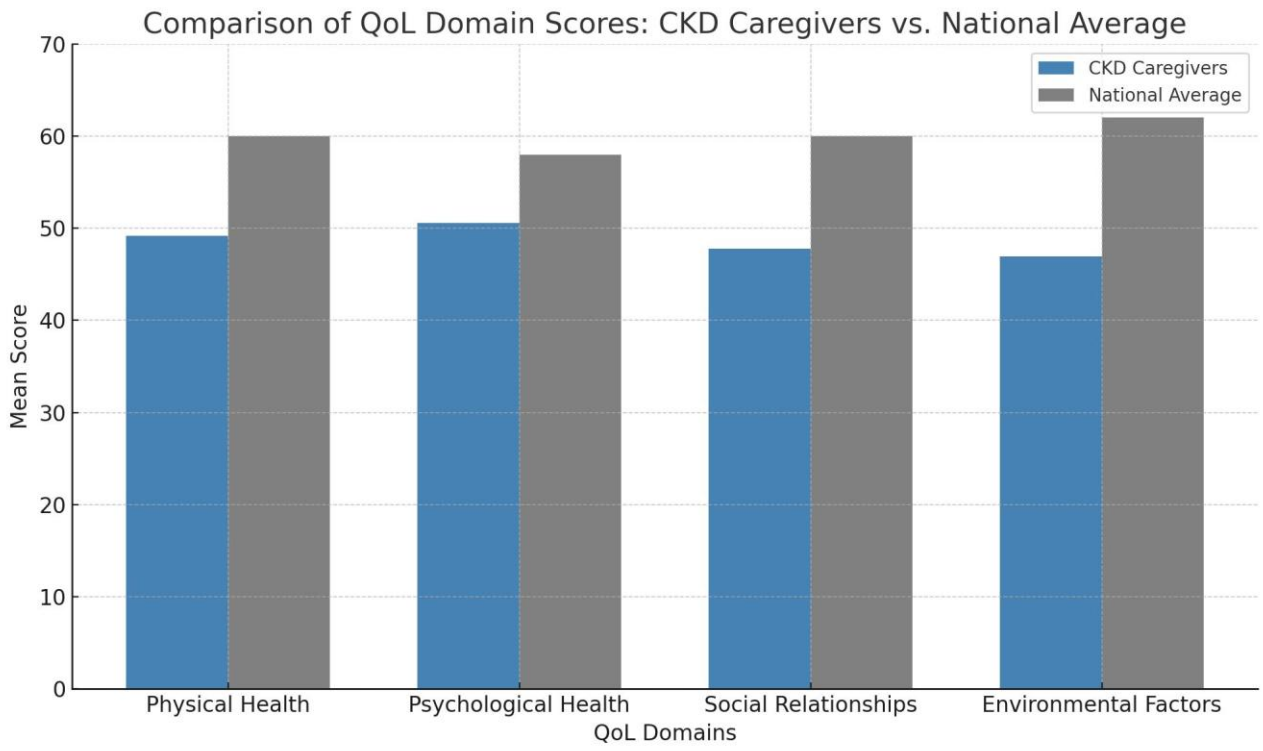
Abstract
Introduction:
Chronic Kidney Disease (CKD) imposes substantial physical, emotional, and economic strain not only on patients but also on their families, especially in resource-constrained regions such as Odisha, India. Family members often serve as primary caregivers, taking on responsibilities that disrupt their personal lives, affect their physical and mental well-being, and introduce significant financial burden. Despite the rising CKD prevalence in India, little is known about the quality of life (QoL) of caregivers, particularly in rural eastern regions. This study aimed to assess QoL among family caregivers of CKD patients in Odisha and to identify key socio-demographic and clinical determinants affecting their well-being.
Methods:
A cross-sectional survey was conducted among 285 primary caregivers of CKD patients receiving dialysis or conservative treatment at public hospitals in Odisha. The World Health Organization Quality of Life – BREF (WHOQOL-BREF) tool, validated in the Odia language, was used to assess four domains: physical health, psychological well-being, social relationships, and environmental conditions. Socio-demographic data and clinical characteristics of patients were also collected. Data were analyzed using descriptive statistics and multivariate regression to examine factors significantly associated with QoL scores.
Results:
Caregivers had a mean age of 42.7 ± 10.3 years; 61% were female and 70% lived in rural areas. Mean QoL scores were below national norms across all domains: physical health (49.2), psychological health (50.6), social relationships (47.8), and environmental factors (46.9). Compared with national averages, the most notable gaps were observed in the social and environmental domains, reflecting social isolation and financial stress. Figure 1 illustrates the comparative scores across domains. Regression analysis revealed that rural residence, female gender, lower income, and caregiving for dialysis-dependent patients were significantly associated with lower QoL. Additionally, longer caregiving duration correlated with physical and emotional fatigue, further decreasing QoL scores.
Conclusions:
Family caregivers of CKD patients in Odisha experience significantly reduced quality of life, primarily due to caregiving burden, financial challenges, and limited access to health and social support services. Interventions such as caregiver support programs, travel and medical subsidies, and enhanced rural nephrology services are urgently needed to improve outcomes for both caregivers and patients.

Keywords：Chronic Kidney Disease; caregiver quality of life; family burden; Odisha; WHOQOL-BREF.


Comparison of QoL Domain Scores: CKD Caregivers vs. National Average
CKD Caregivers
National Average
Mean Score
0
10
20
30
40
50
60
70
Physical Health
Psychological Health
Social Relationships
Environmental Factors
QoL Domains

Poster Presentation：Chronic Kidney Disease
Poster No.：B0142
Abstract Submission No.：APCN20250459

Molecular Signature Profiling of Targeted Therapeutics to Prevent Cachexia via Kidney-Muscle Axis Modulation in Chronic Kidney Disease

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Abstract
Introduction:
Two out of every three adults with chronic kidney disease (CKD) show symptoms of cachexia. Muscle wasting in CKD is a debilitating complication and significantly contributing to poor clinical outcomes. Chronic inflammation, metabolic acidosis, and dysregulated protein metabolism promote a catabolic state, shifting toward muscle protein degradation. Elevated of pro-cachectic factors, activin A, stimulating proteolytic pathways, often compound muscle loss. However, investigation between kidney and muscle function poorly understood. This study aimed to investigate the molecular drivers of muscle wasting in CKD.
Method:
Gene chip data of GSE169316 was retrieved from the GEO expression database and analysed to identify differentially expressed genes (DEGs) in CKD with cachexia animal models. Protein-protein interaction (PPI) networks of these DEGs were constructed using STRING database. The key hub genes were identified based on their central positions within the network.
Result:
Following comprehensive data analysis of of 35.088 transcripts, Vsig4, Pik3c3, Mmrn1 emerged as significant dysregulation, potentially serving as biomarkers and therapeutic targets. These upregulation and downregulation of gene expressions are attributed to sustained overproduction of pro-cachectic factors and impaired renal clearance in CKD patients. It contributes to the progression of skeletal muscle degradation. Systemic administration of soluble activin receptor type IIB (sActRIIB) was employed to inhibit activin A signaling. This intervention successfully prevented muscle atrophy, restored muscle strength, and normalized mitochondrial function. Furthermore, these protective effects were further supported by the restoration of protein synthesis and modulation of genes involved in proteasome-mediated degradation and autophagy pathways. These results support activin A blockade as a viable therapeutic strategy for treating muscle wasting in CKD patients
Conclusion:
This study suggests that Vsig4, Pik3c3, Mmrn1 as important biomarker that improves targeting accuracy and reduced risk of recurrence. The modulation of activin signaling as a potential therapeutic strategy for skeletal muscle wasting in CKD

Keywords：Activin A, Cachexia, Chronic Kidney Disease, Inhibition, sActRIIB

Poster Presentation：Chronic Kidney Disease
Poster No.：B0143
Abstract Submission No.：APCN20250483

Mechanisms and therapeutic strategies of stone-associated chronic kidney disease mediated by Trpml1-Legumain pathway

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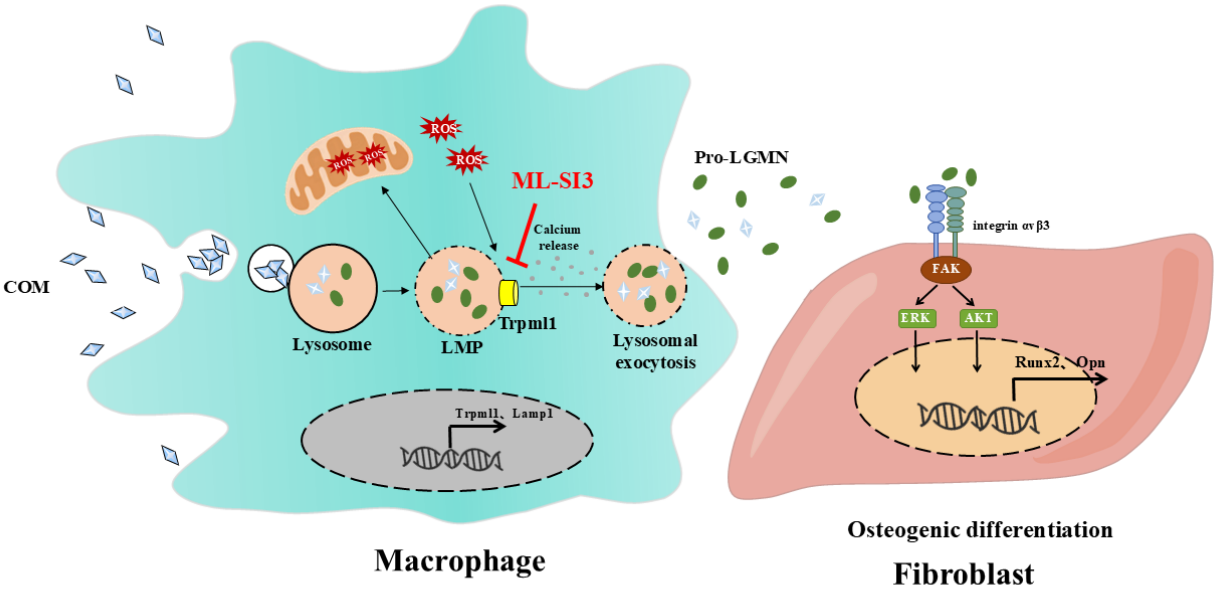
Abstract
Background:
Calcium oxalate (CaOx) nephrolithiasis is a major contributor to kidney injury and may progress to chronic kidney disease (CKD) through persistent inflammation and tubulointerstitial fibrosis. However, the underlying molecular mechanisms remain incompletely understood. This study investigates the role of macrophage-derived Legumain (Lgmn) in promoting CaOx crystal-induced CKD and explores how Lgmn triggers fibroblast osteogenic transdifferentiation.
Methods:
Lgmn-knockout ($Lgmn^{-/-}$) and wild-type (WT) male C57BL/6J mice were used to establish a CaOx nephrolithiasis model by glyoxylate injection and low-calcium, high-oxalate diet. Kidney injury was assessed by serum creatinine, BUN, polarized light microscopy, Von Kossa, and PAS staining. The expression of Lgmn, injury markers (Kim1, Lcn2), and osteogenic factors (Runx2, Opn) was examined by qPCR, Western blot, and immunohistochemistry. Single-cell transcriptomic analysis and immunofluorescence identified macrophages as the main Lgmn source. In vitro, RAW264.7 macrophages were stimulated with CaOx crystals, and Lgmn secretion was evaluated in relation to lysosomal acidification, autophagy flux, and mitochondrial ROS. Trpml1 channel involvement in lysosomal exocytosis was assessed via Fluo-4 calcium imaging, transmission electron microscopy, and Lamp1 staining. Conditioned media from CaOx-treated macrophages were applied to primary renal fibroblasts and 3T3 cells to evaluate osteogenic differentiation via the Fak/Erk/Runx2 pathway. Inhibition of Trpml1 with a kidney-targeted liposomal formulation of ML-SI3 (Lipo@ML-SI3) was tested in vivo.
Results:
Lgmn deficiency significantly reduced crystal deposition, tubular damage, inflammation, and renal dysfunction. Macrophage-derived Lgmn was markedly elevated in the CaOx model. CaOx crystal internalization disrupted lysosomal homeostasis, induced mitochondrial ROS, and activated Trpml1-mediated exocytosis of Lgmn. Secreted Lgmn bound to integrin αvβ3 on fibroblasts, activating Fak/Erk/Runx2 signaling and promoting osteogenic transdifferentiation. This process contributed to interstitial calcification and chronic structural remodeling. Trpml1 inhibition with Lipo@ML-SI3 significantly suppressed Lgmn secretion, fibroblast transdifferentiation, and CKD progression.
Conclusion:
Macrophage-derived Lgmn promotes CaOx crystal-induced chronic kidney injury by inducing fibroblast osteogenic transdifferentiation and interstitial calcification. Targeting the Trpml1–Lgmn axis may represent a novel therapeutic strategy to prevent nephrolithiasis-associated CKD.

Keywords：chronic kidney disease (CKD), nephrolithiasis, Legumain, TRPML1, osteogenic transdifferentiatio


COM
ROS
ROS
ROS
ROS
ML-SI3
Calcium release
Lysosome
LMP
Trpml1
Lysosomal exocytosis
Trpml1、Lamp1
Pro-LGMN
integrin αvβ3
FAK
ERK
AKT
Runx2、Opn
Osteogenic differentiation
Macrophage
Fibroblast

Poster Presentation：Chronic Kidney Disease
Poster No.：B0144
Abstract Submission No.：APCN20250511

Can sodium-glucose cotransporter 2 (SGLT2) inhibitor be a solution to chronic kidney disease (CKD)-related cognitive disorder?

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Abstract

Introduction: Chronic kidney disease (CKD) is characterized by progressive loss of renal function and complicated with varies comorbidities, including cardiovascular diseases (CVD) and neurological dysfunction. Our previous study has suggested that uremic toxins (including indoxyl sulfate and p-cresyl sulfate) could pass blood brain barrier (BBB) promoting neuroinflammation that eventually contributes to CKD-induced cognitive dysfunction. Empagliflozin (EMPA), a sodium-glucose co-transporter 2 (SGLT2) inhibitor, has emerged as a novel therapeutic approach for CVD and neurodegenerative disorders. This study aims to investigate pathogenic mechanisms of CKD-induced cognitive impairment, furthermore, to determine whether EMPA improves CKD-induced neurological dysfunction.

Methods: Eight-week-old male C57B6 wide type mice received sham and 5/6 nephrectomy to mimic CKD status. EMPA was given orally for 12 weeks. The Morris water maze (MWM) test was applied to evaluate cognitive function, including the short-term and long-term memory in shame, CKD, and EMPA-treated CKD mice. Fe3O4 Brain MRI and Evans blue were used to evaluate the permeability of BBB in mouse brain. Trans-well assay was used to evaluate the permeability of in vitro BBB model, constructed by bEnd3 cell, an endothelial cell from mouse brain. Western blot was used to identify the protein expression of target molecules in the brain tissue and endothelial cells.

Results: The results of in vitro study revealed that 20 μM indoxyl sulfate (IS), but not p- cresyl sulfate (PCS), dysregulated permeability of brain endothelial cells (bEnd3) via disruption of tight-junction integrity. The brains of 5/6-nephrectomy-induced CKD mice also showed BBB leakage by Fe3O4 tail vein-injected magnetic resonant images (MRI). In addition, Morris water maze tests indicated the impaired learning capability and long-term memory in CKD mice. The treatment of EMPA restored permeability of bEnd3 cells as well as ameliorated CKD-induced cognitive impairment that were consequences of EMPA maintaining BBB integrity of CKD mice. The evidence was demonstrated by Evans blue brain perfusion, brain MRI, MWM, and transwell permeability assay.

Conclusion: EMPA attenuated CKD-induced memory impairment in CKD mice, which may be explained by the improvement of uremic toxin-related BBB dysregulation.

Keywords：CKD, cognitive disorder, BBB, SGLT2 inhibitors

Poster Presentation：Chronic Kidney Disease
Poster No.：B0145
Abstract Submission No.：APCN20250545

Lower serum selenoprotein P level is a risk factor for peripheral artery disease in patients with stage 3 to 5 chronic kidney disease: A pilot study

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Abstract

Background: Selenoprotein P (SePP), which functions as a selenium carrier to tissues, regulates cellular reactive oxygen species. Low selenium levels have been associated with an increased risk of cardiovascular disease. Peripheral arterial disease (PAD) is associated with all-cause mortality and cardiovascular events, and a key pathological process is atherosclerosis. The present study aimed to determine the relationship between serum SePP level and PAD in patients with chronic kidney disease (CKD) stage 3 to 5.

Methods: Fasting blood samples and baseline characteristics were obtained from 130 CKD stage 3 to 5 patients. Ankle-brachial index (ABI) values were measured using an automated oscillometric device. Patients with ABIs of <0.9 were categorized into the low ABI group. Serum SePP levels were measured using a commercial enzyme-linked immunosorbent assay.

Results: In the study cohort, 20 of the 130 patients (15.4%) had low ABIs. The rates of diabetes mellitus (DM, p = 0.046), older age (p < 0.001), as well as the spot urine protein-to-creatinine ratio
(UPCR, p = 0.022), serum level of C-reactive protein (p < 0.001), were higher, while serum SePP (p
< 0.001) levels were lower in the low ABI group compared with the normal ABI group. The multivariable logistic regression analysis revealed that serum levels of SePP (odds ratio [OR]: 0.580, 95% confidence interval [CI]: 0.340–0.989, p = 0.045) were independently associated with PAD in patients with CKD stage 3 to 5. Left and right log-transformed ABI (log-ABI) values were also positively correlated with log-SePP (r = 0.623 and r = 0.588, p < 0.001, respectively).

Conclusions: In this study, lower serum SePP levels were associated with PAD in patients with CKD stage 3 to 5.

Keywords：Selenoprotein P, Chronic kidney disease, Peripheral arterial disease, Ankle-brachial index.

Poster Presentation：Chronic Kidney Disease
Poster No.：B0146
Abstract Submission No.：APCN20250594

GLP-1 Receptor Agonists Reduce Dementia and Alzheimer Disease Risk in Diabetic Patients with Advanced Chronic Kidney Disease: Real-World Evidence from TriNetX

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Abstract

Background: Patients with chronic kidney disease (CKD) and type 2 diabetes mellitus (T2DM) are at an elevated risk of dementia and Alzheimer's disease owing to vascular dysfunction, insulin resistance, and chronic inflammation. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) have demonstrated neuroprotective properties; however, their impact on dementia risk in diabetic patients with CKD remains unclear. This study evaluated the association between GLP-1RAs use and dementia risk in patients with CKD stage 3b or later, compared to dipeptidyl peptidase-4 inhibitors (DPP4is).

Methods: This retrospective cohort study analyzed data from the TriNetX global research network including electronic medical records of 67 healthcare organizations in the US Collaborative Network. We identified patients with CKD stage 3 or later and T2DM who were newly prescribed GLP-1RAs or DPP4is between January 1, 2015, and December 31, 2020. Patients with prior GLP-1RAs or DPP4is use, dementia diagnosed within 12 months before the index date, or recent hospitalization were excluded. The primary outcome was the incidence of dementia, Alzheimer's disease, vascular dementia, frontotemporal dementia, Parkinson's disease, extrapyramidal and movement disorders, and dementia with Lewy bodies, assessed over a follow-up period of 90 days to 5 years. Statistical analyses included Kaplan–Meier survival curves and Cox proportional hazards models.

Results: GLP-1RAs use was associated with a significantly lower risk of dementia (HR: 0.80, 95% CI: 0.71–0.91, p = 0.001) and Alzheimer's disease (HR: 0.76, 95% CI: 0.59–0.98, p = 0.033) compared to DPP4is use. However, no significant differences were observed in vascular dementia, frontotemporal dementia, Parkinson's disease, extrapyramidal and movement disorders, or dementia with Lewy bodies.

Conclusions: GLP-1RAs therapy may reduce dementia and Alzheimer's disease risk in patients with CKD stage 3b or later, offering potential neuroprotective benefits beyond glucose control. Research is needed to confirm these findings and optimize treatment strategies for this vulnerable population.

Keywords：GLP-1 receptor agonists, dementia, Alzheimer's disease, chronic kidney disease, type 2 diabetes.

Poster Presentation：Chronic Kidney Disease
Poster No.：B0147
Abstract Submission No.：APCN20250598

Omentum Patch Transplantation Restores GPX3 Expression and Mitigates Kidney Fibrosis in Ischemic Injury

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Abstract

Background: Ischemia-reperfusion injury (IRI) is a leading cause of acute kidney injury (AKI), characterized by oxidative stress, ferroptosis, and fibrosis. Reactive oxygen species (ROS)-induced ferroptosis contributes to kidney damage, with GPX3, a ferroptosis inhibitor gene, showing reduced expression as injury progresses. Mesenchymal stem cells (MSCs) have demonstrated regenerative potential by modulating inflammation, oxidative stress, and tissue repair. The omentum, a rich source of MSCs, could infiltrate in injured tissue, yet its therapeutic role in IRI-induced kidney injury remains unclear.

Methods: To explore the regenerative potential of omentum-derived MSCs, we harvested the omentum and engineered it into a 3D-printed patch, implanting it beneath the kidney capsule in a mouse IRI model. One week post-transplantation, kidney tissues were analyzed using single-cell and spatial RNA sequencing to assess GPX3 expression and ferroptosis-related gene regulation. ROS levels and fibrosis markers were also evaluated. Additionally, primary cultures of omental tissue were established and co-cultured with HK-2 cells to investigate the paracrine effects of omentum on oxidative stress and fibrosis-related responses in vitro.

Results: IRI-induced oxidative stress triggered ferroptosis, fibrosis, and kidney dysfunction, with a decline in GPX3 expression. However, omentum patch transplantation restored GPX3 expression, downregulated ferroptosis-related genes, and reduced ROS levels, leading to attenuated fibrosis and improved kidney function. The omentum-derived MSCs contributed to repair by reducing oxidative stress, inhibiting mitochondria dysfunction, and ferroptosis.

Conclusion: GPX3 expression was increased in omentum patched kidney based on scRNA-seq and spatial profiling, suggesting a potential protective role against ferroptosis. In vitro, GPX3 was downregulated by Erastin-induced ferroptosis, and further experiment using omentum-drived conditioned media are ongoing to assess its modulatory role. Primary culture of omentum-derived cells was successfully established for conditioned media preparation. Omentum patch transplantation delivers MSCs to injured kidney tissue, suppressing ferroptosis and oxidative stress, restoring GPX3, and alleviating kidney injury.

Keywords：Ferroptosis / Acute Kidney Injury / Mesenchymal stem cells / ROS stress / Kidney fibrosis

Poster Presentation：Chronic Kidney Disease
Poster No.：B0148
Abstract Submission No.：APCN20250607

MIT-001 mitigate renal fibrosis in mice.

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Abstract
Background : Ischemia-reperfusion injury (IRI) is a major pathological trigger in the development of renal fibrosis, a defining feature of chronic kidney disease (CKD). During CKD progression, IRI exacerbates inflammation, oxidative stress, and ferroptosis, ultimately leading to irreversible structural damage and fibrosis in renal tissue. MIT-001, a small molecule with established anti-necrotic and anti-inflammatory properties in various organ injury models, was investigated in this study for its potential to counteract IRI-induced renal fibrosis. This research aimed to explore whether MIT-001 can modulate key pathological pathways—including inflammation, oxidative stress, and ferroptosis—using both in vivo and in vitro models.
Methods : Male C57BL/6 mice (8 weeks old) were assigned to experimental groups based on IRI induction and MIT-001 administration. Renal IRI was induced via 24 hours of bilateral ischemia followed by reperfusion. MIT-001 was administered post-injury, and kidney tissue and blood samples were collected at 1, 3, and 7 days. In parallel, HK-2 human renal tubular epithelial cells were treated with TGF-β to mimic fibrotic conditions in vitro and assess the cellular mechanisms of MIT-001.
Results : MIT-001 treatment significantly improved renal function in IRI mice, as evidenced by reduced blood urea nitrogen (BUN) and serum creatinine levels at days 3 and 7 post-injury. Histological and molecular analyses revealed that MIT-001 markedly suppressed fibrotic and inflammatory markers, including F4/80, collagen IV, α-smooth muscle actin (α-SMA), and TGF-β. Conversely, E-cadherin expression, indicative of preserved epithelial phenotype, was notably increased. In terms of ferroptosis regulation, MIT-001 enhanced the expression of xCT, SLC7A11, and GPX4—key proteins involved in antioxidant defense—and reduced levels of 4-HNE, a lipid peroxidation marker. Furthermore, MIT-001 significantly decreased the expression of HMGB1 and nuclear NF-κB, suggesting a robust anti-inflammatory effect in renal tissue.
Conclusion : This study confirms the detrimental role of IRI in driving renal fibrosis and functional decline. Importantly, MIT-001 demonstrated protective efficacy by targeting multiple damaging pathways—namely, inflammation, oxidative stress, and ferroptosis. These findings support the potential of MIT-001 as a promising therapeutic candidate for preventing or attenuating CKD progression following ischemic renal injury.

Keywords：MIT-001, renal fibrosis, ferroptosis

Poster Presentation：Chronic Kidney Disease
Poster No.：B0149
Abstract Submission No.：APCN20250609

NQO1 Deficiency Disrupts Glomerular and Tubular Adaptation in Diabetic Kidney Disease

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Abstract
Background:
Diabetic kidney disease (DKD) is a leading cause of end-stage renal disease, characterized by progressive damage to both glomerular and tubular structures. Oxidative stress is a central driver of this damage, disrupting redox homeostasis and accelerating cellular dysfunction. NAD(P)H:quinone oxidoreductase 1 (NQO1), a key antioxidant enzyme regulated by the Nrf2 pathway, plays an important role in protecting renal tissue from oxidative injury. However, its specific functions in different renal compartments under diabetic stress remain unclear. This study aimed to investigate how NQO1 deficiency affects compartment-specific responses in the kidney during diabetes, with a focus on transcriptomic and functional changes.
Methods:
DKD was induced in wild-type (WT) and NQO1-knockout (NKO) mice via streptozotocin (STZ) injection. Renal function and injury were assessed by measuring fasting blood glucose levels, urinary albumin-to-creatinine ratio (ACR), and ultrastructural changes using electron microscopy. To explore molecular mechanisms, transcriptomic profiling was performed on isolated glomeruli and proximal convoluted tubules (PCT), defined by histological markers (CD10, CD31, PanCK). Gene Set Enrichment Analysis (GSEA) was conducted to identify key pathways altered by NQO1 deficiency.
Results:
Despite similar levels of hyperglycemia, NKO-STZ mice exhibited significantly higher ACR and more severe podocyte foot process effacement compared to WT-STZ mice, indicating greater glomerular vulnerability. Thickening of the glomerular basement membrane was also more pronounced in the NKO group. GSEA revealed that ribosome biogenesis and immune-related pathways were upregulated in WT-STZ glomeruli, suggesting an adaptive response to hyperglycemia. These pathways were notably suppressed in NKO-STZ glomeruli, pointing to impaired adaptation. The adherens junction pathway—critical for podocyte integrity—was activated in WT-STZ but diminished in NKO-STZ glomeruli, with downregulation of key genes such as Actg1, Ctnna1, Tjp1, Rhoa, and Iqgap1. In PCTs, metabolic and cytoskeletal pathways were elevated in WT-STZ mice but consistently suppressed in NKO-STZ mice, indicating reduced tubular resilience.
Conclusion:
NQO1 deficiency disrupts compartment-specific adaptive mechanisms in the diabetic kidney, resulting in aggravated glomerular damage and impaired tubular metabolic flexibility. These findings highlight NQO1 as a promising therapeutic target for preserving renal function under diabetic conditions.

Keywords：Diabetic Kidney Disease , NQO1 Deficiency, Glomerular and Tubular Adaptation, Transcriptomic Analysis

Poster Presentation：Chronic Kidney Disease
Poster No.：B0150
Abstract Submission No.：APCN20250611

Isovitexin effectively alleviate renal fibrosis by targeting ferroptosis

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Abstract

Renal fibrosis is a major pathological disease of chronic kidney disease (CKD). Without timely intervention, progressive fibrosis can lead to a gradual loss of renal function, ultimately resulting in uremia and even kidney failure. Therefore, the identification of effective therapeutic agents capable of alleviating renal fibrosis and delaying its progression is of urgent clinical importance. Isovitexin, a food-derived phytochemical found in dietary rice products, has been previously reported to exhibit a variety of biological activities, including antioxidant, anti-inflammatory, antibacterial, and anti-tumor effects. However, its potential role and underlying mechanisms in renal fibrosis remain unexplored. This study aimed to investigate the protective effects and molecular mechanisms of isovitexin against renal fibrosis using both in vivo and in vitro models. In unilateral ureteral obstruction (UUO) animal model, followed by daily oral administration of isovitexin at doses of 25 mg/kg/day and 50 mg/kg/day. The results demonstrated that isovitexin markedly alleviated UUO-induced renal hypertrophy, reduced collagen deposition, and suppressed the expression of fibrosis-related markers such as α-smooth muscle actin (α-SMA), collagen I, and fibronectin, thereby effectively mitigating renal fibrosis. Furthermore, isovitexin exhibited non-toxicity, indicating a favorable safety profile. Additional, we found that isovitexin attenuated chronic injury and the expression of fibrosis-associated proteins in NRK-52E renal tubular epithelial cells induced by indoxyl sulfate (IS). FerroOrange staining further revealed that IS treatment led to intracellular ferrous iron accumulation, while isovitexin significantly reduced IS-induced iron contents and restored the expression of glutathione peroxidase 4 (GPX4). In addition, isovitexin decreased the production of reactive oxygen species (ROS) and the lipid peroxidation end-product malondialdehyde (MDA), thereby alleviating oxidative damage. In conclusion, this study is the first to demonstrate that isovitexin can effectively inhibit ROS-induced renal fibrosis by targeting ferroptosis. These findings provide experimental evidence supporting the potential of isovitexin as a novel therapeutic candidate for the treatment of renal fibrosis.

Keywords：Renal fibrosis, isovitexin, ferroptosis, ROS

Poster Presentation：Chronic Kidney Disease
Poster No.：B0151
Abstract Submission No.：APCN20250630

Regulatory Mechanisms of HNF4a Isoform Expression in Diabetic Kidney Disease

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Abstract

HNF4α is a key transcription factor, known for its pivotal role in regulating various cellular metabolism, proliferation and differentiation. In humans, HNF4α is expressed in 12 isoforms, due to selective promoter usage (P1 and P2) and alternative splicing. Theses isoforms exhibit distinct biological functions and tissue-specific expression patterns, contributing to the development of various diseases including diabetes, cancer and metabolic syndromes. Despite growing evidence suggesting that HNF4a isoform expression may play a role in disease progression, the functional relevance and regulation of HNF4a isoforms in diabetic kidney disease (DKD) remain unclear.

In this study, we aimed to investigate isoform-specific expression changes of HNF4α under injury conditions and the relation between isoform and DKD. Using the renal proximal tubular cell line, we mimicked DKD-related injury by exposing cells to high glucose and hypoxic conditions. Semi-quantitative RT-PCR analysis was conducted to assess the differential expression of HNF4α isoforms. We also examined isoform profiles in human kidney tissues from healthy individuals and patients with DKD and evaluated relevance between HNF4α isoform and DKD.

Our findings revealed that injury conditions induced alterations in the expression profile of HNF4α isoforms. Specifically, isoforms transcribed from the P1 promoter exhibited significantly reduced expression under injury condition, whereas P2-derived isoforms were notably upregulated. Furthermore, we observed time-dependent changes in isoform expression, suggesting dynamic regulation in response to sustained injury.

These results suggest that DKD progression is associated with a shift in the alternative splicing and promoter usage of HNF4α, potentially altering its regulatory function in proximal tubule cells. As HNF4α plays a critical role in maintaining metabolic homeostasis in renal proximal tubule cells, isoform-specific regulation may represent a key mechanism in DKD pathogenesis. Further elucidation of the splicing mechanisms and regulatory factors influencing HNF4α isoform dynamics may offer new therapeutic opportunities, such as isoform-targeted gene modulation or epigenetic therapies, for the treatment or prevention of DKD.

Keywords：HNF4a, DKD

Poster Presentation：Chronic Kidney Disease
Poster No.：B0152
Abstract Submission No.：APCN20250647

The Functions of Tubular Cell MST1 in Kidney Injury

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Abstract
Introduction: Mammalian sterile twenty-like 1 (MST1), also known as Serine/threonine-protein kinase 4 (STK4), serves as a core component of the Hippo signaling pathway, which controls the organ size and cell numbers by modulating cell proliferation, differentiation and death. MST1 is abundantly expressed in the kidney. Our previous study revealed that renal tubule-specific Mst1 and Mst2 double knockout induced chronic kidney disease through mutual activation of the TNF-α and the Yap signaling pathways in mice. However, the specific role of tubular cell MST1 in kidney injury and fibrosis remains to be elucidated.
Methods: We generated renal tubule-specific Mst1 knockout (M1KO) mice by intercrossing floxed Mst1 mice with Ksp-Cre mice. Kidney injury was induced by unilateral ureteral obstruction (UUO) or intraperitoneal injection of cisplatin. Staurosporine (STS) was applied to trigger apoptosis in TKPTS mouse proximal tubular cells in vitro. We employed Masson's trichrome staining, Picrosirius red staining, PAS staining and TUNEL assay to evaluate kidney injury and fibrosis after UUO or cisplatin. Moreover, qPCR and Western blotting were used to determine the mechanisms responsible for the phenotypes observed.
Results: MST1 protein expression in the kidney significantly increased in different kidney injury mouse models although the mRNA levels did not change. Compared with WT mice, M1KO mice exhibited increased tubular cell apoptosis and renal fibrosis following both UUO and cisplatin treatment, and these effects were independent of YAP. Inhibition of MST1 expression enhanced cell apoptosis induced by serum starvation or by STS in TKPTS cells, without any effects on the YAP activity. Conversely, MST1 overexpression inhibited apoptosis dose-dependently in TKPTS cells.
Conclusion: MST1 deficiency significantly exacerbates kidney injury and fibrosis independently of YAP. Increased MST1 expression in response to kidney injury protects against tubular cell death.

Keywords：MST1; UUO; Cisplatin; Kidney injury; Apoptosis; Fibrosis

Poster Presentation：Chronic Kidney Disease
Poster No.：B0153
Abstract Submission No.：APCN20250654

Electrolyte and Renal Biomarker Variations in Diabetic and Non-Diabetic Chronic Kidney Disease Patients

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Abstract

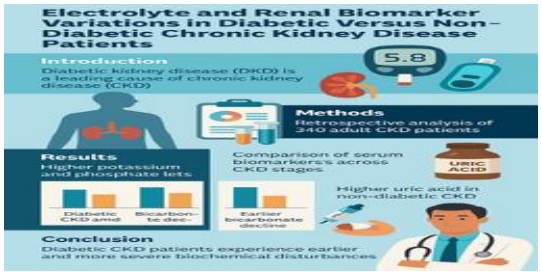
Diabetic kidney disease (DKD) is a leading cause of chronic kidney disease (CKD) worldwide and in Malaysia, where the prevalence of diabetes continues to rise. Despite well-established links between diabetes and renal damage, few studies have directly compared electrolyte and renal biomarker profiles in diabetic versus non-diabetic CKD across disease stages. We aimed to delineate these biochemical differences to improve early risk identification and personalized management.

We performed a retrospective cross-sectional analysis of laboratory records from 340 adult CKD patients at two nephrology centers in Malaysia. Patients were stratified into diabetic and non-diabetic CKD groups. We compared serum sodium, potassium, phosphate, bicarbonate, uric acid, creatinine, and estimated glomerular filtration rate (eGFR) across CKD stages 1–5. Statistical analyses included ANOVA for group comparisons, logistic regression, and subgroup analyses by age and hemoglobin A1c (HbA1c) were available.

Diabetic CKD patients had significantly higher potassium ($p = 0.003$) and phosphate ($p = 0.01$) levels than non-diabetic CKD patients, along with an earlier decline in bicarbonate that suggests accelerated metabolic acidosis. The decline in eGFR was also steeper in the diabetic group across all stages. In contrast, serum uric acid was significantly higher in the non-diabetic patients, pointing to divergent inflammatory or tubular injury pathways. Subgroup analysis indicated that insulin-dependent diabetic patients had more pronounced acidosis and electrolyte derangements than those on oral hypoglycemic therapy.

Our study reveals distinct biochemical trajectories in diabetic versus non-diabetic CKD, with diabetic patients experiencing earlier and more severe electrolyte and acid–base disturbances. These findings reinforce the need for diabetes-specific monitoring protocols and may inform future risk models for diabetic kidney disease progression in Southeast Asia.

Keywords：Diabetic kidney disease, Electrolyte imbalance, Uric acid, eGFR, Metabolic acidosis, CKD phenotyping



Poster Presentation：Chronic Kidney Disease
Poster No.：B0154
Abstract Submission No.：APCN20250655

Biochemical Trajectories of Diabetic Kidney Disease in a Malaysian Type 2 Diabetic Mellitus versus Non-Diabetic CKD patients

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Abstract

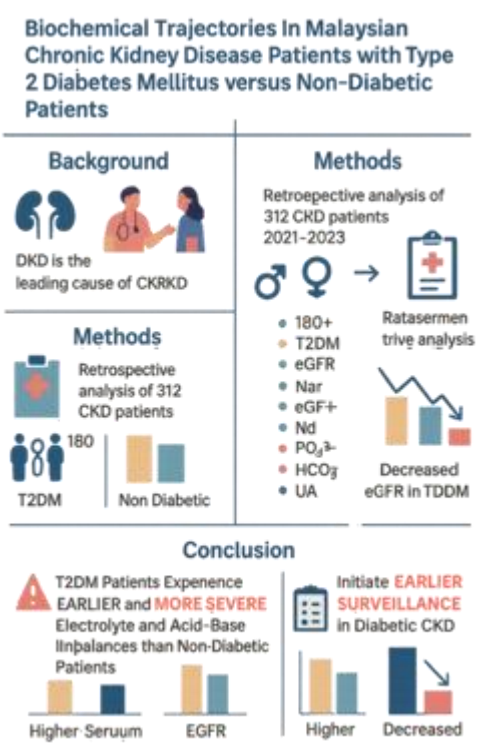
Diabetic kidney disease (DKD) is the leading cause of chronic kidney disease (CKD) worldwide and in Malaysia, contributing to an increasing proportion of end-stage kidney failure cases. While standard renal markers like serum creatinine and estimated glomerular filtration rate (eGFR) guide CKD diagnosis, the role of electrolyte and acid–base abnormalities in diabetic versus non-diabetic CKD remains underexplored in Southeast Asia.

We retrospectively analyzed laboratory records of 312 CKD patients treated at two nephrology centers in Malaysia (2021–2023). Patients were categorized into a type 2 diabetes mellitus (T2DM) CKD group (n = 180) and a non-diabetic CKD group (n = 132). We compared serum sodium, potassium, phosphate, bicarbonate, uric acid, and eGFR between these groups. Subgroup analyses were performed by age (< 60 vs ≥ 60 years), glycated hemoglobin (HbA1c) level, and insulin therapy status. Multivariate linear regression and ANOVA were used for statistical comparisons.

CKD patients with T2DM had significantly higher serum potassium (mean 5.3 ± 0.6 vs 4.8 ± 0.5 mmol/L, $p < 0.001$) and lower bicarbonate (20.1 ± 2.4 vs 22.5 ± 2.2 mmol/L, $p < 0.001$) than non-diabetic CKD patients, indicating a greater burden of subclinical hyperkalemia and metabolic acidosis. Serum phosphate and uric acid were also elevated in the diabetic cohort. Furthermore, eGFR was consistently lower at each CKD stage in the T2DM group (mean 34.6 ± 12.3 vs 41.2 ± 10.7 mL/min/1.73 m², $p = 0.005$). Subgroup analyses revealed more pronounced electrolyte and acid–base derangements in patients with poor glycemic control (HbA1c > 8%) and those on insulin therapy.

CKD patients with diabetes experience earlier and more severe electrolyte and acid–base imbalances than their non-diabetic counterparts particularly hyperkalemia and metabolic acidosis. These findings underscore the importance of initiating biochemical surveillance earlier in diabetic CKD management. Incorporating electrolyte profiles into DKD risk stratification may help improve patient outcomes in high-burden settings like Malaysia.

Keywords：Diabetic kidney disease, Potassium and phosphate, HbA1c, Insulin therapy, eGFR decline, Metabolic markers


Biochemical Trajectories In Malaysian Chronic Kidney Disease Patients with Type 2 Diabetes Mellitus versus Non-Diabetic Patients
Background
DKD is the leading cause of CKRKD
Methods
Retrospective analysis of 312 CKD patients
180
T2DM
Non Diabetic
Methods
Retroepective analysis of 312 CKD patients 2021-2023
180+
T2DM
eGFR
Nar
eGF+
Nd
PO_4^{3-}
HCO_3^-
UA
Ratasermen trive analysis
Decreased eGFR in TDDM
Conclusion
T2DM Patients Expenence EARLIER and MORE ŞEVERE Electrolyte and Acid-Base IInbalances than Non-Diabetic Patients
Higher Seruum
EGFR
Initiate EARLIER SURVEILLANCE in Diabetic CKD
Higher
Decreased

Poster Presentation：Chronic Kidney Disease
Poster No.：B0155
Abstract Submission No.：APCN20250656

Assessment of Inflammatory Burden in Chronic Kidney Disease Using Albumin to Globulin Ratio and Hematological Markers

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Abstract

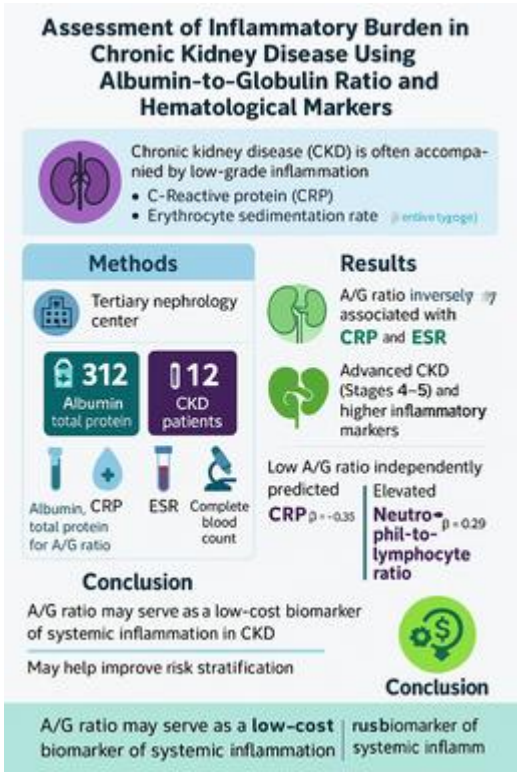
Chronic kidney disease (CKD) is a progressive condition often accompanied by chronic low-grade inflammation, which accelerates renal decline and elevates cardiovascular risk. While C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) are routinely used to assess inflammation, the albumin-to-globulin (A/G) ratio has emerged as a potential surrogate marker of inflammatory and immunologic status. However, the utility of the A/G ratio in CKD has not been well studied in Southeast Asian populations.

We retrospectively analyzed data from 312 adult CKD patients (Stages 1–5) at a tertiary nephrology center in Malaysia. Laboratory parameters included serum albumin and total protein (to calculate A/G ratio), CRP, ESR, and complete blood count (CBC). Patients were stratified by CKD stage, and inflammatory markers were compared across these stages. We used correlation and multivariate regression analyses to examine associations between the A/G ratio and established inflammatory markers such as CRP, ESR, and the neutrophil-to-lymphocyte ratio.

The A/G ratio was inversely correlated with CRP ($r = –0.48$, $p < 0.001$) and ESR ($r = –0.44$, $p < 0.001$) across all CKD stages. Patients with advanced CKD (Stages 4–5) had lower A/G ratios alongside higher CRP and ESR levels. In a multivariate model adjusting for age, sex, and comorbidities, a low A/G ratio remained an independent predictor of elevated CRP ($\beta = –0.35$, $p = 0.002$) and an increased neutrophil-to-lymphocyte ratio ($\beta = –0.29$, $p = 0.006$). Notably, the A/G ratio appeared to capture cumulative inflammatory burden in CKD patients even in those with normal nutritional status.

The albumin-to-globulin ratio shows strong potential as a low-cost, readily available biomarker of systemic inflammation in CKD. Its significant associations with CRP, ESR, and blood cell indices underscore its clinical relevance. Incorporating the A/G ratio into routine monitoring could improve risk stratification and early identification of patients with a high inflammatory burden. Ultimately, this simple measure may aid more precise CKD management in resource-limited settings.

Keywords：Albumin-to-globulin ratio, Chronic inflammation, Hematological biomarkers, CRP, ESR, Inflammation in CKD


Assessment of Inflammatory Burden in Chronic Kidney Disease Using Albumin-to-Globulin Ratio and Hematological Markers
Chronic kidney disease (CKD) is often accompanied by low-grade inflammation
C-Reactive protein (CRP)
Erythrocyte sedimentation rate
Methods
Tertiary nephrology center
312
Albumin total protein
12
CKD patients
Albumin, total protein for A/G ratio
CRP
ESR
Complete blood count
Results
A/G ratio inversely associated with CRP and ESR
Advanced CKD (Stages 4–5) and higher inflammatory markers
Low A/G ratio independently predicted CRP β = -0.35
Elevated Neutrophil-to-lymphocyte ratio β = 0.29
Conclusion
A/G ratio may serve as a low-cost biomarker of systemic inflammation in CKD
May help improve risk stratification
Conclusion
A/G ratio may serve as a low-cost biomarker of systemic inflammation
rusbiomarker of systemic inflamm

Poster Presentation：Chronic Kidney Disease
Poster No.：B0156
Abstract Submission No.：APCN20250658

Whole Exome Sequencing and Polygenic Risk Assessment for Kidney Functions: Validation with international cohorts

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Abstract
Introduction: Taiwan has the highest prevalence of chronic kidney disease (CKD) and end-stage kidney disease (ESKD) globally, making them major public health concerns with significant morbidity, mortality, and healthcare burden. While genetic risk factors for kidney disease have been identified in previous studies, the contribution of rare genetic variants remains unclear.
Methods: This study utilized whole-exome sequencing (WES) to investigate the role of missense rare variants in CKD and ESKD susceptibility, providing new insights into their genetic architecture. We rigorously recruited 500 Taiwanese individuals from Taipei Medical University using strict clinical diagnostic criteria, including 200 CKD cases, 200 ESKD cases, and 100 healthy controls. Independent validation was performed using cohorts from the All of Us Research Program (AoU) (N=222) and the Tohoku Medical Megabank Organization (ToMMo) (N=140).
Results: We identified rare pathogenic variants in known monogenic kidney disease genes, including PKD1 and COL4A4, confirming their role in disease susceptibility. We replicated GWAS-reported genes such as SPI1, RIN3, FTO, SIPA1L3, and EEF1E1, highlighting their contribution through both common and rare variants. Beyond previously reported genes, we identified novel rare pathogenic variants in PEX1, GANAB, DYNC2H1, and PROKR2. Pathway enrichment analysis suggested that ciliopathies, inflammation, and metabolic dysfunction may contribute to kidney disease progression. Furthermore, the polygenic score (PGS) for ESKD demonstrated strong predictive utility for kidney function, with high genetic risk having a greater influence than comorbidities such as diabetes and overweight. The prediction power of ESKD PGS was further validated in the AoU Asian population.
Conclusions: This study provides novel insights into the genetic architecture of CKD and ESKD in the Taiwanese population, leveraging a well-defined hospital-based cohort with strict clinical diagnostic criteria to ensure precise phenotype classification. We purpose that individuals with high genetic risk may benefit from earlier interventions, while those with lower PGS may be better managed through lifestyle modifications targeting comorbidities. The findings highlight the importance of preventive strategies and precision medicine in kidney disease management.

Keywords：Chronic kidney disease, end-stage kidney disease, rare variants, whole-exome sequencing, genetic susceptibility, polygenic risk score, risk stratification

Poster Presentation：Chronic Kidney Disease
Poster No.：B0157
Abstract Submission No.：APCN20250661

Manipulation of gut microbiota attunes renal fatty acid metabolism and immune landscape to ameliorate chronic kidney disease

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Abstract
Introduction: Although intestinal dysbiosis has been linked to the progression of chronic kidney disease (CKD), the causality of gut microbiota in renal health is not yet entirely established. To address this, nephroprotective potential of Bacteroides eggerthii (B. eggerthii), a gut bacterium associated with CKD progression, was tested in a high-fat diet fed induced metabolic CKD model.
Method: Clinical isolates from the feces of a healthy donor were used to denote the change of renal function. Microalbuminuria, serum creatinine and histologic examination were conducted to evaluate the effectiveness associated with oral gavage of Bacteroides eggerthii. Single-cell RNA sequencing (scRNA-seq) and spatial transcriptomics (ST) were employed to interrogate the possible renal protective mechanism associated with mouse kidneys.
Results: We found that oral administration of a strain of B. eggerthii was accompanied with altered gut microbiota compositions. Further examination of the renal tissues via scRNA-seq revealed that supplementation with B. eggerthii alleviated metabolic and immune disturbance in diverse tubule epithelial cell types and renal endothelial cells of CKD mice. Predictions of intercellular crosstalk inferred a restoration of cell-cell communications between renal T cells and other cell types by B. eggerthii supplement. In addition, a shift of the dynamic transition in infiltrating macrophage activation in CKD was reversed after replenishing B. eggerthii. Moreover, ST analyses of mouse kidneys demonstrated spatially distinct effects of B. eggerthii supplement on reprogramming transcriptional aberrations in CKD, highlighted by improvement of dysregulated fatty acid metabolism and complement activation in the outer and inner portion of kidneys, respectively.
Conclusion: These findings provide a mechanistic basis for the beneficial effects of a potentially nephroprotective bacterial strain, further extending our understanding of the gut-kidney axis in CKD.

Keywords：chronic kidney disease, gut microbiota, Bacteroides eggerthii, fatty acid metabolism

Poster Presentation：Chronic Kidney Disease
Poster No.：B0158
Abstract Submission No.：APCN20250662

Nicotinamide confers partial renoprotection in Pkd1-deficient mice

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Abstract
Autosomal dominant polycystic kidney disease (ADPKD) is a slowly progressive disease characterized by cyst formation and renal function decline with variable speed in individual patients. How to better protect renal function and delay the onset of end-stage kidney disease is still not optimized. Nicotinamide (NAM) has been shown to provide energy and protect against acute kidney injury and neurodegenerative diseases. Whether NAM supplement provides renal protective effects in ADPKD remains controversial. Here we investigated the effects of NAM on kidney protection in Pkd1 miR-Tg mice which is a moderately progressive model of ADPKD. Pkd1 miR-Tg mice were treated with NAM (0.25 and 0.5 g/kg/day) by intraperitoneal injection from postnatal day (PN)35 to PN90 and the protective effects on cyst growth and kidney function were assessed. We found that AM did not reduce fractional kidney weight but reduced cyst burden slightly. NAM significantly reduced blood urea nitrogen and the mRNA expression of profibrotic genes (Fn1, Col1a2, and Acta2), EMT-related genes (Vimentin and Claudin4), and TNF pathway-related genes (Il1b, Tnf, Il6, Tcf7, Jag2, Mcp1). The elevated plasma levels of TNF in Pkd1 mutant mice were significantly suppressed with NAM treatment. Western blotting showed significantly reduced expression of SIRT1 protein with NAM treatment. However, NAM treatment did not alter the proliferation indices or fibrosis scores on kidney sections. In conclusion, we demonstrated a partial treatment effect of NAM in a Pkd1-deficient mouse model. These results suggest that NAM could have a renoprotective effect on disease progression in ADPKD through modulating inflammation.

Keywords：nicotinamide, polycystic kidney disease

Poster Presentation：Chronic Kidney Disease
Poster No.：B0159
Abstract Submission No.：APCN20250674

Ellagic acid mitigates renal fibrogenesis by inhibiting the process of epithelial-to-mesenchymal transition.

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Abstract

Objective: Ellagic acid (EA), a polyphenolic compound present in various fruits and vegetables, exhibits anti-inflammatory, anti-apoptotic, antioxidant, and anti-fibrotic properties across a range of diseases; however, its specific function in renal fibrogenesis remains to be elucidated.

Materials and Methods: This study employed an in vivo mouse model of unilateral ureteral obstruction (UUO) alongside an in vitro model utilizing HK-2 cell lines subjected to treatment with EA and transforming growth factor β1 (TGF-β1). The expression of proteins associated with epithelial-to-mesenchymal transition (EMT) in UUO mice was assessed through immunohistochemical staining. Biochemical assays were conducted to evaluate liver and renal function, while Western blot analysis was utilized to quantify EMT-related proteins. The MTT assay was performed to assess cell viability.

Results: In UUO mice administered EA, both microscopic examination via immunohistochemical staining and Western blot analysis revealed a significant reduction in the expression of fibrotic markers (α-SMA, fibronectin, and collagen I) and EMT markers (vimentin and N-cadherin) compared to the sham control group. In HK-2 cells treated with TGF-β1, EA was found to diminish cell motility and the expression levels of α-SMA, collagen I, fibronectin, N-cadherin, and vimentin.

Conclusion: EA effectively attenuated the progression of morphological changes and concurrently inhibited the expression of fibrotic and EMT-related proteins both in vitro and in vivo. These findings enhance our understanding of the role of EA in mitigating renal fibrogenesis and suggest its potential therapeutic application in the management of chronic kidney disease.

Keywords：Ellagic acid, Epithelial-to-mesenchymal transition, Transforming growth factor β1, Unilateral ureteral obstruction

Poster Presentation：Chronic Kidney Disease
Poster No.：B0160
Abstract Submission No.：APCN20250682

Are inflammatory cytokines and adipokines different in obese patients with and without chronic kidney disease (CKD)?

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Abstract

Introduction: Global epidemic of obesity & metabolic disorders are fuelling increasing cases of CKD worldwide. Besides having higher incidence of DM & hypertension, obesity is independent risk factor for CKD. Mechanisms involved are poorly studied, though obesity related inflammation mediated by shift in adipokine and cytokine production towards pro-inflammatory state is implicated.

We in this case control study looked at important pro-inflammatory mediators (leptin, IL-6, TNF-α) and anti-inflammatory mediators (adiponectin, IL-10) in obese with & without CKD, non-obese CKD & healthy controls.

Methods: 50 consenting subjects in each group were studied. Besides detailed history, co-morbidity charting, BMI calculation; serum levels of HsCRP, adipokines (leptin & adiponectin) & cytokines (IL-6, TNF-α & IL-10) were assessed using commercially available ELISA kits.

Results: Table shows demographic, clinical & study parameters of each group. Patient groups had similar representation of DM & were slightly older than controls. Obese subjects with & without CKD had higher HsCRP, leptin & IL6 than controls & CKD patients, with obese patients with CKD showing maximum aberrations. Adiponectin concentration was higher in patients with obesity alone but suppressed in patients with obesity & CKD.

Conclusion: Inflammation & pro-inflammatory milieu as evidenced by high levels of Hs-CRP, IL-6 & leptin and low levels of adiponectin might be important drivers for obesity related complications like CKD. Larger, prospective studies are required to confirm the same

Keywords：Obesity, Inflammation, Diabetic, Adiponectin, Cytokines

Poster Presentation：Chronic Kidney Disease
Poster No.：B0161
Abstract Submission No.：APCN20250753

Cystatin C: A Novel Endogenous Predictive Marker for Cardiovascular Risk Assessment in Chronic Kidney Disease Patients

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Abstract
Background:
Cystatin C, an endogenous cysteine protease inhibitor, has emerged as a superior marker over serum creatinine for evaluating renal function, particularly for detecting early or subtle declines in glomerular filtration rate (GFR). In patients with chronic kidney disease (CKD), cardiovascular disease (CVD) remains the leading cause of morbidity and mortality, often surpassing progression to end-stage renal disease. This study investigates the potential of cystatin C as a predictive marker for cardiovascular risk in CKD patients, and proposes a novel model for estimating serum cystatin C levels using routine biochemical parameters, thereby enabling cost-effective cardiovascular risk stratification.
Methods:
A cross-sectional study was conducted involving 397 participants divided into four groups: CKD (n=71), CVD (n=127), CKD with CVD (n=37), and healthy controls (n=162). Blood samples were analyzed for biochemical parameters including total cholesterol, triglycerides, HDL, LDL, VLDL, urea, creatinine, glucose, sodium, potassium, total protein, albumin, hs-CRP, and cystatin C. Measurements were performed using an automated biochemistry analyzer (Olympus AU-400), and hematological parameters including hemoglobin and total leukocyte count were assessed using Beckman Coulter LH-500.
Results:
The prevalence of diabetes was significantly higher in all disease groups compared to controls ($p<0.0001$). CKD and CVD groups exhibited significantly elevated levels of total cholesterol, triglycerides, VLDL, urea, creatinine, and cystatin C ($p<0.05$). In CKD patients with coexisting CVD, potassium, glucose, urea, creatinine, cystatin C, and total leukocyte count were markedly higher than in healthy controls ($p<0.05$). A multivariable linear regression model was developed to estimate serum cystatin C levels based on independent predictors including age, potassium, albumin, urea, creatinine, hemoglobin, and a constant term. The model demonstrated statistically significant contributions from all predictors ($p<0.05$), with creatinine and urea having the highest coefficients.
Conclusion:
This study establishes cystatin C as a sensitive and reliable biomarker for cardiovascular risk in CKD patients and introduces a novel predictive equation derived from standard laboratory parameters. This model may serve as a clinically accessible and economically feasible alternative to direct cystatin C assays for early cardiovascular risk detection. Further validation through large-scale prospective studies is recommended to confirm the model's utility in routine clinical practice.

Keywords：Cystatin C Chronic Kidney Disease (CKD) Cardiovascular Disease (CVD) Renal Biomarkers, Glomerular Filtration Rate (GFR), Endogenous Marker

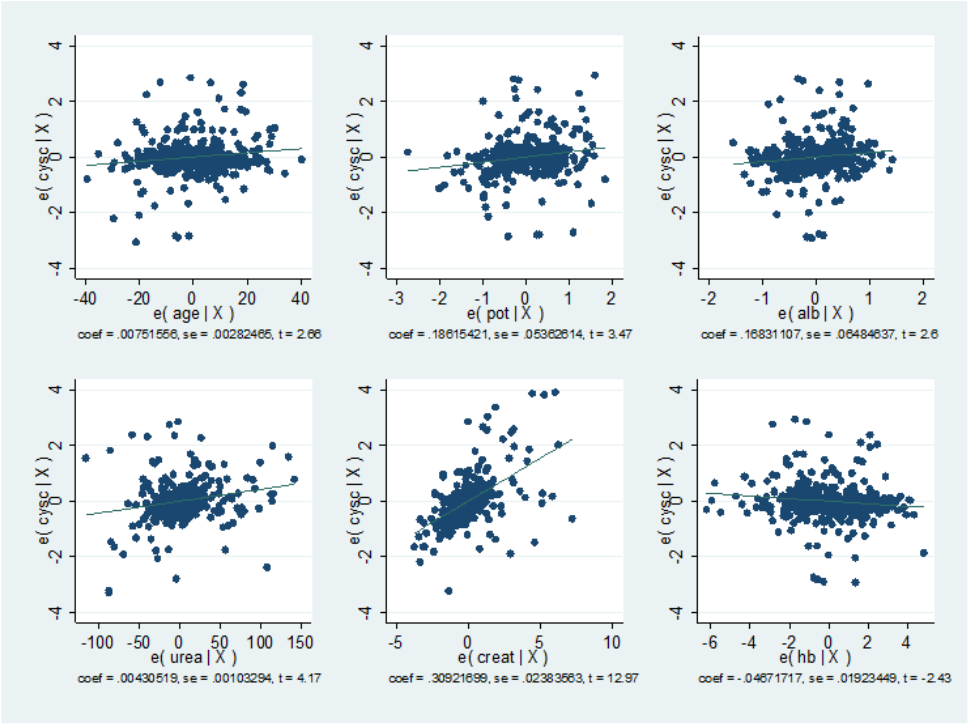

Figure: The figure indicating the effect of independent variables like age, potassium, albumin urea, creatinine, hemoglobin, constant on dependent variable, i.e. cystatin C. There was significant effect of age (p=0.008), potassium (p=0.001), albumin (p=0.01), urea (p=0.001), creatinine (p=0.0001), hemoglobin (p=0.016), constant (p=0.022) on Cystatin level, after controlling for other independent variables.

Poster Presentation：Chronic Kidney Disease
Poster No.：B0162
Abstract Submission No.：APCN20250757

ALOX5 Regulates Renal Fibrosis Through Modulation of Matrix Remodeling and Inflammatory Responses

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Abstract

Introduction: Renal fibrosis, a common pathological feature of chronic kidney disease (CKD), exhibits excessive extracellular matrix accumulation and inflammatory cell infiltration. 5-lipoxygenase (ALOX5), a key enzyme in leukotriene biosynthesis, plays crucial roles in inflammatory cell recruitment and inflammatory responses. While ALOX5-mediated leukotriene production is known to regulate inflammatory cascade activation and immune cell recruitment, its specific contribution to collagen deposition and matrix remodeling in renal fibrosis remains unclear.

Methods: We investigated the role of ALOX5 in renal fibrosis using TGFβ-stimulated HKC8 cells (human proximal tubular cell) with pharmacological ALOX5 inhibition. We analyzed the expression of fibrotic markers and pro-inflammatory cytokines (IL-6, IL-1β) using Western blot and qPCR. We further examined matrix metalloproteinases (MMPs), total soluble collagen expression, and collagen turnover through molecular and biochemical analyses. To evaluate immune cell recruitment, we established co-culture systems with immune cells (THP-1).

Results: ALOX5 inhibition effectively suppressed TGFβ-induced increases in kidney injury molecule-1 (KIM-1) and alpha-smooth muscle actin (α-SMA) expression in HKC8 cells. Modulation of the ALOX5 pathway significantly affected collagen expression, attenuating the expression of total soluble collagen. Notably, collagen composition and turnover were also affected, as evidenced by the suppression of the TGF-β-induced elevation in the collagen III to I ratio, which even showed changes at different time points during the fibrotic processes. Mechanistically, ALOX5 inhibition restored the TGFβ-suppressed expression and enzymatic activity of MMP3, a key collagenase with substrate specificity for collagen III over collagen I, thereby maintaining appropriate collagen turnover and limiting fibrotic progression, particularly through collagen III downregulation. Furthermore, blockade of ALOX5 pathway attenuated TGF β-induced surges in pro-fibrotic Smad2, pro-inflammatory cytokines IL-6, IL-1β levels, while increasing p56 expression and reducing immune cell infiltration in co-culture experiments, highlighting its regulatory role in both matrix remodeling and inflammatory responses.

Conclusion: Our findings reveal that ALOX5 serves as a critical mediator in renal fibrosis by regulating both collagen deposition and inflammatory responses. ALOX5 pathway modulation affects multiple aspects of fibrotic progression: regulation of collagen turnover, inflammatory cytokine production, and immune cell recruitment. These results establish ALOX5 as a potential therapeutic target in renal fibrosis, particularly through its novel role in matrix remodeling.

Keywords：ALOX5, renal fibrosis, proximal tubule, collagen, matrix remodeling

Poster Presentation：Chronic Kidney Disease
Poster No.：B0163
Abstract Submission No.：APCN20250760

Correlation of Socioeconomic Factors with the Prevalence of Chronic Kidney Disease Due to Diabetes Mellitus in Indonesia

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Abstract
Background:
The relationship between socioeconomic status (SES) and the burden of chronic kidney disease (CKD) due to type I and type II diabetes mellitus (CKD-DMI and CKD-DMII) remains underexplored in Indonesia. This study aimed to examine the correlation between various SES indicators and the prevalence and incidence of CKD-DMI and CKD-DMII.

Methods:
This study utilized prevalence and incidence data from the Global Burden of Disease (GBD) study, along with socioeconomic data from the Indonesian Population Census. Socioeconomic indicators analyzed included poverty rates (percentage of poor individuals), gender ratio (percentage ratio of male to female), employment rate (percentage of employed individuals), and average number of completed years education of each province. Age-standardized prevalence and incidence rates of CKD-DMI and CKD-DMII were analyzed using Spearman's correlation test to assess correlations with SES variables.

Results:
Data from 34 Indonesian provinces were included in the analysis. The average CKD-DMI and CKD-DMII prevalence rates were 55.27 and 1860 per 100,000 individuals, respectively. Employment rate and average years of education showed a statistically significant positive correlation with CKD-DMII ($r = 0.553$ and $r = 0.567$, respectively; $p < 0.01$). No significant correlations were observed for CKD-DMI.

Conclusion:
There is a significant correlation between the prevalence of CKD due to type II diabetes and sociodemographic factors in Indonesia, particularly employment rate and education level. These findings highlight the complex interplay between SES and the burden of diabetic kidney disease and may inform future public health strategies.

Keywords：Chronic kidney disease, Socieconomy, Diabetes mellitus

	Mean (per 100000 individuals)	Poverty rate	Unemployment rate	Gender ratio	Employment rate	Number of education years
CKD-DMI Prevalence	55.27	-0.051	0.276	-0.094	0.320	0.047
CKD-DMII Prevalence	1860	-0.167	0.220	0.084	0.553**	0.567**

**$p<0.01$

Poster Presentation：Chronic Kidney Disease
Poster No.：B0164
Abstract Submission No.：APCN20250766

Adipocyte-secreted factors compromise podocyte cytoskeletal integrity by inducing oxidative stress

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Abstract

Podocytes are specialized epithelial cells that cover the outer surfaces of glomerular capillaries, forming a critical part of the glomerular filtration barrier. Damage to podocytes leads to albuminuria, the most important early clinical characteristic of various glomerular diseases, which correlates with the development of chronic kidney disease (CKD). Metabolic syndrome-related nephropathy is one of the leading causes of CKD in Taiwan and worldwide. Although many studies have implicated obesity and adipose tissue-associated signaling in the development of glomerulopathy, whether and how these factors directly damage podocytes needs to be further explored. In this study, conditionally immortalized mouse podocytes were used to investigate the effects of adipocyte-secreted factors on podocytes. 3T3-L1 cells were induced to differentiate into adipocytes for the preparation of adipocyte-conditioned medium (ACM), while parental undifferentiated cells were used for preadipocyte-conditioned medium (PCM). We found that ACM treatment directly damaged the cultured podocytes, characterized by a reduction in focal adhesions, stress fibers, and interdigitating cellular junctions, as well as decreased levels of synaptopodin and podocin proteins, compared to untreated and PCM controls. Furthermore, a significant increase in intracellular reactive oxygen species (ROS) in ACM-treated podocytes was detected. Co-treatment with antioxidants alleviated the elevated cellular ROS and cytoskeletal damage in podocytes. Our findings suggest that adipocyte-secreted factors may directly impair structural integrity through the induction of oxidative stress, thereby contributing to podocyte dysfunction and leading to the progression of metabolic syndrome-related nephropathy.

Keywords：podocyte injury, adipocyte-secreted factors, oxidative stress, cytoskeleton

Poster Presentation：Chronic Kidney Disease
Poster No.：B0165
Abstract Submission No.：APCN20250772

Effects of aloe emodin against vascular calcification in ApoE knockout chronic kidney disease mice fed with high phosphate diet

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Background: The hallmark of vascular calcification (VC) is accumulation of calcium phosphate salts, primarily in the form of hydroxyapatite, within the vascular tissue, involving an active transdifferentiation of vascular smooth muscle cells (VSMCs) into osteochondroblast-like cells. It is commonly associated with pathological conditions such as chronic kidney disease (CKD), diabetes mellitus, and atherosclerosis. Vascular calcification (VC) decreases arterial elasticity and compliance, contributing to luminal narrowing and an elevated risk of cardiovascular events. Aloe-emodin (AE), a natural compound found in Aloe-vera leaves, has been in various pharmacological activities and showed antiviral, antimicrobial, and hepato-protective roles and anti-inflammatory activities. We investigated the effects of AE on inorganic phosphate (Pi)-induced VC in VSMCs and high phosphorus diet with induced vascular calcification in ApoE-knockout chronic kidney disease mice.
Methods: In vitro experiments were carried out by using Pi-induced vascular calcification in a mouse VSMCs, co-treated without or with different concentration of AE. In vivo study of vascular calcification was induced by oral high-phosphorus diet (1. 5% total phosphorus) for 8 weeks after 5/6 nephrectomy in ApoE-/- mice. A total of 24 male mice were divided to 3 groups, the control group (C57BL/6 undergone sham surgery and fed with chow diet), the VC group (ApoE -/- undergone 5/6 nephrectomy and fed with high-phosphorus diet), and the VCP group (ApoE -/- of 5/6 nephrectomy fed with high-phosphorus diet and phthiocol) (each n = 8). Transdermal glomerular filtration rate measurement (tGFR), pulse wave velocity (PWV), blood biochemical measurement, and pathology (Von Kossa stain) were performed.
Results: AE-treatment in VSMCs that cultured in high Pi medium suppressed reactive oxidative species production, ferroptosis, and subsequent cell death and calcification in a dose-dependent manner. AE suppressed osteogenic trans-differentiation (Runx2) via restoration of the PI3K/Akt pathway, subsequent activation of Nrf2/HO-1 anti-oxidation, and down-regulating inflammation (IL-1β, TNFα) as well. Chemical inhibition of either PI3K, Nrf2, or HO-1 all significantly abolished the protective effect of AE on Pi-induced apoptosis and calcification. Five-sixth nephrectomy and high-phosphorus diet in ApoE-/- mice significantly increased serum Ca, serum phosphorus, Mean arterial pressure (MAP), PWV levels, and calcium deposition in the aortic sections shown in Von Kossa stain. AE treatment in the VCP group significantly decreased serum phosphorus, MAP, PWV levels and aorta calcification, while increased tGFR, compared with VC group.
Conclusions: Aloe Emodin ameliorates phosphate-induced osteogenic trans-differentiation of VSMCs, inflammation/oxidative stress, ferroptosis, and subsequent vascular calcification through restoration of the PI3K/Akt pathway and subsequent Nrf2/HO-1 activation.
Keywords： Aloe Emodin, Vascular calcification, High-phosphorus diet, ApoE-knockout mice, Chronic kidney disease, PI3K/Akt pathway, Nrf2/HO-1 activation.

Poster Presentation：Chronic Kidney Disease
Poster No.：B0166
Abstract Submission No.：APCN20250781

SLC34A2 Drives Tubulointerstitial Fibrosis Through Metabolic Reprogramming and Partial Epithelial–Mesenchymal Transition

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Abstract

Introduction: Chronic kidney disease (CKD) is a growing global health burden with limited treatment options and a high risk of progression to end-stage renal disease. Tubulointerstitial fibrosis is a major contributor to CKD progression, making its inhibition a key therapeutic goal. The type II sodium-dependent phosphate cotransporter family (SLC34) includes SLC34A1 and SLC34A3, which are localized in renal proximal tubules, and SLC34A2, primarily found in intestinal epithelial cells. While SLC34A2 is traditionally associated with intestinal phosphate absorption, recent evidence suggests it may also play a role in kidney pathophysiology. This study explores the role of SLC34A2 in renal tubular metabolism and fibrosis, aiming to uncover a potential therapeutic target for CKD intervention.

Methods: In search of potential therapeutic targets for human renal fibrosis, we conducted bioinformatics analysis using cDNA microarray data, identified a candidate gene, SLC34A2, which is elevated in human fibrotic kidneys compared to normal kidneys, as observed in two CKD datasets from the public Gene Expression Omnibus (GEO) gene expression microarrays. To investigate the role of SLC34A2 in CKD, we employed unilateral ureteral obstruction (UUO) mouse models known to induce renal fibrosis and related syndromes. We utilized heterozygous knockout mice (Slc34a2+/-) and renal proximal tubule-specific knockout mice (Slc5aCreERT2;Slc34a2f/f) to examine the contribution of SLC34A2 to renal fibrosis. We employed HK-2 cells to elucidate molecular mechanisms how SLC34A2 mediates the progression of CKD.

Results: Data mining analysis revealed that SLC34A2 expression is relatively low in healthy kidneys but significantly elevated in various diseases types of CKD, including diabetic nephropathy, focal segmental glomerulosclerosis, lupus nephritis CKD tissues. In vitro, overexpression of SLC34A2 in HK-2 cells impaired cell proliferation, mitochondrial respiration, fatty acid β-oxidation, and ATP production, while enhancing glycolysis. Mechanistically, these metabolic alterations, potentially linked to HIF-1α activation and mitochondrial dysfunction, led to partial epithelial–mesenchymal transition (EMT), marked by decreased E-cadherin and increased α-SMA and fibronectin. Metabolomic analysis revealed increased glycolytic intermediates and decreased TCA cycle metabolites. In vivo, heterozygous (Slc34a2+/−) and proximal tubular-specific knockout (Slc5aCreERT2;Slc34a2f/f) mice exhibited significantly reduced tubulointerstitial fibrosis in UUO model. This was confirmed by Masson's trichrome staining and reduced expression of fibrotic markers such as α-SMA, fibronectin, and collagen.

Conclusion: Our findings demonstrate a causal role of SLC34A2 in regulating cellular metabolism, survival, and the progression of tubulointerstitial fibrosis.

Keywords：Chronic Kidney Disease, SLC34A2, Epithelial–Mesenchymal Transition

Poster Presentation：Chronic Kidney Disease
Poster No.：B0167
Abstract Submission No.：APCN20250784

CRISPR/Cas9-Mediated TLR4 Knockout Protects Male Mice from HFD-Induced Gut–Liver–Kidney Axis Dysfunction via Microbiota–Immune Modulation

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Abstract

Background

Obesity-induced metabolic stress contributes to progressive dysfunction in the gut, liver, and kidney. The gut–liver–kidney axis is increasingly recognized as a pathophysiological network linking gut dysbiosis, increased gut permeability, systemic inflammation, and immune dysregulation. Toll-like receptor 4 (TLR4), a microbial sensor that detects endotoxins such as lipopolysaccharide, plays a central role in mediating inflammatory cascades that drive chronic low-grade inflammation in obesity. Recent studies indicate that metabolic dysfunction-associated steatotic liver disease (MASLD), a redefined liver condition, is not only influenced by gut microbial composition and endotoxemia, but also closely associated with kidney injury. However, the mechanistic role of TLR4 in coordinating these inter-organ responses—particularly under high-fat diet (HFD)–induced metabolic stress—remains unclear. Furthermore, sex-specific differences in immune responses and organ vulnerability have been reported but are poorly understood. To address these gaps, we employed a CRISPR/Cas9-mediated TLR4 knockout (CTLR4) mouse model to investigate how TLR4 influences gut–liver–kidney axis dysfunction under HFD conditions in both sexes. We hypothesized that TLR4 deletion would mitigate systemic inflammation, improve epithelial barrier integrity, and reduce end-organ damage in a sex-dependent manner.

Methods

Male and female wild-type (WT) and CTLR4 mice were fed HFD for 16 weeks. Body weight and serum creatinine/BUN were recorded. Tissue morphology of the gut, liver, and kidney was examined using H&E and PAS staining. Immune cell infiltration ($Ly6C^+$ monocytes, $CD11b^+F4/80^+$ macrophages, $Foxp3^+$ Tregs) was analyzed by flow cytometry. Inflammatory cytokines (IL-1β, IL-6, TNF-α) were quantified via ELISA. Gut permeability was assessed using Ussing chambers and FITC-dextran flux. Gut microbiota composition was analyzed by 16S rRNA sequencing.

Results

WT males showed the most pronounced body weight gain and tissue damage. H&E/PAS staining revealed shortened intestinal villi, hepatic ballooning, and renal tubular injury. CTLR4 males exhibited improved tissue integrity, reduced body weight gain, lower creatinine/BUN, and attenuated

infiltration of proinflammatory monocytes/macrophages and Foxp3^{+} Tregs. ELISA confirmed reduced IL-1β, IL-6, and TNF-α. Gut barrier function was significantly restored in CTLR4 mice. Microbiota analysis showed enrichment of beneficial taxa in CTLR4 males; SCFA-related signatures are under further investigation. A positive correlation between body weight and renal inflammation was observed in WT males but not in CTLR4 mice.

Conclusion

CRISPR/Cas9-mediated TLR4 knockout protects against HFD-induced systemic inflammation and multi-organ injury through preservation of gut–liver–kidney axis homeostasis, particularly in male mice. These findings highlight the importance of microbial sensing pathways and sex-specific immune responses in metabolic disease progression.

Keywords：Toll-like receptor 4 (TLR4) , Gut–liver–kidney axis , High-fat diet

Poster Presentation：Chronic Kidney Disease
Poster No.：B0168
Abstract Submission No.：APCN20250801

Water Quality Matters: Linking Drinking Water Sources to Chronic Kidney Disease in Indonesian Communities

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Abstract
Chronic kidney disease resulting from glomerulonephritis is a persistent renal failure condition caused by glomerular impairment. A contributing risk factor for the manifestation of this illness is infection. Contaminants in potable water can induce renal illnesses. This study aims to investigate the impact of drinking water sources on the prevalence of chronic kidney disease resulting from glomerulonephritis.
The prevalence numbers of chronic kidney disease due to glomerulonephritis in Indonesia were sourced from the Global Burden of Disease research (GBD). The estimated characteristics utilized age-standardized prevalence with percentages adjusted from the total cases of the disease attributable to all causes. The results were assigned a 95% confidence interval. Additionally, the proportion of drinking water sources was acquired from the central statistics agency of each province in Indonesia. Sources of drinking water include bottled water, water from municipal facilities, wells, springs, and rainwater.
The average prevalence of chronic renal disease in Indonesia is 0.0028 (0.0022-0.0036 95% UIs). In Indonesia, Jambi has the greatest prevalence at 0.0035 (0.0026-0.0047 95% UIs), followed by Gorontalo at 0.0031 (0.0025-0.0040 95% UIs) and West Papua at 0.003 (0.0026-0.0041 95% UIs). Southeast Sulawesi has the lowest incidence at 0.0026 (0.0020-0.0033 95% UIs). The Pearson correlation test indicates that springs (R=0.816; p=0.048) and rainwater (R=0.972; p=0.002) significantly influence the prevalence of chronic renal disease.
Drinking water sources have a significantly influence the prevalence of chronic renal disease due to glomerulonephritis, especially water from unprotected springs and rainwater.

Keywords：Chronic Kidney disease, rainwater, springs

Poster Presentation：Chronic Kidney Disease
Poster No.：B0169
Abstract Submission No.：APCN20250824

Lifestyle-Mediated Exacerbation of Sleep Apnea Outcomes: Impacts on Insulin Resistance, Chronic Kidney Disease, and Dialysis Risk in Southeast Asia

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Abstract

Background:

Sleep apnea is a significant contributor to insulin resistance and chronic kidney disease (CKD). In Southeast Asia, lifestyle factors such as smoking and alcohol consumption may amplify these effects. Understanding how sleep apnea, lifestyle behaviors, and metabolic dysfunction interact is essential for improving health outcomes and preventing dialysis dependency.

Methods:

A systematic review and meta-analysis of 30 studies (n=14,237) from 2010–2025 was conducted. Sleep apnea severity was assessed using the Apnea-Hypopnea Index (AHI), while metabolic and renal outcomes included insulin resistance (HOMA-IR), kidney function (eGFR), and dialysis dependency. Control variables included age, BMI, smoking, alcohol consumption, and comorbidities. Subgroup analyses were conducted by age group (18–39, 40–59, ≥60). Meta-regression evaluated the impact of lifestyle factors.

Results:

The study population had a mean age of 53.1 years (±11.2), with 47.6% identifying as female. The average BMI was 27.8 (±4.1), indicating an overweight population, although 23.2% fell within the normal BMI range. Type 2 diabetes was prevalent in 41.3% of participants, and 36.8% had chronic kidney disease (CKD), with 14.7% requiring dialysis. Additionally, 29.4% of the participants reported smoking, and 18.1% reported alcohol consumption, highlighting the presence of modifiable lifestyle risk factors within this metabolically at-risk group. Severe sleep apnea was present in 23.2% of participants, with higher prevalence in older adults (31.1%). T2D and CKD prevalence were 41.3% and 36.8%, respectively. A 5-unit AHI increase was associated with a 0.21-unit rise in HOMA-IR ($p<0.001$) and a 3.8 mL/min decline in eGFR ($p<0.001$). Severe sleep apnea increased dialysis risk (OR: 2.62, $p<0.001$). Smoking (29.4%) and alcohol consumption (18.1%) worsened insulin resistance ($\beta=0.31$, $p=0.006$) and eGFR decline ($\beta=0.22$, $p=0.02$), particularly in older adults.

Conclusions:

Sleep apnea significantly worsens metabolic and renal outcomes, with lifestyle factors further amplifying these risks. Older adults and those with high smoking or alcohol consumption exhibit faster disease progression. Integrative strategies targeting sleep apnea management and lifestyle modifications are essential for reducing T2D, CKD, and dialysis burden in Southeast Asia.

Outcome	Effect Size (95% CI)	p-value
HOMA-IR (per 5-unit AHI increase)	0.21 (0.14-0.27)	<0.001
eGFR (mL/min decline per 5-unit AHI increase)	-3.8 (-5.2 to -2.4)	<0.001
Dialysis Odds Ratio (Severe vs. Mild Sleep Apnea)	2.62 (1.98-3.44)	<0.001
Smoking Prevalence (β)	0.31	0.006
Alcohol Consumption (β)	0.22	0.02

Keywords：Sleep Apnea, Insulin Resistance, Chronic Kidney Disease, Dialysis, Southeast Asia, Lifestyle Factors

Poster Presentation：Chronic Kidney Disease
Poster No.：B0170
Abstract Submission No.：APCN20250830

Integrated Genomic and Immunologic Profiling in Chronic Kidney Disease and Dialysis: Age-Stratified Insights for Novel Therapeutic Target Discovery

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Abstract
Background:
Chronic Kidney Disease (CKD) involves genomic and immune dysregulation, influencing progression and dialysis outcomes. Understanding these interactions can reveal novel therapeutic targets. This systematic review evaluates genomic and immune profiling across age groups to identify personalized treatment strategies.
Methods:
A systematic review following PRISMA guidelines analyzed studies from 2010-2025 on genomic and immune profiling in CKD. Data extraction included demographics, immune markers, genomic variations, and clinical outcomes. Primary outcomes were CKD progression (eGFR decline, proteinuria) and dialysis response (inflammation markers, survival rates). Control variables included comorbidities, BMI, and lifestyle factors. Regression and meta-analyses evaluated the predictive value of immune and genetic factors.
Results:
The study included CKD and dialysis patients across multiple cohorts, with a mean age of 52.1 years (SD = 15.4). Patients were categorized as young (<40), middle-aged (40–65), and elderly (>65), the latter being more prone to immune and dialysis-related complications. Mean eGFR was 38.4 mL/min/1.73m² (SD = 14.6), indicating declining kidney function. Elevated CRP levels (mean 6.5 mg/L, SD = 2.4) were associated with faster CKD progression and higher complication rates. Elevated IL-6 (OR: 2.38, p<0.001) and TNF-α (OR: 1.92, p=0.002) were strongly linked to CKD progression. Genetic variations in immune-related genes (SNPs) (OR: 1.81, p=0.004) correlated with faster disease progression, particularly in younger patients. Age-stratified immune dysregulation indicated inflammatory dominance in elderly patients. Deep learning models integrating genomic and immune data improved predictive accuracy.
Conclusions:
Genomic and immune profiling provides critical insights into CKD progression and dialysis response. IL-6, TNF-α, and immune-related SNPs are significant predictors. Age-specific immune dysregulation suggests tailored therapeutic approaches. Future research should integrate real-time genomic monitoring to optimize CKD management.

Keywords：Genomics, Immune Profiling, Chronic Kidney Disease, Dialysis, Inflammation, Age-Stratified Analysis, Therapeutic Targets

Summary of study populations		
Variable	**Mean (SD)**	**Range**
Age (years)	52.1 (15.4)	20-85
eGFR (mL/min/1.73 m2)	38.4 (14.6)	10-80
Inflammatory markers (CRP)	6.5 (2.4)	2-15
Predictors of CKD progression		
Variable	**Odds Ratio (95% CI)**	**p-value**
IL-6 levels	2.38 (1.72-3.22)	<0.001
TNF-α levels	1.92 (1.44-2.75)	0.002
SNPs in immune genes	1.81 (1.33-2.41)	0.004

Poster Presentation：Chronic Kidney Disease
Poster No.：B0171
Abstract Submission No.：APCN20250835

AI-Driven Diagnostics and Monitoring Systems for Chronic Kidney Disease and Dialysis

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Abstract

Background:

Chronic Kidney Disease (CKD) is a major global health concern, often progressing to dialysis or transplantation. AI-driven diagnostics and monitoring systems offer promising advancements in early detection and disease management. This study systematically reviews AI models' effectiveness in diagnosing and monitoring CKD and dialysis patients.

Methods:

A systematic review was conducted following PRISMA guidelines, analyzing peer-reviewed studies from 2010-2025. Data were extracted on demographics, clinical variables, AI model types, and performance metrics. Primary outcomes included CKD progression (measured by eGFR decline) and dialysis efficiency (Kt/V, urea reduction ratio). Control variables included age, BMI, comorbidities, and lifestyle factors. AI models' performance was assessed using AUC-ROC, accuracy, and sensitivity analyses. Logistic regression identified predictors of CKD progression.

Results:

Data showed a mean patient age of 58.3 years and eGFR of 45.2 mL/min/1.73 m², spanning CKD stages 2 to 4. AI models achieved an average accuracy of 89.4%, highlighting their potential for reliable CKD diagnosis and monitoring across age groups and disease severity. AI models demonstrated high diagnostic accuracy (mean: 89.4%), with Deep Learning (AUC=0.92) outperforming traditional Machine Learning (AUC=0.86). Regression analysis identified diabetes (OR: 2.45, $p<0.001$), hypertension (OR: 1.98, $p=0.002$), and low albumin (OR: 2.21, $p<0.001$) as significant predictors. AI models incorporating real-time monitoring improved risk stratification. Figures include AUC-ROC comparisons and CKD progression probabilities.

Conclusions:

AI-driven diagnostics and monitoring systems offer high accuracy in predicting CKD progression and optimizing dialysis treatment. Deep Learning models outperform traditional approaches. Key predictors include diabetes, hypertension, and albumin levels. Integrating real-time data enhances predictive accuracy. Future research should explore AI-based personalized interventions to improve CKD patient outcomes.

Keywords：AI Diagnostics, Chronic Kidney Disease, Dialysis, Machine Learning, Deep Learning

Poster Presentation：Chronic Kidney Disease
Poster No.：B0172
Abstract Submission No.：APCN20250858

miR-30a-3p modulates epithelial-mesenchymal transition and renal fibrosis through downregulating CNPY2

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Abstract

Chronic kidney disease (CKD) leads to the shortening of life and huge burdens of medical care. Unfolded protein response (UPR) plays important roles in the acute kidney injury (AKI) to CKD transition. To understand the downstream signaling of UPR in kidney disease, we analyzed microRNA profiles from the public datasets and found miR-30a-3p was downregulated both in Xbp1-depleted cells and ischemic AKI. In vivo, miR-30a-3p was downregulated along with the progression of renal fibrosis in murine model. In vitro, miR-30a-3p alleviated the expression of TGF-beta1-induced profibrotic factors. Overexpression miR-30a-3p inhibited CNPY2 expression, which was significantly elevated in the murine model and CKD patients. CNPY2 level showed negative correlation with the expression of miR-30a-3p. Mechanistically, CNPY2 activated PERK-CHOP pathway and induced profibrotic and epithelial-to-mesenchymal transition (EMT) factors. CNPY2 facilitated TGF-betaR1 expression on the cell membrane. Our study revealed the role of miR-30a-3p/CNPY2 axis in renal fibrosis through modulating the expression of EMT and profibrotic factors, which may provide a new insight for developing therapeutic strategy for renal disease progression.

Keywords：miR-30a-3p, CNPY2, EMT, renal fibrosis

Poster Presentation：Chronic Kidney Disease
Poster No.：B0173
Abstract Submission No.：APCN20250859

DDX17-Mediated Regulation of Epithelial–Mesenchymal Transition in Renal Fibrosis

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Abstract

DEAD box RNA helicase 17 (DDX17) induced epithelial-to-mesenchymal transition (EMT) in cancers and cell differentiation, but the association of DDX17 and EMT in renal fibrosis remained unclear. Using transforming growth factor β (TGF-β)-treated HK-2 cells to induce EMT, we investigated the effects of DDX17 on markers of EMT and renal fibrosis. The effects of miR-30a-3p on expression of DDX17 were studied and the interaction between them was validated by luciferase assay. We found that TGF- induced EMT with DDX17 upregulated in HK-2 cells. Silencing DDX17 diminished the TGF-β-induced changes of markers for EMT in HK-2 cells, whereas overexpressing DDX17 enhanced EMT. We revealed that miR-30a-3p attenuated TGF-β-induced EMT through DDX17. In a unilateral ureteral obstruction mouse model mimicking renal fibrosis, DDX17 expression increased and miR-30a-3p decreased. In conclusion, we found that DDX17, which can be downregulated by miR-30a-3p, promotes EMT and renal fibrosis.

Keywords：DEAD box helicase 17, miR-30a-3p, epithelial-to-mesenchymal transition, renal fibrosis

Poster Presentation：Chronic Kidney Disease
Poster No.：B0174
Abstract Submission No.：APCN20250863

Omega-3 Fatty Acids Attenuate Myostatin Expression and Mitochondrial Dysfunction in Skeletal Muscle and C2C12 Cells Under Uremic Condition

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Abstract

Introduction: Chronic kidney disease (CKD) is associated with sarcopenia, characterized by the progressive loss of skeletal muscle mass and function. Myostatin, a key negative regulator of muscle growth, is known to be upregulated in CKD. Mitochondria play a vital role in energy production in skeletal muscle and mitochondrial dysfunction plays a central role in muscle wasting. Omega-3 fatty acids (FAs), known for their anti-inflammatory properties, may attenuate CKD-induced sarcopenia. This study aimed to evaluate whether omega-3 FAs modulate myostatin expression and mitochondrial dysfunction mediators under uremic conditions.

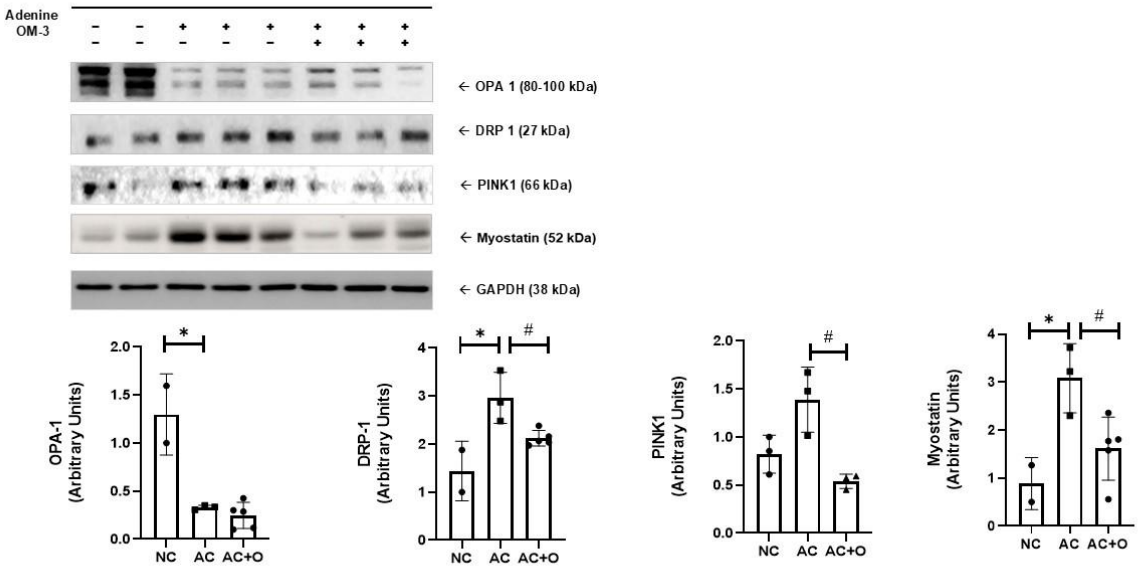
Methods: Male Sprague-Dawley rats were fed a diet containing 0.75% adenine and 2.5% protein for three weeks to induce CKD. Rats were then randomly assigned to two groups and treated with or without omega-3 FAs (300 mg/kg/day) for two weeks. All rats received cholecalciferol (3,000 IU/kg/week) throughout the 5-week period. In vitro, C2C12 myoblasts were exposed to 1 mM indoxyl sulfate (IS), with or without omega-3 FA co-treatment, for 24 hours. Western blotting was used to analyze the expression of myostatin and markers of mitochondrial biogenesis (PGC-1α, Nrf2), dynamics (OPA1, Drp1, PINK1), and muscular atrophy (MuRF1, FOXO1). Mitochondrial DNA (mtDNA) content was quantified using real-time PCR. Groups included: normal controls (n=3 at 3 weeks, n=4 at 5 weeks), adenine controls (n=6 at 3 weeks; n=5 at 5 weeks), and omega-3 FA–treated rats (n=5 at 5 weeks).

Results: Adenine-fed rats showed significantly elevated serum creatinine levels, which were partially ameliorated by omega-3 FA supplementation. Mitochondrial markers (PGC-1α, Nrf2, OPA1, and mtDNA) were downregulated in CKD rats but partially recovered with omega-3 FA treatment. Myostatin, Drp1, and PINK1 were elevated in the adenine control group and suppressed following omega-3 FA treatment. In C2C12 cells, IS exposure led to decreased mtDNA and increased expression of myostatin, Drp1, MuRF1, and FOXO1—all of which were improved with omega-3 FA treatment.

Conclusion: Omega-3 fatty acids suppressed myostatin expression and mitigated mitochondrial dysfunction in both in vivo and in vitro models of uremia. These findings suggest that omega-3 FA supplementation may serve as a potential therapeutic strategy to preserve skeletal muscle health in CKD.

Keywords：omega-3 fatty acid, myostatin, mitochondrial dysfunction, uremia

Adenine RAT (MUSCLE) at 5 weeks



Poster Presentation：Chronic Kidney Disease
Poster No.：B0175
Abstract Submission No.：APCN20250869

Monocyte/macrophage infiltration contributes to renal inflammation and fibrosis in breast cancer mice

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Abstract

Background: While the relationship between malignancy and kidney injury is well-recognised, renal complications in cancer patients are often overlooked due to the frequently subtle presentation of renal syndromes. In patients with both malignancy and nephropathy, kidney involvement may become apparent, yet tumour remission is typically accompanied by a marked reduction in proteinuria and nephritis. This observation suggests a potential association with paraneoplastic nephropathy.

Methods: To evaluate potential renal complications in breast cancer mouse models, 4T1 mammary carcinoma cells were orthotopically implanted into the mammary fat pad of BALB/c mice. Two weeks after implantation, kidneys from these mice were collected, and renal biopsies were subjected to histochemical analysis. An increased number of cells were observed in the peritubular area of the kidneys in breast cancer mice. Immunohistochemical examination showed the presence of F4/80-positive and CD11b-positive macrophages as well as IgA in the peritubular region. To further assess the role of macrophages in kidney disease within this model, monocytes or tumor antigen-stimulated monocytes were introduced into both healthy mice and mice with breast cancer.

Results: Breast cancer mice showed large macrophage presence and IgA deposits in the kidneys. Macrophage infiltration correlated with renal fibrosis and inflammation. Tail vein introduction of monocytes confirmed their role in these processes. Introducing tumor antigens to breast cancer mice further worsened renal fibrosis by stimulating monocytes.

Conclusion: When tumor grew in mammary fat pad, mammary carcinoma cells remotely deposits IgA, and increased number of macrophage s in the tubulointerstitial area mibht be the pathological causes for renal inflammation and fibrosis.

Keywords：Renal inflammation, BCSeP, macrophage, gp80, IL-6

Poster Presentation：Chronic Kidney Disease
Poster No.：B0176
Abstract Submission No.：APCN20250870

Chronic Hypoxia Reprograms Metabolism and Aggravates Renal Injury in CKD: Multi-Omics Insights from Plateau Populations

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Abstract

Background and Aims:

Chronic kidney disease (CKD) progression is influenced by metabolic and environmental factors. Chronic hypoxia in high-altitude regions may perturb metabolic homeostasis, potentially exacerbating CKD. This study capitalizes on plateau inhabitants as a singular natural experimental model. This approach grants an exceptional and irreplicable viewpoint for deciphering the central involvement of environmental hypoxia in CKD metabolic dysregulation, a fundamental understanding unattainable through low-altitude research alone. This study aims to elucidate the metabolic and proteomic signatures distinguishing plateau and plain CKD patients, and to uncover hypoxia-driven mechanisms of renal deterioration.

Methods:

Serum from age-/sex-matched CKD stage 2-3 patients (18-60 yrs) and healthy controls in plain (Shenzhen, 81m) and plateau (Xining, 2261m) regions underwent non-targeted LC-MS/MS metabolomics and DIA proteomics. Oxidative markers (ROS, SOD, GSH/GSSG) were enzymatically assayed. Male SD rats (8-weeks-old) received adenine (200mg/kg/d ×21d) or saline at Beijing (4m; n=4 control/6 CKD) and Xining (2261m; n=4 control/6 CKD). Renal function (protein/creatinine, serum creatinine) and fibrosis (Masson) were assessed. Statistics: ANOVA (4-group), t-test (altitude), Pearson correlation (R/GraphPad). Approved by Qinghai University Ethics Committee.

Results:

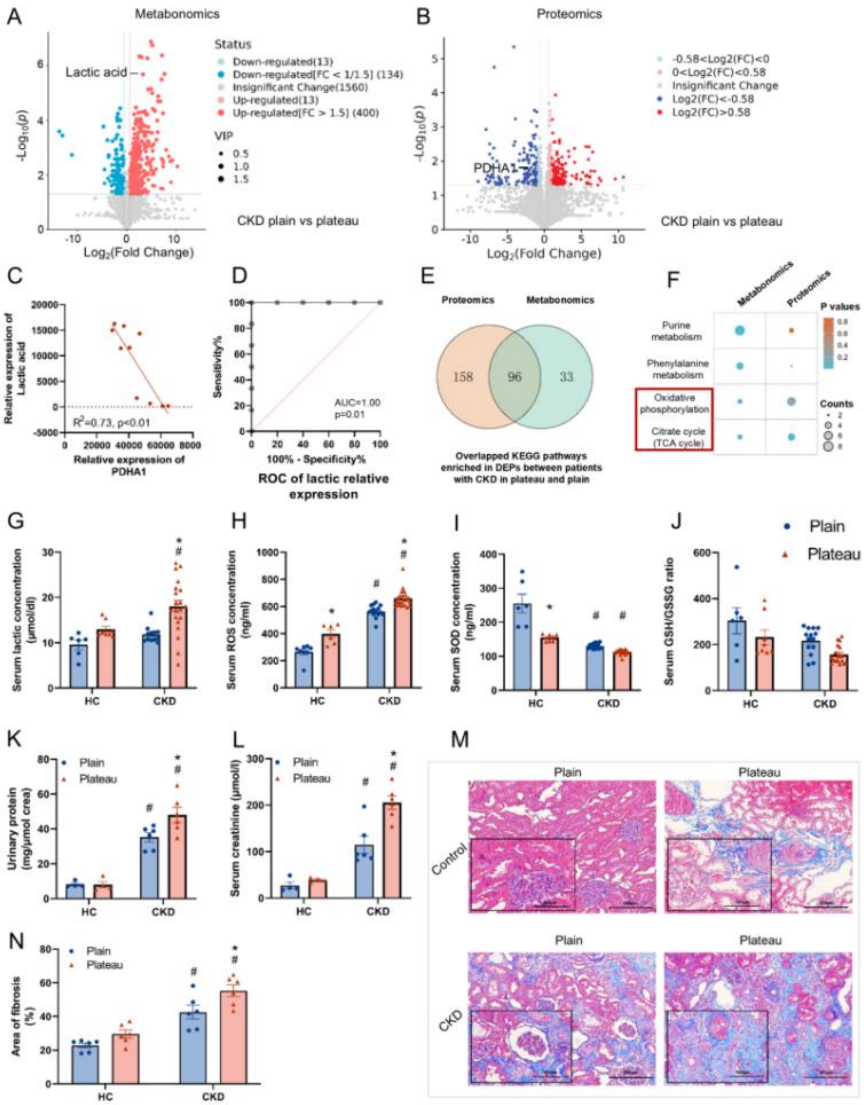
Metabolomics revealed significant lactate upregulation in plateau CKD patients versus plain counterparts (volcano plot, $P<0.05$), while proteomics identified downregulation of PDHA1, a key TCA cycle regulator (volcano plot, $P<0.01$). A negative correlation between lactate and PDHA1 ($r2=0.73$, $P<0.01$) suggested hypoxia-induced suppression of aerobic metabolism. ROC analysis confirmed lactate's diagnostic value for distinguishing CKD patients across regions (AUC=1.00), but not in HCs. Multi-omics integration revealed 96 co-enriched pathways, with TCA cycle and oxidative phosphorylation. Plateau CKD patients exhibited elevated ROS, and reduced levles of SOD as well as GSH/GSSG ratio. In rat models, plateau CKD rats showed exacerbated renal dysfunction (elevated urinary protein/creatinine and serum creatinine, vs. plain CKD rats, $P<0.01$) and intensified fibrosis.

Conclusions:

Chronic hypoxia in plateau regions drives CKD progression through metabolic reprogramming—characterized by PDHA1-mediated TCA cycle suppression, glycolytic activation (lactate accumulation), and oxidative stress amplification. Serum lactate serves as a robust biomarker for altitude-associated CKD severity. The exacerbated renal injury and fibrosis observed in our plateau-based adenine-induced CKD rat model directly corroborate the detrimental impact of the hypoxic

environment, strengthening the causal inference derived from human multi-omics data. Collectively, our results provide a crucial theoretical foundation for implementing precision prevention strategies, enabling earlier diagnosis, and developing novel therapeutic interventions targeting hypoxia-responsive and metabolic pathways in CKD patients residing in high-altitude regions.

Keywords：CKD;PDHA1;TCA;CKD；



A-B. The volcano plots illustrated the differentially expressed metabolites (DEMs) **(A)** and proteins (DEPs) **(B)** identified through metabolomics and proteomics in the sera of patients with CKD from the plateau and plain regions. Upregulated metabolites and proteins were presented in red, while downregulated ones were shown in blue. Among the DEMs, lactate was significantly upregulated in plateau patients with CKD **(A)**. Regarding the DEPs, PDHA1, which encodes pyruvate dehydrogenase E1 subunit alpha 1 (a key enzyme regulating the tricarboxylic acid (TCA) cycle), was significantly downregulated in plateau patients with CKD **(B)**.

C. A significant negative correlation was observed between the relative expression levels of lactate and PDHA1 in the sera of patients with CKD from both the plateau and plain areas. This finding implies that there might be a regulatory link between the downregulated expression of PDHA1 and the upregulated expression of lactate.

D. The receiver operating characteristic (ROC) curves were utilized to evaluate the diagnostic potential of lactic acid in different groups.

E. The Venn diagram revealed that the differential metabolites and proteins identified by both omics approaches were jointly enriched in 96 signaling pathways.

F. Notably, among the differential substances identified by both omics techniques, the TCA cycle was significantly enriched. This suggests that, in contrast to plain patients with CKD, highland patients with CKD may experience inhibited TCA cycle activity due to hypoxia. Such inhibition potentially leads to abnormal energy metabolism and oxidative stress, which might exacerbate the disease progression in patients with CKD.

G–J. Oxidative stress levels among groups were evaluated by the levels of reactive oxygen species (ROS), superoxide dismutase (SOD), and the ratio of reduced glutathione (GSH) to oxidized glutathione (GSSG), as well as levels of lactate in the serum of plain and highland healthy controls (HC) and patients with CKD. The results demonstrated that serum lactate levels were significantly elevated in highland patients with CKD compared to plain CKD patients **(G)**, which was consistent with the metabolomics results. Compared to plain CKD patients, highland CKD patients showed a significantly increase in serum ROS **(H)** and decrease in serum SOD **(I)** levels, while the GSH/GSSG ratio exhibited a downward trend **(J)**. "#" denoted a significant difference between CKD patients and HC, and "*" denoted a significant difference between the plateau and plain groups.

K–L. The CKD in-vivo models were established in SD rats by intragastrically administering adenine for 21 days in the plain area and the plateau area, respectively. On the 28th day after the conclusion of intragastric administration, the urinary protein/creatinine ratio and serum creatinine level of the rats were measured. The results showed that, compared with the healthy control group, all CKD rats had an increased urinary protein/creatinine ratio **(K)** and serum creatinine level **(L)**. Moreover, CKD rats in the plateau group had higher urinary protein/creatinine ratio and serum creatinine level than those in the plain group.

M-N. The Masson staining results indicated that, when compared with CKD rats in the plain area, the blue - stained area in the renal tissues of CKD rats on the plateau occupied a larger proportion within the field of view, signifying a more severe degree of fibrosis.

Poster Presentation：Chronic Kidney Disease
Poster No.：B0177
Abstract Submission No.：APCN20250902

Repurposing Manidipine as an MMP7 Inhibitor for Renal Protection in Chronic Kidney Disease

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Introduction: Matrix metalloproteinases (MMPs) are essential proteolytic enzymes involved in the remodeling and degradation of the extracellular matrix (ECM). Among them, MMP7 has been identified as a key contributor to the development and progression of kidney diseases, demonstrating a more significant pathological role than other MMPs. This study aimed to identify a potential bioactive compound targeting MMP7 and to elucidate its molecular effects in the context of chronic kidney disease (CKD), using both in vitro and in vivo models.

Methods: A virtual screening of 11,586 compounds from the DrugBank database was conducted to identify candidates with high binding affinity to the MMP7 protein. Top hits were evaluated for biological activity using HK-2 human proximal tubular epithelial cells. Key parameters assessed included cytotoxicity, autophagy, inflammasome activation, and pro-fibrotic responses. The most promising compound was further validated in a mouse model of adenine-induced CKD.

Results: Ten top candidate compounds were shortlisted, with docking analysis indicating that manidipine had a higher binding affinity to the MMP7 receptor than a known MMP7 inhibitor. Molecular modeling revealed that manidipine exhibits two favorable ionic pharmacophore features contributing to its interaction with MMP7. Functionally, manidipine enhanced autophagic activity and inhibited the activation of NLRP3 and NLRP6 inflammasomes in vitro. In vivo, manidipine treatment significantly reduced the expression of pro-fibrotic markers, improved renal function, and attenuated structural kidney damage in CKD mice. These renoprotective effects were associated with the modulation of autophagy, inflammasome signaling, and fibrosis pathways in renal tissues.

Conclusion: This study is the first to identify manidipine as a potential MMP7-targeting agent with a novel mechanism of action in CKD. By promoting autophagy and suppressing inflammasome activation and fibrotic signaling, manidipine offers therapeutic promise in slowing CKD progression. These findings highlight a previously unrecognized renoprotective role of manidipine, supporting its potential repositioning as a treatment for chronic kidney disease.

Keywords：MMP7, Chronic Kidney Disease, Manidipine, Autophagy, Inflammasome, Drug Repurposing

Poster Presentation：Chronic Kidney Disease
Poster No.：B0178
Abstract Submission No.：APCN20250903

Lipocalin-2 Protects Against Chronic Kidney Disease-Associated Vascular Calcification By Promoting Fatty Acid Oxidation Via Interaction With Very Long Chain Acyl Coenzyme A Dehydrogenase

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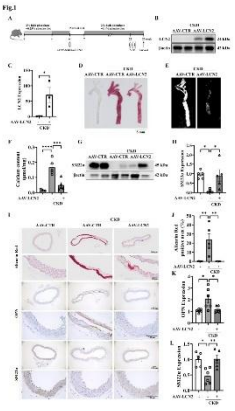
Abstract

Introduction: Vascular calcification (VC) is a critical risk factor for cardiovascular events and mortality in chronic kidney disease (CKD) patients. Osteogenic transdifferentiation of vascular smooth muscle cells (VSMCs) drives CKD-associated VC, yet underlying mechanisms remain incompletely understood. Lipocalin-2 (LCN2), a multifunctional secreted protein implicated in various diseases, has an undefined role in VC. This study aims to elucidate the role and mechanism of LCN2 in CKD-associated VC.

Methods: LCN2 expression was assessed in radial arteries from CKD patients and rat VSMCs. Calcification was induced in rat VSMCs and human/rat arterial rings using high-phosphate medium, and in mice using an adenine/phosphate diet plus low-dose Vitamin D3 administration. LCN2 levels were modulated using recombinant protein, small interfering RNA, plasmid overexpression, and adeno-associated virus. Osteogenic markers (Bone Morphogenetic Protein 2, Osteopontin) and contractile markers (Smooth Muscle Protein 22-Alpha) were analyzed via western blotting, immunofluorescence, and immunohistochemistry. Calcification was evaluated by Alizarin Red S staining, micro-computed tomography, and calcium content assays. Mechanisms were investigated using transcriptomics, proteomics, and co-immunoprecipitation-mass spectrometry. Lipid metabolism was assessed with BODIPY and Oil Red O staining.

Results: LCN2 expression was significantly reduced in radial arteries from CKD patients with VC and in high-phosphate-treated rat VSMCs. Recombinant LCN2 attenuated high-phosphate-induced calcification and osteogenic differentiation in VSMCs and aortic rings, while LCN2 knockdown exacerbated calcification. In CKD mice, adeno-associated virus-mediated LCN2 overexpression alleviated aortic calcification. Transcriptomic and proteomic analyses revealed activation of the fatty acid β-oxidation (FAO) pathway by LCN2. FAO inhibition via etomoxir exacerbated calcification in vitro. Co-immunoprecipitation (IP)-mass spectrometry identified an interaction between LCN2 and very long-chain acyl-CoA dehydrogenase (Acadvl), which was confirmed by Co-IP experiment. Acadvl knockdown exacerbated calcification and abolished the protective effect of LCN2 in VC progression.

Conclusion: LCN2 interacts with Acadvl and then protects against CKD-associated VC by promoting FAO. These findings establish the LCN2-Acadvl-FAO axis as a key regulator of VSMC phenotype, identifying potential therapeutic targets for VC in CKD.



Keywords：LCN2, Vascular Calcification, fatty acid β oxidation, Acadvl, vascular smooth muscle cells

Poster Presentation：Chronic Kidney Disease
Poster No.：B0179
Abstract Submission No.：APCN20250924

Altered Short-Chain Fatty Acid Profiles in Chronic Kidney Disease and Their Microbiota Correlates: A Cross-Sectional Study

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Abstract

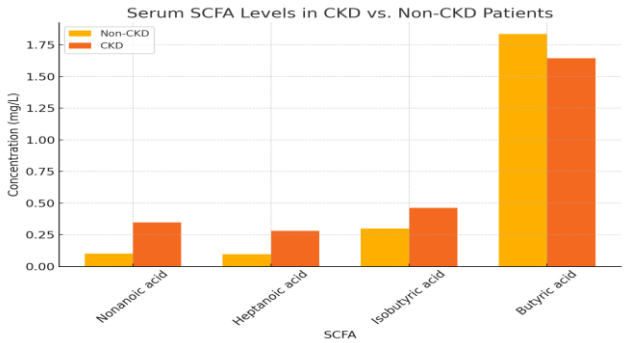
Introduction: Chronic kidney disease (CKD) is closely linked to gut dysbiosis, with changes in short-chain fatty acid (SCFA) levels implicated in disease progression. However, detailed SCFA profiling and microbial correlations in Asian CKD populations remain limited.

Methods: In this cross-sectional study, 100 adult participants (80 CKD and 20 non-CKD controls) were enrolled. Serum SCFA levels—including acetic acid, propionic acid, isobutyric acid, valeric acid, and caproic acid—were quantified via gas chromatography-mass spectrometry. Gut microbiota composition was assessed using 16S rRNA sequencing. Correlation analyses were performed between individual SCFA concentrations and specific bacterial taxa, metabolic markers, and CKD status.

Results: The mean age of population was 64.15 years old, 46 (46.0%) were man and 47 (47.0%) had T2DM. The mean eGFR was 59.68mL/min/1.73 m2. From 11 SCFAs, the serum levels of Isobutyric acid (0.4629 mg/L vs. 0.2994 mg/L, p= 0.015), Heptanoic acid (0.2807 mg/L vs. 0.0967 mg/L, p= 0.004), and Nonanoic acid (0.3475 mg/L vs. 0.0994 mg/L, p= 0.014) were higher in CKD patients compared to normal controls, respectively. However, the levels of Butyric acid (1.6444 mg/L vs. 1.8342 mg/L, p=0.250) were lower in CKD patients than controls. Pearson correlation analysis revealed significant associations between SCFA levels and renal function index for Isobutyric acid (positive correlated with blood urea nitrogen: r=0.47, p<0.001; serum creatinine: r=0.39, p<0.001 and uACR: r=0.45, p<0.001), for Heptanoic acid (positive correlated with blood urea nitrogen: r=0.38, p<0.001; serum creatinine: r=0.36, p<0.001 and uACR: r=0.41, p<0.001) and Nonanoic acid (positive correlated with blood urea nitrogen: r=0.44, p<0.001; serum creatinine: r=0.34, p<0.001 and uACR: r=0.41, p<0.001), respectively. The relative abundances of genus Alistipes, particularly Alistipes indistinctus, Alistipes obesi, Alistipes putredinis, Alistipes sp. An31A showed significant positive correlations with the serum concentrations of these four SCFA.

Conclusions: CKD patients exhibited altered SCFA profiles and significant microbiota–metabolite correlations, particularly involving Alistipes species. These findings highlight the gut–kidney–metabolite axis as a potential therapeutic target in CKD and support further longitudinal and interventional studies in Asian populations.

Keywords：Chronic kidney disease, short-chain fatty acid, gut microbiota



Poster Presentation：Chronic Kidney Disease
Poster No.：B0180
Abstract Submission No.：APCN20250968

Tri-n-butyl phosphate Induces Vacuolization of Renal Proximal Tubular Cells via Aryl Hydrocarbon Receptor Signaling

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Abstract
Introduction:
Tri-n-butyl phosphate (TnBP) is an organophosphorus compound extensively utilized as a solvent and plasticizer in various industrial applications, including the production of plastics, resins, and flame retardants. Despite its widespread use, accumulating evidence suggests that TnBP may pose substantial health risks, particularly to renal function. However, experimental data specifically addressing TnBP-induced nephrotoxicity remain limited. This study was conducted to investigate the cytotoxic effects of TnBP on renal tubular cells.

Methods:
Human proximal tubular epithelial cells (HK-2) were employed as an in vitro model to assess the cytotoxic effects of TnBP. The cells were divided into three experimental groups: control, TnBP (10 ng/mL), and TnBP combined with MG-132 (10 μM). The selected concentration of TnBP corresponds to levels relevant to environmental exposure. Vacuolization of HK-2 cells was evaluated using bright-field microscopy. Western blot analysis was performed to examine changes in the expression of aryl hydrocarbon receptor (AhR) proteins.

Results:
Bright-field microscopy revealed that exposure to TnBP at a concentration as low as 10 ng/mL induced vacuolization in HK-2 cells. Additionally, TnBP treatment led to a downregulation of AhR expression. Co-treatment with MG-132, a known inhibitor of proteasomal degradation of AhR, effectively reversed the TnBP-induced reduction in AhR levels and attenuated vacuolization in HK-2 cells.

Conclusion:
Our findings suggest that low-level environmental exposure to TnBP induces vacuolization in renal tubular cells through disruption of AhR signaling. This cellular response implicates TnBP as a potential environmental nephrotoxin and supports its possible contribution to the development of chronic kidney disease of uncertain etiology (CKDu).

Keywords：TnBP, AhR, Renal toxicity

Poster Presentation：Chronic Kidney Disease
Poster No.：B0181
Abstract Submission No.：APCN20250978

Ethyl Acetate Fractions of Salvia miltiorrhiza Bunge (Danshen) Crude Extract Modulate Inflammatory and Fibrotic Signals to Ameliorate Diabetic Kidney Injury

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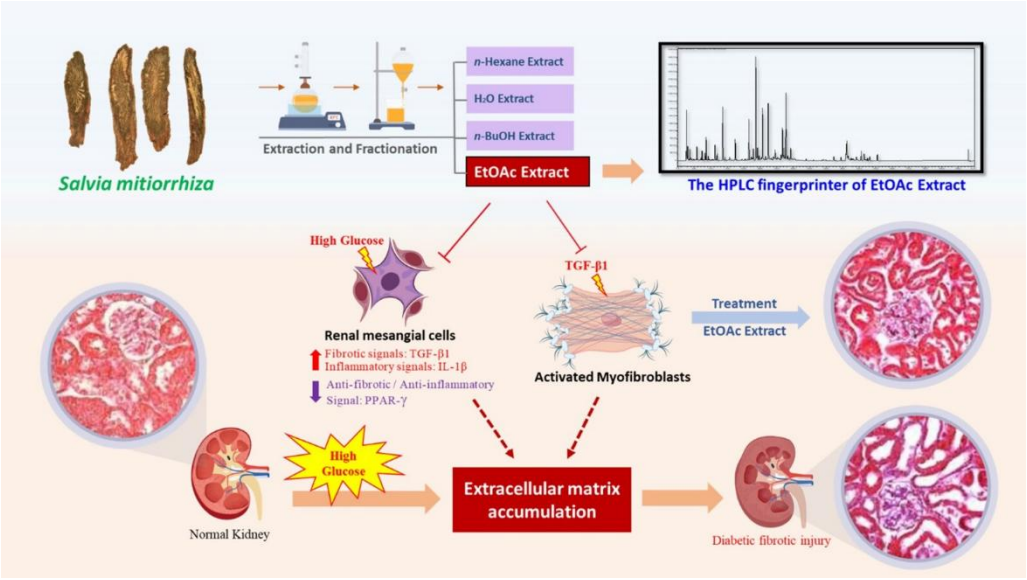
Abstract

Diabetic nephropathy, a major cause of end-stage renal disease, contributes substantially to both morbidity and mortality. It is characterized by glomerular microinflammation and myofibroblast activation within the tubulointerstitium. Salvia miltiorrhiza Bunge, a traditional Chinese medicinal herb, has demonstrated anti-inflammatory and anti-fibrotic properties, suggesting potential renoprotective effects. This study aims to identify which specific component of S. miltiorrhiza can attenuate diabetic nephropathy-induced renal damage in a controlled setting. Among the extracted fractions, the ethyl acetate (EtOAc) layer was shown to activate peroxisome proliferator-activated receptor (PPAR)-α and PPAR-γ in renal mesangial cells, as evidenced by a dual-luciferase reporter assay. In mesangial cells cultured under high-glucose (HG) conditions, the EtOAc fraction significantly suppressed HG-induced upregulation of interleukin-1β, transforming growth factor-β1 (TGF-β1), and fibronectin, while restoring the expression of downregulated PPAR-γ. Furthermore, among the various S. miltiorrhiza extracts, the EtOAc fraction most effectively inhibited TGF-β1-induced myofibroblast activation. In vivo, the EtOAc layer also demonstrated strong efficacy in reducing renal hypertrophy, proteinuria, and fibrotic progression in a streptozotocin (STZ)-induced diabetic mouse model, primarily by suppressing diabetes-related proinflammatory mediators, extracellular matrix accumulation, and PPAR-γ downregulation. Collectively, these in vitro and in vivo findings highlight the therapeutic potential of the EtOAc fraction of S. miltiorrhiza for the treatment of diabetic nephropathy.

Keywords：Salvia miltiorrhiza; ethyl acetate layer; diabetic nephropathy; mesangial cells; myofibroblast activation; renal fibrosis


Salvia mitiorrhiza
Extraction and Fractionation
n-Hexane Extract
H2O Extract
n-BuOH Extract
EtOAc Extract
The HPLC fingerprinter of EtOAc Extract
High Glucose
Renal mesangial cells
Fibrotic signals: TGF-β1
Inflammatory signals: IL-1β
Anti-fibrotic / Anti-inflammatory
Signal: PPAR-γ
TGF-β1
Activated Myofibroblasts
Treatment
EtOAc Extract
Normal Kidney
High Glucose
Extracellular matrix accumulation
Diabetic fibrotic injury

Poster Presentation：Chronic Kidney Disease
Poster No.：B0182
Abstract Submission No.：APCN20250982

Awareness of Dialysis Costs Among Patients with Chronic Kidney Disease: A Cross-Sectional Survey

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Abstract
Background:
Patients with chronic kidney disease (CKD) often face challenges in understanding the potential costs of dialysis treatment. This understanding is crucial for enhancing patient education and facilitating informed decision-making.
Methods:
A cross-sectional survey was conducted among patients with CKD stages 3 to 5, followed up at Singapore General Hospital. This survey aimed to evaluate CKD patients' understanding of the costs associated with kidney replacement therapies, particularly dialysis.
Results:
A total of 89 CKD patients responded to the survey. The cohort was 44% male, 75% Chinese, and 35% aged ≥65, with 65% married. 42% had tertiary education. Employment varied: 54% were working full-time, and 28% retired. Most (36%) reported a monthly per-person income between $2,000–$5,000. Regarding housing, 70% lived in HDB flats, 52% were covered by public insurance, and 43% had private insurance. Notably, 88% had not received counselling from a renal coordinator or social worker about dialysis costs. When asked about kidney replacement therapy costs, 62% of patients reported limited knowledge, and 33% were somewhat aware but not entirely sure. Additionally, 74% did not know their out-of-pocket expenses for dialysis. About 45% said cost information would greatly influence their treatment decisions, while 35% said it would somewhat influence them. Around 46% considered cost differences between dialysis modalities extremely important for decision-making, and another 46% said it was important but not the only factor. Over half reported that out-of-pocket cost was one of several key factors in choosing a dialysis modality. Similarly, 53% believed knowing their out-of-pocket costs would significantly reduce their anxiety about dialysis.
Conclusion:
These findings highlight the need for better patient education on the financial aspects of dialysis. Providing clear and accessible cost-related information could empower patients to make informed decisions regarding their treatment options.

Keywords：CKD

Poster Presentation：Chronic Kidney Disease
Poster No.：B0183
Abstract Submission No.：APCN20250995

Combined Effects of Evolocumab and Atorvastatin on Renal and Cardiovascular Function in Adenine-Induced CKD: Modulation of PCSK9 and Statin Pathways

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Abstract
Introduction
Non-dialysis chronic kidney disease (ND-CKD) is a progressive disorder marked by declining renal function before end-stage renal disease (ESRD), commonly driven by diabetes, hypertension, cardiovascular disease, obesity, nephrotoxins, and obstructive uropathy. PCSK9 inhibitors and statins are vital in mitigating cardiovascular risks, especially in diabetic patients. This study explores the combined therapeutic impact of evolocumab (PCSK9 inhibitor) and atorvastatin (statin) in an adenine-induced ND-CKD rat model, focusing on their synergistic effects in modulating PCSK9 expression, enhancing statin activity, and improving both renal function and cardiovascular outcomes.
Methods
A non-dialysis chronic kidney disease (ND-CKD) rat model was induced using adenine (60 mg/kg) and a high-fat diet. Treatment with evolocumab (10 mg/kg/week) and atorvastatin (20 mg/kg/day) showed significant renoprotective and cardiometabolic effects. Key assessments included PCSK9 expression, lipid profile, HOMA-IR, HOMA-S, and oxidative stress markers (AMPK, PPARα, SOD, CAT, GSH). Inflammatory markers (IL-6, TNF-α, adiponectin) and renal function indicators (serum creatinine, cystatin C, BUN, albumin-to-creatinine ratio, γ-GT) were measured. Histological analysis confirmed improved renal structure and function, highlighting the therapeutic potential of the combination treatment
Result
Combination therapy with evolocumab and atorvastatin significantly reduced PCSK9 expression by $43.4 \pm 3.2\%$ and inhibited HMG-CoA reductase activity ($P < 0.05$), leading to improved lipid metabolism, including a 36% drop in triglycerides, 22% reduction in VLDL, and increased HDL levels. The treatment also lowered inflammatory markers (IL-6, TNF-α) and enhanced antioxidant defense. Renal function was preserved, as shown by decreased serum creatinine, cystatin C, BUN, and albumin-to-creatinine ratio. These findings highlight the combination's strong nephroprotective and cardiometabolic effects, supporting its potential as a therapeutic strategy in ND-CKD.
Conclusion
The synergistic combination of PCSK9 inhibition and statin therapy significantly improved renal and cardiovascular function in ND-CKD, primarily through anti-inflammatory actions, improved lipid metabolism, enhanced antioxidant defense, and increased insulin sensitivity. These findings underscore the therapeutic potential of this strategy for translational and clinical application.

Keywords：Keywords:- ND-CKD, ESRD, PCSK9 inhibitor, statin, oxidative stress markers

Poster Presentation：Chronic Kidney Disease
Poster No.：B0184
Abstract Submission No.：APCN20251004

Prevalence and risk factors of anxiety and depression among chronic kidney disease patients initiated with hemodialysis or peritoneal dialysis in a tertiary hospital

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Abstract
Background: Chronic kidney disease (CKD) is the seventh leading cause of death in the Philippines, with a prevalence of 35.94%, significantly higher than the global average of 9.1%–13.4%. Patients with CKD are at a four times higher risk for anxiety and depression compared to the general population, with the transition to dialysis (hemodialysis or peritoneal dialysis) exacerbating these mental health challenges. While CKD is known to be associated with anxiety and depression, there is a lack of local studies in the Philippines investigating how the dialysis procedure itself impacts these conditions. The overlapping symptoms of uremia and depression complicate the assessment of mental health in CKD patients. Further research is needed to evaluate the prevalence of anxiety and depression during dialysis initiation and after a two-week follow-up, using the Hospital Anxiety and Depression Scale (HADS) to guide psychiatric referrals and improve holistic care.

Objectives: The general objective of this study was to determine prevalence and risk factors of anxiety and depression of admitted CKD patients for initiation of hemodialysis or peritoneal dialysis using the validated English, Filipino and Ilokano version of HADS questionnaire.

Methods: This was a cross-sectional study. It included all CKD patients initiated with peritoneal dialysis or hemodialysis in a government tertiary hospital from April 13, 2025 – May 12, 2025 using primary data collection.

Results: Anxiety and depression were more prevalent among females, patients aged 45–64, those with lower education, limited social support, and comorbid hypertension and diabetes. Although associations were not statistically significant, trends indicated higher psychological burden in recently diagnosed patients and those living alone. HADS scores before and two weeks after dialysis showed no significant changes, suggesting short-term dialysis initiation may not substantially affect anxiety or depression levels.

Keywords：Anxiety, Depression, Chronic Kidney Disease, Peritoneal Dialysis, Hemodialysis

Poster Presentation：Chronic Kidney Disease
Poster No.：B0185
Abstract Submission No.：APCN20251028

Comparative Analysis of Subjective Symptoms and Quality of Life in Genetically Confirmed Gitelman Syndrome and Phenotypic Gitelman-like Patients Without Pathogenic Variants

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Abstract

Introduction: Some patients with genetically confirmed Gitelman syndrome (GS) experience significant symptoms affecting quality of life (QOL), but few studies compare them with GS-like cases lacking pathogenic variants. This condition, referred to as pseudo-Gitelman syndrome (pseudo-GS), may result from chronic laxative use or appetite loss, but is also observed in some underweight women without an identifiable cause. Understanding symptom burden in both groups is essential for optimizing patient care.

Methods: We conducted a cross-sectional study of patients clinically suspected of GS who underwent genetic testing. At the time of testing, patients over 10 years old completed a 21-item self-reported questionnaire assessing salt craving, sweet taste aversion, urinary frequency, muscle symptoms, and nonspecific complaints. Each item was scored from 0 to 4, and total symptom scores were calculated. Patients aged 10 or younger were excluded, as they were considered unable to reliably complete the questionnaire. Patients with heterozygous variants in SLC12A3 or with pathogenic variants in other tubulopathy-related genes were excluded. The remaining patients were classified into two groups: those with biallelic pathogenic SLC12A3 variants (GS group) and those without identifiable pathogenic variants (pseudo-GS group). We compared total scores between the two groups and analyzed symptom-score correlations within the GS group.

Results: Among 153 patients aged 10 years or older who were asked to complete the questionnaire, 142 (92.8%) responded. Of these, 66 were included in the GS group and 62 in the pseudo-GS group after excluding 10 patients with heterozygous SLC12A3 variants, 3 with type 3 Bartter syndrome, and 1 with HNF1β-associated kidney disease. There was no statistically significant difference in total symptom scores between the GS and pseudo-GS groups. Salt craving during early childhood, sweet taste aversion, and the presence and duration of nocturnal enuresis were significantly more common in the GS group. In contrast, muscle symptoms and nonspecific complaints were significantly more severe in the pseudo-GS group. Within the GS group, patients over 20 years of age had significantly higher total scores, particularly for muscle and nonspecific symptoms.

Conclusion: Patients without pathogenic variants exhibited a symptom burden comparable to those with GS. Notably, childhood salt craving, sweet taste aversion, and a history of nocturnal enuresis may serve as clinical clues strongly indicative of GS. In GS, symptoms—especially nonspecific ones—worsened with age, suggesting lower QOL in adulthood.

Keywords：Gitelman syndrome

Poster Presentation：Chronic Kidney Disease
Poster No.：B0186
Abstract Submission No.：APCN20251033

Nesfatin-1 Attenuates Renal Fibrosis in a UUO Mouse Model and TGF-β-Stimulated Renal Fibroblasts by Modulating Inflammation, Oxidative Stress, and Fibrotic Pathways

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Abstract

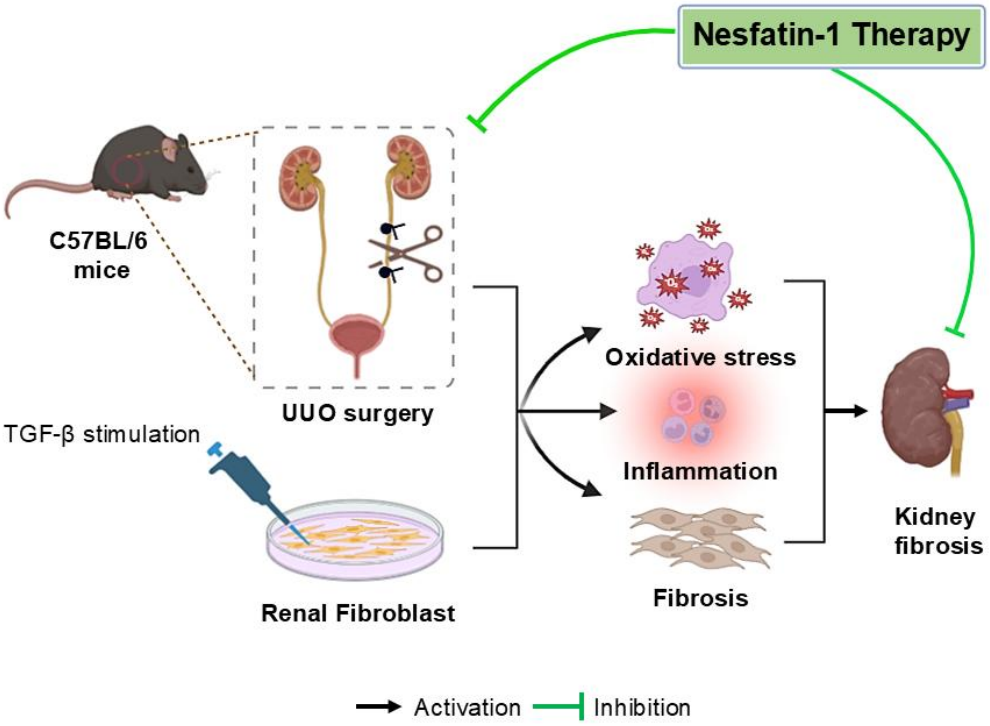
Introduction: Tubulointerstitial fibrosis is a hallmark of chronic kidney disease (CKD), irrespective of the underlying etiology. It is characterized by inflammatory cell infiltration, oxidative stress, and excessive secretion of profibrotic mediators. Nesfatin-1, a satiety peptide, has demonstrated protective effects in various disease models by reducing oxidative stress, inflammation, and apoptosis. However, its role in unilateral ureteral obstruction (UUO)-induced renal fibrosis remains poorly understood. This study aimed to investigate the therapeutic potential of nesfatin-1 in a UUO mouse model and its underlying mechanisms in TGF-β-induced fibrosis in renal fibroblast cells.

Methods: A chronic renal fibrosis model was established in mice via UUO surgery. Nesfatin-1 (5 μg/kg) was administered intraperitoneally on alternate days for 21 days post-surgery. Pirfenidone and Ramipril served as reference drugs. The effects of nesfatin-1 were evaluated through serum biochemistry, histological staining, Western blotting, qRT-PCR, ELISA, and flow cytometry. Parallel in vitro studies were conducted using TGF-β-stimulated NRK-49F renal fibroblast cells to assess molecular mechanisms.

Results: UUO significantly reduced endogenous nesfatin-1 expression level in mice kidney as compared to control mice, which was restored upon exogenous nesfatin-1 administration. Serum creatinine and BUN levels were elevated in UUO mice, while antioxidant enzymes, SOD, CAT, and GSH were reduced. These altered changes were significantly reversed following nesfatin-1 treatment. Histological analyses confirmed that nesfatin-1 improved renal function and ameliorated tubular injury and interstitial fibrosis. Immunohistochemistry, Western blot, and ELISA analyses revealed that nesfatin-1 suppressed the expression of pro-inflammatory cytokines IL-1β, IL-6, TNF-α, and extracellular matrix proteins α-SMA, collagen I, and vimentin. Furthermore, nesfatin-1 attenuated key fibrotic signaling pathways, including TGF-β1/Smad2/3/4 and decreased immune cells infiltration, while upregulated anti-fibrotic markers such as E-cadherin and Smad7 in both in vivo and in vitro models.

Conclusion: These findings suggest that nesfatin-1 confers significant renoprotective effects in UUO-induced renal fibrosis in mice and in vitro fibroblast cells by mitigating oxidative stress, inflammation, fibrosis, and tubular cell injury. Nesfatin-1 may serve as a promising therapeutic candidate for managing CKD-associated fibrosis; however, further validation in other renal disease models is required.

Keywords：Unilateral ureteral obstruction; Nesfatin-1; Renal fibrosis; Inflammation; Oxidative stress; TGF-β signaling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C57BL/6
mice
UUO surgery
TGF-β stimulation
Renal Fibroblast
Oxidative stress
Inflammation
Fibrosis
Kidney
fibrosis
Activation
Inhibition

Poster Presentation：Chronic Kidney Disease
Poster No.：B0187
Abstract Submission No.：APCN20251034

A Fatal case of a Non-uremic Calciphylaxis initially presenting as Leukocytoclastic vasculitis with Thrombotic vasculopathy: A Diagnostic and Therapeutic Dilemma

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Abstract
Introduction: Calciphylaxis, or calcific uremic arteriolopathy, is a rare, life-threatening condition typically seen in end-stage renal disease but can also occur in earlier stages of CKD, acute kidney injury, or with normal renal function—referred to as non-uremic calciphylaxis. It involves calcium deposition in the arteriolar microvasculature of the dermis and subcutaneous adipose tissue, leading to ischemia, necrosis, and painful skin lesions, with high morbidity and mortality. This report presents a case of non-uremic calciphylaxis initially diagnosed as leukocytoclastic vasculitis with thrombotic vasculopathy, highlighting the diagnostic complexity, therapeutic limitations, and poor prognosis of the disease.
Methods: A 57-year-old Filipino male with hypertension, heart failure (EF 28%), chronic kidney disease (baseline creatinine 2.06 mg/dL), and chronic atrial fibrillation was admitted for disorientation, with progressive bilateral leg swelling, erythema, severe pain, and necrotic ulcers with ruptured bullae. Workup showed acute tubular necrosis on top of CKD, which improved with hydration and diuretics (creatinine plateaued at 4.8 mg/dL). Skin biopsy on day 4 revealed leukocytoclastic vasculitis with thrombotic vasculopathy, with differential diagnosis including calciphylaxis. Further staining (Von Kossa) was recommended but unavailable locally. Steroids were not initiated. Management included wound care, debridement, antibiotics, and workup for underlying causes, which was negative. Dialysis was commenced on day 21 after consent. Sodium thiosulfate access was limited. On day 25, the patient developed a fatal arrhythmia; despite resuscitation, the family opted for palliation, and the patient expired.
Results: Skin biopsy on hospital day 4 showed superficial and deep perivascular and interstitial inflammation with neutrophils, nuclear debris, and thrombosed vessels with fibrinoid degeneration and extravasation of erythrocytes Calciphylaxis, both uremic and non-uremic, despite being well-studied has no definitive histologic criteria for diagnosing calciphylaxis, initial skin biopsy findings may be nonspecific and appear in other vascular and skin disorders as in the case of the patient. Repeat histopathology after debridement on day 17 showed changes suggestive of calciphylaxis (Figure 1). The patient had normal calcium and phosphorus levels (days 6 and 25), positive anti-cardiolipin IgG (19.3), and negative ANCA and coagulation workup. Management involves wound care, pain control, and addressing risk factors. Sodium thiosulfate has shown benefit in some cases, but infection-related mortality remains high.
Conclusion: Non-uremic calciphylaxis is a diagnostic and therapeutic challenge due to its nonspecific presentation and high morbidity. While skin punch biopsy is essential for diagnosis, it depends on adequate sampling and expertise. Limited availability of special stains and sodium thiosulfate further complicates management.

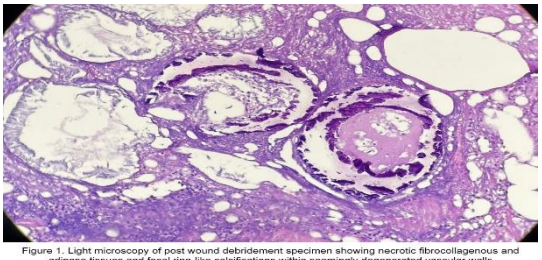
Figure 1. Light microscopy of post wound debridement specimen showing necrotic fibrocollagenous and adipose tissues and focal ring-like calcifications within seemingly degenerated vascular walls

Keywords：non-uremic calciphylaxis, chronic kidney disease

Poster Presentation：Chronic Kidney Disease
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Identification of Behavioral Risks, Environmental Risks, Dietary Risks on Death and Years Lived with Disability in Patients with Chronic Kidney Diseases in Indonesia

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Abstract

Objectives: The prevalence of Chronic Kidney Disease (CKD) is increasing, in 2040 it is projected that CKD will become one of the leading causes of death in the world. Based on the 2018 Basic Health Research, the prevalence of Chronic Kidney Disease in Indonesia increased by 0.5%, and the highest mortality rate was in productive age. This study aims to identify behavioral risks, environmental risks, dietary risks on death and Years Lived with Disability (YLDs) in productive age patients suffering from Chronic Kidney Diseases in Indonesia.

Methods: The method used is quantitative descriptive. Data sourced from the Global Burden of Disease issued by the Institute for Health Matrix Evaluation. The indicators used are mortality, Disability Adjusted Life Years (DALYs), and risk factors consisting of behavioral risk (lead exposure), environmental risk (high temperature), dietary risk (diet in whole grains, diet in high in red meat, diet in hug sugar sweetened beverages, diet low in fiber, diet low in fruits, diet high in processed meat).

Results: The results of the study showed that there was an increase in mortality from 1990-2021, with the highest mortality rate being aged 14-49 years. When viewed from the prevalence and DALYs, the most CKD is over 50 years old, followed by ages 14-59 and the lowest is 0-14 years old. The highest risk factor for death from CKD in Indonesia is a low-fruit diet, followed by a diet in vegetables, a diet in sodium, lead exposure, the rest is around 0-1%. The same thing also happens with DALYs which are also mostly caused by a low-fruit diet, followed by a diet in vegetables, a diet in sodium, lead exposure, the rest is around 0-1%.

Conclusions: Thus, dietary changes that increase consumption of whole grains and fruits, high sodium diets and sugary drinks may reduce the burden of chronic kidney disease.

Keywords：Behavioral Risks, Environmental Risks, Dietary Risks , Chronic Kidney Disease , Indonesia

Poster Presentation：Chronic Kidney Disease
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Chronic Kidney Disease Risk from Mercury-Laden Seafood: An Experimental Study in Indonesian Coastal Communities

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Abstract

Introduction

Chronic kidney disease (CKD) is increasingly linked to environmental exposure to nephrotoxic heavy metals such as mercury (Hg). Seafood, particularly fish and shellfish, can bioaccumulate methylmercury, which poses a potential threat to renal function when consumed regularly. This study aimed to assess the ecological and health risks of mercury exposure through seafood consumption and to estimate its potential contribution to kidney dysfunction among coastal populations.

Methods

An experimental risk assessment study was conducted across five coastal districts in Makassar, Indonesia. Environmental samples including sediment, fish, and shellfish were collected from 10 locations, and mercury concentrations were measured. A total of 114 respondents who regularly consumed local seafood were surveyed. Risk was evaluated using ecological hazard quotient (HQ) and human health risk indices: risk quotient (RQ) for non-carcinogenic effects and excess cancer risk (ECR). The findings were interpreted in the context of potential CKD development.

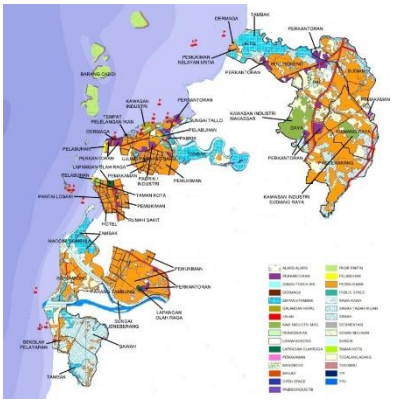
Results

Mercury concentrations in sediments were highest in Cambayya (mean: 2.91 mg/kg) and lowest in Barombong (mean: 1.79 mg/kg). Bioaccumulation was observed in local fish and shellfish species, with mercury levels peaking in Anadara grandis (2.50 mg/kg) and Auratus sp fish (1.46 mg/kg). Ecological risk assessment showed high-risk levels (HQ > 10) for mercury at all sampling sites. RQ analysis indicated significant health risk, particularly in Cambayya (RQ fish: 18.27; RQ shellfish: 17.30) and Untia (RQ shellfish: 20.24), well above the safe limit (RQ > 1). Prolonged exposure in these ranges suggests potential for nephrotoxicity and increased susceptibility to CKD, especially in vulnerable populations such as coastal fishermen, women, and children.

Conclusion

Mercury exposure through seafood in the Makassar coastal region poses a significant ecological and public health threat, with strong implications for kidney function deterioration. This study highlights the urgent need for environmental monitoring, public education, and policy intervention to reduce exposure and mitigate long-term CKD risk. Future research should integrate biomarker testing to directly correlate exposure levels with renal outcomes in affected populations.

Keywords：Chronic Kidney Disease, Mercury, Health Risks, Seafood



Poster Presentation：Chronic Kidney Disease
Poster No.：B0190
Abstract Submission No.：APCN20251082

Effect of Hypnotherapy on Stress Reduction and Disease Progression in Non-Dialysis Chronic Kidney Disease Patients

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Abstract
Effect of Hypnotherapy on Stress Reduction and Disease Progression in Non-Dialysis Chronic Kidney Disease Patients
Background: Chronic kidney disease (CKD) remains a global health concern, with a progressive trajectory toward end-stage renal disease (ESRD) and increasing psychosocial burden. Patients with non-dialysis CKD often endure heightened stress levels due to uncertainty, lifestyle restrictions, and disease burden. Chronic stress contributes to dysregulation of the autonomic nervous system, promoting inflammation and accelerating renal function decline (Jeong et al., 2024). Non-pharmacological approaches, such as hypnotherapy, offer promising adjunctive strategies for stress management and disease modulation.
Objective: This study aimed to evaluate the effects of hypnotherapy on psychological stress, autonomic balance, and disease progression in patients with non-dialysis CKD.
Methods: A randomized controlled trial was conducted involving 80 patients with stage 3–4 CKD not requiring dialysis. Participants were randomized into an intervention group receiving hypnotherapy ($n = 40$) and a control group receiving standard medical care ($n = 40$). The hypnotherapy protocol consisted of six weekly sessions, each lasting 45 minutes, focusing on relaxation, guided imagery, and autosuggestion. Stress levels were measured using the Perceived Stress Scale (PSS), while autonomic function was assessed through heart rate variability (HRV) analysis. Renal function was monitored via changes in estimated glomerular filtration rate (eGFR) over a 12-week period.
Results: Post-intervention analysis revealed that the hypnotherapy group had a statistically significant reduction in PSS scores (mean reduction of 6.8 points; $p < 0.01$), indicating improved psychological well-being. HRV parameters, particularly the high-frequency (HF) component, increased significantly in the intervention group ($p < 0.05$), reflecting enhanced parasympathetic activity and reduced sympathetic dominance. Importantly, the decline in eGFR was slower in the hypnotherapy group compared to controls (mean decline of 1.2 vs. 3.6 mL/min/1.73 m^2; $p < 0.05$), suggesting a potential renoprotective effect.
Conclusion: Hypnotherapy significantly reduces stress and improves autonomic regulation in non-dialysis CKD patients. Moreover, it may contribute to delaying disease progression through modulation of stress-related physiological pathways. As CKD prevalence continues to rise, incorporating hypnotherapy into standard care protocols offers a feasible, cost-effective, and patient-centered adjunct to improve outcomes. Future longitudinal studies are warranted to validate long-term clinical benefits and elucidate underlying mechanisms.
Keywords：Chronic Kidney Disease, Hypnotherapy, Stress Management, Heart Rate Variability, eGFR, Autonomic Nervous System, Non-Pharmacological Intervention

Table : Evidence on the *Effect of Hypnotherapy on Stress Reduction and Disease Progression in Non-Dialysis Chronic Kidney Disease Patients*

No.	Papers	Methode	Population Sample	Findings	Results	Conclusions	Reference
1.	Autonomic modulation with mindfulness-based stress reduction in chronic kidney disease: a randomized controlled trial	▪ Randomized controlled trial design with two groups. ▪ Microneurography to measure muscle sympathetic nerve activity.	▪ Sample size: 29 participants with chronic kidney disease. ▪ Randomized controlled trial sampling method used.	▪ MBSR reduced sympathetic reactivity to mental stress in CKD patients. ▪ No change in sympathetic activity observed in control group (HEP).	▪ MBSR reduced sympathetic reactivity to mental stress in CKD patients. ▪ No change in sympathetic activity observed in HEP group.	▪ MBSR reduces sympathetic reactivity in CKD patients. ▪ Mindfulness training may improve autonomic function in CKD.	Jeong, J., Hu, Y., Zanuzzi, M., DaCosta, D., Sabino-Carvalho, J. L., Li, S., & Park, J. (2024). Autonomic modulation with mindfulness-based stress reduction in chronic kidney disease: a randomized controlled trial. *The Journal of Physiology*. https://doi.org/10.1113/jp287321
2.	Mindfulness-Based Stress Reduction and Autonomic Modulation in Chronic Kidney Disease	▪ Randomized controlled trial comparing MBSR and health education program. ▪ Microneurography for measuring sympathetic nervous system activity.	▪ Sample size: 29 participants (17 MBSR, 12 HEP). ▪ Randomized controlled trial sampling method.	▪ MBSR reduced sympathetic reactivity in CKD patients during mental stress. ▪ No changes observed in HEP group or resting measures.	▪ MBSR reduced sympathetic reactivity during mental stress in CKD patients. ▪ No changes observed in HEP group or resting measures.	▪ MBSR reduced sympathetic reactivity during mental stress in CKD patients. ▪ Mindfulness training showed potential beneficial effects on autonomic function in CKD.	Jeong, J., Hu, Y., Zanuzzi, M., DaCosta, D., Sabino-Carvalho, J. L., Li, S., & Park, J. (2024). Autonomic modulation with mindfulness-based stress reduction in chronic kidney disease: a randomized controlled trial. *The Journal of Physiology*. https://doi.org/10.1113/jp287321
3.	Reducing the hemodialysis patient stress level through progressive relaxation	▪ Quasi-experimental pre-test and post-test study ▪ Progressive relaxation training conducted at least two times a week	▪ Sample size: 58 respondents divided into two groups. ▪ Sampling method: Random sampling used for group selection.	▪ Progressive relaxation significantly reduces stress levels in hemodialysis patients. ▪ Training conducted twice weekly for three weeks is effective.	▪ Progressive relaxation training effectively reduces stress levels in hemodialysis patients. ▪ The decrease in stress levels was statistically significant ($p < 0.05$).	▪ Progressive relaxation reduces stress in hemodialysis patients. ▪ Effective training requires two sessions weekly for three weeks.	Reducing the hemodialysis patient stress level through progressive relaxation. (2022). *Bangladesh Journal of Medical Science*, *21*(4), 842–847. https://doi.org/10.3329/bjms.v21i4.60283
4.	Progressive Muscle Relaxation Therapy on Reducing Anxiety of Renal Failure Patients	▪ Progressive muscle relaxation therapy applied for 5 days. ▪ Descriptive analysis using HARS questionnaire for anxiety measurement.	▪ Sample size: Chronic kidney failure patients with anxiety. ▪ Sampling method: Not specified in the study.	▪ Progressive muscle relaxation reduces anxiety in kidney failure patients. ▪ Anxiety levels decreased from severe to none after 5 days.	▪ Progressive muscle relaxation reduced anxiety in patients. ▪ Anxiety levels decreased from severe to none.	▪ Progressive muscle relaxation reduces anxiety in kidney failure patients. ▪ Anxiety levels decreased from severe to none after therapy	Amalia, M., Nurhusna, N., & Kartika, A. M. (2024). Terapi Relaksasi Otot Progresif Terhadap Penurunan Kecemasan Pasien Gagal Ginjal. *JIK (Jurnal Ilmu Kesehatan)*, *8*(2), 351. https://doi.org/10.33757/jik.v8i2.1128
5.	Hypnotic-based intervention for people with non-communicable diseases : A scoping review	▪ Systematic search of multiple databases conducted. ▪ Included Randomized Controlled Trials published from 2001 to 2021.	▪ Sample size: 589 participants across 11 studies. ▪ Sampling method: Randomized Controlled Trials (RCTs).	▪ Hypnotic interventions show promise for NCD patients' psychological symptoms. ▪ Evidence of effectiveness remains inconclusive; further research needed.	▪ Hypnotic interventions show promise for NCD patients' psychological symptoms. ▪ Evidence of effectiveness remains inconclusive; further research needed.	▪ Hypnotic interventions show promise for NCD patients' psychological symptoms. ▪ Evidence of effectiveness remains inconclusive; further research needed.	Baskoro, D. S., Yuniarti, K. W., & Lufityanto, G. (2024). Hypnotic-based intervention for people with non-communicable diseases : A scoping review. *Cognicia*, *12*(2). https://doi.org/10.22219/cognicia.v12i2.35762
6.	Psychotherapies for chronic kidney disease patients with hemodialysis: A systematic review of randomized control trials and quasi-experiments	▪ Cognitive behavioral therapy, diaphragmatic breathing relaxation, meditation, hypnotherapy. ▪ Murottal Al-Qur'an therapy, spiritual therapy, Kidney Optimal Health Program.	▪ Sample size: 716 CKD patients included in analysis. ▪ Sampling method: Randomized control trials and quasi-experiments reviewed.	▪ Psychotherapy improves mental health in CKD hemodialysis patients. ▪ Cognitive behavioral therapy and murottal Al-Qur'an therapy showed significant benefits.	▪ Psychotherapy improved quality of life, reducing anxiety and depression. ▪ Meditation and KOHP showed no significant improvement in mental health.	▪ Psychotherapy improves mental health in CKD hemodialysis patients. ▪ Further studies needed to assess intervention efficacy.	Zahra, Z., Effendy, E., Mawarpury, M., Marthoenis, M., & Jaya, I. (2023). Psychotherapies for chronic kidney disease patients with hemodialysis: A systematic review of randomized control trials and quasi-experiments. *Narra J*. https://doi.org/10.52225/narra.v3i3.215
7.	Application of Benson Relaxation Therapy Combined with Spiritual Therapy to Reduce Anxiety in Chronic Kidney Disease Patients Undergoing Hemodialysis	▪ Pre-experimental research design with one-group pre-test-post test. ▪ Sample of 50 respondents undergoing relaxation and dhikr therapy.	▪ Population sample size: 50 respondents ▪ Sampling method: Pre-experimental research with one-group pre-test-post test design	▪ Dhikr relaxation therapy significantly reduces anxiety levels. ▪ p-value of 0.000 indicates strong effectiveness.	▪ Dhikr therapy reduces anxiety in hemodialysis patients significantly. ▪ Combination therapy can become standard procedure for all hemodialysis patients	▪ Dhikr relaxation therapy significantly reduces anxiety in hemodialysis patients. ▪ Recommended as a standard procedure for all hemodialysis patients.	Muhith, A., Rohmawati, R., Faizah, I., Sari, R., & Hasina, S. N. (2024). Application of Benson Relaxation Therapy Combined with Spiritual Therapy to Reduce Anxiety in Chronic Kidney Disease Patients Undergoing Hemodialysis. *RGSA*. https://doi.org/10.24857/rgsa.v18n5-067

Poster Presentation：Chronic Kidney Disease
Poster No.：B0191
Abstract Submission No.：APCN20251098

XBP1 Promotes Adaptive Renal Repair through Metabolic Modulation in Kidney Disease

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Introduction: It is believed that the kidney could initiate adaptive/ successful repair to compensate from acute kidney injury (AKI); however, maladaptive/failed repair can result in permanent changes and functional impair, leading to chronic kidney disease (CKD) and processing to end-stage kidney disease (ESKD). The expression of X-box binding protein 1 (XBP1) progressively declines in various models of irreversible renal injury, such as ischemic and obstructive nephropathy. Meanwhile, proximal tubular XBP1 conditional knockout (XBP1cKO) mice induced cell cycle arrest and profibrotic gene expression, which are associated with maladaptive repair and exhibited more severe renal fibrosis. It implies that XBP1 has renoprotective roles in AKI to CKD transition. IXA4 is a transient activator for inducing adaptive IRE1α/XBP1s arm of the unfolded protein response (UPR) without globally activating other signaling pathways related to UPR-related stress responses. This study aims to explore the potential and mechanism of adaptive XBP1 activator for treating renal disease. **Methods:** XBP1cKO mice were subjected to nephrotoxic AKI for examining the influences of XBP1 during kidney injury. IXA4 was administrated in ischemic and nephrotoxic AKI and CKD model. Renal function was evaluated by measuring serum creatinine (Scr) and blood urea nitrogen (BUN) levels. For evaluting the adaptive repair, the expression of H2AX, KIM1, p21, CyclinB1 and CyclinD1 were analyzed by immnuofluorescence staining and immunoblot. For evaluting the the extent of renal fibrosis, sirus red staining was performed, and the level of Tgfb and Col1a1 was evaluated by qPCR. We performed single-cell RNA sequencing (scRNAseq) to elevate the potential mechanisms of IXA4 for renal protection. **Results:** XBP1cKO mice showed more severe damages compared to their littermate control in cisplatin-induced AKI. In contrast, IXA4 induced XBP1s and significantly mitigated kidney dysfunction. By using scRNAseq, we clearly demonstrated that IXA4 administration specifically targets proximal tubular cells and enhances XBP1s transcriptional activity. We identified apoptosis and inflammation as key contributors to cisplatin-induced nephrotoxicity, which were significantly reversed by IXA4 administration. IXA4 was shown to promote adaptive repair of tubular cells following AKI. Moreover, using two CKD models, we directly demonstrated that IXA4 enhances adaptive repair processes in tubular cells. Mechanistically, we found that IXA4 regulated a range of downstream genes that are strongly associated with metabolic pathways and mitochondrial function. **Conclusion:** The results confirm not only the protective role of XBP1 in AKI treatment, but also in reducing CKD progression. The findings will serve as a foundation for developing innovative therapies in the future.

Keywords：Acute kidney injury; chronic kidney disease; unfolded protein response; X-box binding protein 1

Poster Presentation：Chronic Kidney Disease
Poster No.：B0192
Abstract Submission No.：APCN20251140

Effect of Hydroxymethylbutyrate (HMB) Supplementation on Muscle Mass, Strength, and Renal Function in a Patient with Chronic Kidney Disease and Sarcopenia

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Abstract
Background:
An 81-year-old man (height: 154.2 cm, weight: 40.4 kg) with a history of prostate cancer, diabetes, and stage 3b CKD visited our nutrition clinic on April 30, 2025, due to weight loss (from 40.6 kg to 37.9 kg in one month, BMI 16.0), poor glycemic control, and weakness. Nutritional assessment revealed sarcopenia (calf circumference 21.3 cm; male cutoff <34 cm) and mild dysphagia (coughing while drinking). His intake was only 1000 kcal/day (protein 17.2%, carbs 42.3%, fat 40.5%), achieving merely 62.5% of his energy target (1600 kcal/day).

Methods:
HMB, a leucine metabolite, can activate the mTOR pathway, promote protein synthesis, and inhibit proteolysis, supporting muscle maintenance. The patient was advised to consume one can daily of a high-protein supplement containing 330 kcal, 20 g protein, and 1210 mg HMB, protein restriction 1.2 g/kg/day total.

Results:
On follow-up on June 11, body weight increased to 40.4 kg, calf circumference to 23.5 cm, with improved swallowing and mental status. Caloric intake rose to 1400 kcal/day.

Conclusion:
Research on HMB's effects on renal function in CKD remains limited. This case suggests potential benefits of HMB supplementation for nutritional status, muscle mass, and function in diabetic CKD patients with sarcopenia, warranting further investigation regarding its impact on renal parameters.

Keywords：sarcopenia, Hydroxymethylbutyrate, chronic kidney disease, oral supplement

Poster Presentation：Chronic Kidney Disease
Poster No.：B0193
Abstract Submission No.：APCN20251145

Molecular Signatures and Hypoxia-Immune Pathways in Nephrosclerosis and Renal Adaptation After Tumor Nephrectomy

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Abstract
Nephrosclerosis is a progressive kidney disorder marked by glomerulosclerosis and frequently driven by hypoxia-induced injury. Clarifying its molecular landscape and differentiating it from the renal adaptive responses following tumor nephrectomy may reveal novel therapeutic targets. This study aims to identify differentially expressed genes (DEGs), analyze their functional pathways, and explore genetic variants that influence disease progression in nephrosclerosis compared to post-nephrectomy conditions. Gene expression datasets from the GEO were analyzed to identify DEGs between nephrosclerosis and post-nephrectomy samples. Protein–protein interaction (PPI) networks were constructed using Cytoscape to detect central molecular hubs. Functional enrichment analysis was conducted using DAVID and Enrichr. Additionally, genetic variant databases including dbSNP, Prosite, SIFT, and PolyPhen were applied to assess the potential functional impact of gene mutations. In nephrosclerosis, significantly upregulated genes such as DDX3Y, RPS4Y1, EIF1AY, and EDNRB were linked to hypoxia, inflammation, and fibrosis. CXCR4 and CX3CR1 were also elevated, highlighting the role of the CXCR4/CXCL12 signaling axis in hypoxia-induced glomerular injury. In contrast, post-nephrectomy samples showed higher expression of GMFG, HCLS1, PYCARD, and HLA-DQB1, reflecting immune activation, apoptotic processes, and tissue remodeling. Key regulators such as TYROBP, FN1, and ICAM2 emerged from PPI network analysis. Downregulated genes, including XIST, FOS, and EGR1, were implicated in metabolism and stress response, possibly indicating maladaptive renal changes. This study identifies distinct molecular signatures in nephrosclerosis and post-nephrectomy adaptation, emphasizing hypoxia and immune modulation as pivotal mechanisms. Genes like CXCR4, EDNRB, and PYCARD may serve as potential biomarkers or therapeutic targets, warranting further experimental validation.

Keywords：Gene Expression, Glomerulosclerosis, Hypoxia, Nephrosclerosis, Post-Nephrectomy Adaptation

Poster Presentation：Chronic Kidney Disease
Poster No.：B0194
Abstract Submission No.：APCN20251150

Cost-Effectiveness Analysis of Dialysis Modalities for CKD Management in Indonesia

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Abstract
Chronic Kidney Disease (CKD) poses a major economic challenge to Indonesia's universal health coverage system (JKN), particularly due to the high and recurrent costs of dialysis for end-stage renal disease (ESRD). While hemodialysis (HD) remains the dominant modality, peritoneal dialysis (PD) has gained attention as a potentially more cost-effective alternative, especially in resource-constrained and remote settings. Understanding the comparative value of these treatment options is critical for optimizing public health spending under BPJS Kesehatan.
This study applies a Markov model-based cost-utility analysis to compare the cost-effectiveness of HD and PD from the payer's perspective (BPJS), using real-world cost data and quality-adjusted life year (QALY) metrics. Cost parameters were obtained from INA-CBG tariffs and published BPJS reimbursement reports, while effectiveness data were derived from recent Indonesian CKD cohorts and global meta-analyses. The model simulated a cohort of 10,000 ESRD patients over a lifetime horizon, discounting costs and outcomes at 3% annually. Key assumptions included average patient age of 52, transition probabilities based on mortality and complication rates, and average monthly costs of IDR 7.8 million for HD and IDR 6.4 million for PD.
The results showed that PD is the dominant strategy. The lifetime cost per patient for HD was estimated at IDR 1.37 billion, with an effectiveness of 4.22 QALYs, while PD incurred IDR 1.28 billion for 4.79 QALYs. This yields an incremental cost-effectiveness ratio (ICER) of –IDR 150 million per QALY gained, indicating that PD is both less costly and more effective. Sensitivity analysis confirmed the robustness of results across variations in discount rates, utility scores, and hospitalization risks. Scenario analysis for national scaling of PD-first policy suggests potential cumulative savings of IDR 2–3 trillion annually for BPJS if PD adoption rises to 80% coverage.
In conclusion, peritoneal dialysis offers a cost-effective alternative to hemodialysis in Indonesia and supports fiscal sustainability within JKN. Given its favorable cost-effectiveness profile, lower infrastructure dependency, and suitability for decentralized regions, a "PD-first" policy should be prioritized in national kidney care strategies. Additionally, aligning provider reimbursement rates and expanding PD training in health facilities would further enhance implementation feasibility and ensure equitable access to renal care services across Indonesia.

Keywords：Chronic Kidney Disease, Cost-Effectiveness Analysis, Peritoneal Dialysis, Quality-Adjusted Life Years

Poster Presentation：Chronic Kidney Disease
Poster No.：B0195
Abstract Submission No.：APCN20251181

A Rare Case of Bilateral Kidney Malrotation Presenting with Nephrotic Range Proteinuria in a 31 Year Old Female Patient

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Abstract
The case study presented here is that of a 31 year old Filipino female who had episodes of recurrent UTI in the past 15 years, in which no single imaging investigation was done until she presented with complicated pyelonephritis and acute kidney injury at a provincial hospital where computed tomography scan of the abdomen showed bilateral malrotated kidneys. Consult at our institution was done where the patient presented with azotemia, hematuria, and nephrotic range proteinuria. Kidney biopsy was attempted but was deemed technically difficult due to the malrotation and thinning of the cortex in one kidney. The patient was managed as a probable case of secondary glomerulonephritis and chronic pyelonephritis secondary to bilateral malrotated kidneys. Supportive management was done with an ACE inhibitor, healthy diet and lifestyle.

This case adds to the limited literature on bilateral renal malrotation and its possible short and long term complications.

Keywords：Renal malrotation, kidney, proteinuria, case report

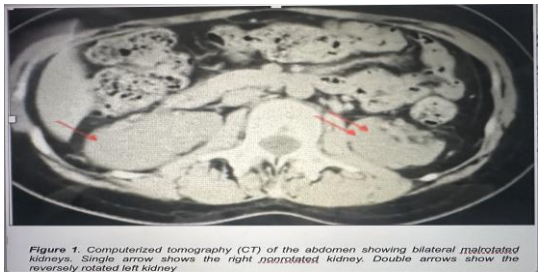

Figure 1. Computerized tomography (CT) of the abdomen showing bilateral malrotated kidneys. Single arrow shows the right nonrotated kidney. Double arrows show the reversely rotated left kidney

Poster Presentation：Chronic Kidney Disease
Poster No.：B0196
Abstract Submission No.：APCN20251214

Water Quality as a Risk Factor for Chronic Kidney Disease in the Context of Agricultural Areas in Indonesia: A Narrative Review

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Abstract
Objectives: Chronic kidney disease (CKD) is a common and increasing public health problem globally, including in Indonesia. One of the causes of this disease is environmental factors, namely drinking water quality. Indonesia's large agricultural areas are known to affect the quality of drinking water consumed by the public. Therefore, the increase in CKD incidence in Indonesia over the past decade has raised suspicions of a link between exposure to contaminated water and kidney damage.
Methods: This narrative review uses PubMed and Google Scholar databases, using articles published between 2018 and 2025. The keywords used in this study are agricultural communities, chronic kidney disease, and water quality. The water quality was assessed by the contamination of heavy metals, pesticides, and other chemical water parameters. This study also highlights the relevance of the findings in the context of Indonesia's agrarian region.
Results: The author identified a total of 10 relevant studies. Most of the studies were conducted as experimental studies and literature reviews relevant to the topic. The international and local literature shows that long-term exposure to heavy metals and pesticides in drinking water has nephrotoxicity to the kidney, which correlates with the prevalence of CKD. Several heavy metals, such as arsenic, lead, chromium, fluoride, and cadmium, are suspected to be anthropogenic sources. This can be exacerbated by other water pollution from industrial discharge, agricultural runoff, and untreated domestic wastewater. In Indonesia, data is still limited, but similar cases have been reported in several regions.
Conclusions: There is a relationship between water quality and the incidence of CKD, particularly in areas with high agricultural output. Further local research is needed on water quality in agricultural areas and its implications for kidney health in the community.

Keywords：Agricultural Communities, Chronic Kidney Disease, Water Quality

Poster Presentation：Chronic Kidney Disease
Poster No.：B0197
Abstract Submission No.：APCN20251226

Safety and optimization strategy of polypharmacy in elderly patients with chronic kidney disease

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Abstract
Objective : Elderly patients with chronic kidney disease (CKD) are often combined with hypertension, diabetes and other basic diseases. Multiple medications (≥ 5) are common, which can easily lead to drug interactions and nephrotoxicity. The purpose of this study is to analyze the risk of drug safety, establish an optimization strategy based on renal function and evidence-based standards, and provide a basis for clinical rational drug use.
Methods : From January 2023 to December 2024,60 elderly patients (≥ 65 years old) with CKD stage 3-5 were retrospectively included. The type, dose and course of treatment were recorded. The inappropriate medication (PIM) and potential missed medication (POM) were evaluated by Beers criteria and STOPP / START criteria. The patients were divided into routine management group (30 cases, maintaining the original treatment plan) and optimized intervention group (30 cases, adjusting the dose of nephrotoxic drugs according to eGFR, discontinuing / replacing PIM, supplementing POM). The patients were followed up for 6 months, and the adverse drug reactions and renal function changes were compared between the two groups.
Results : The average number of drugs used in the patients was 7.3 ± 2.1. The high incidence of PIM mainly involved non-steroidal anti-inflammatory drugs, sulfonylurea hypoglycemic drugs and high-dose ACEI / ARB. POM is mainly lack of vitamin D and statins. After 6 months of optimized intervention, the incidence of adverse drug reactions in the optimized group was significantly lower than that in the conventional group ($P < 0.01$). The decrease rate of eGFR in the optimized group was significantly slower than that in the conventional group ($P < 0.01$), and the medication compliance score of patients was improved ($P < 0.05$).
Conclusion : The risk of polypharmacy in elderly patients with CKD is high, and PIM and POM coexist. The medication optimization strategy based on renal function and evidence-based standards can significantly reduce adverse drug reactions, delay the progression of renal function, and improve patient compliance, which has important clinical application value.

Keywords：elderly patients with chronic kidney disease;multiple medication;security;optimization strategy

Poster Presentation：Chronic Kidney Disease
Poster No.：B0198
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Proteomic analysis from pericardial fluid derived extracellular vesicles in uremia patients

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Abstract

Background: Uremic cardiomyopathy in chronic kidney disease is characterized by reduced renal function and cardiac changes, including cardiac hypertrophy, a significant predictor of mortality, and pericardial effusions in end-stage renal disease. The mechanisms behind uremic pericardial effusion are not fully understood. We aim to collect pericardial fluid from uremia patients with pericardial effusion and isolate the extracellular vesicles for protein mass spectrum

Method: Pericardial fluid was collected from 14 uremia patients and 33 disease control who received cardiovascular surgery. Pericardial fluid-derived extracellular vesicles (EVs) were isolated by ultracentrifuge and then were further analyzed by LC–MS/MS. Bioinformatic analyses were also conducted.

Results: The isolated pericardial fluid-derived EVs were confirmed by nanoparticle tracking analysis and immunoblotting of EVs' markers. Overall, there were 1358 identified proteins from pericardial fluid-derived EVs. Among them, 55 differential expressed proteins were identified based on dual criteria of $p<0.05$ and $|\log2(\text{Uremia/Disease control})|>0.5$ including 35 upregulated and 20 downregulated protein. Gene ontology analysis of identified upregulated proteins showed terms of "positive regulation of protein secretion", "innate immune response", and "regulation of insulin-like growth factor receptor signaling pathway" consistent with inflammatory response. Gene ontology analysis of identified downregulated proteins showed terms of "UDP-N-acetylglucosamine biosynthetic process", and "glycogen metabolic process".

Conclusion: This study demonstrated the pericardial fluid-derived EVs could be a non-invasive approach way to evaluate the effect of uremic toxins on pericardium.

Keywords：Pericardial fluid-derived extracellular vesicles, Proteomics, Uremia

Poster Presentation：Chronic Kidney Disease
Poster No.：B0199
Abstract Submission No.：E_APCN20251274

Transmembrane protein 72, expressed in the distal convoluted tubule, may play a potential role in diabetic kidney disease

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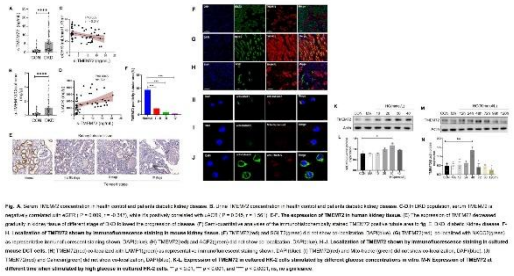
Backgroung: Transmembrane protein 72 (TMEM72) , which involved in normal kidney development and tumorigenesis in renal cell carcinoma, is highly expressed in tubules of the kidney. This study is designed to explore the role and the potential mechanism of TMEM72 in the development of diabetic tubulopathy.
Method: Blood and urine sample were collected in health control, patients with DKD. TMEM72, creatinine, albumin concentration and urine albumin creatinine ratio (uACR) was tested in the two groups mentioned above. Estimated glomerular filtration rate (eGFR) was calculated by the CKD-EPI-2009 formula. Correlation between TMEM72 and renal function and urinary protein was analyzed. Collected normal renal tissue adjacent to renal tumors, as well as kidney biopsy samples from patients with DKD stages II-IV according to Tervaert classification. The variation trend of TMEM72 was determined by immunohistochemistry on kidney tissues from patients in different stage of DKD. Immunofluorescence staining was performed with TMEM72, SGLT2, NKCC2 and AQP2 to identify the expression site of TMEM72 in mice renal tubules. To investigate the potential cellular pathway that TMEM72 was involved, an immunofluorescence test was performed with TMEM72, LAMP1, mito-tracker and calnexin in cultured distal convoluted tubule (DCT) epithelial cells. Western blot was used to detect the activity of TMEM72 in HK-s cells following HG treatment.
Result: The concentration of serum TMEM72 was higher in DKD groups when compared to the health control ($P < 0.001$), same phenomenon can be seen in urine samples. In DKD population, there is a negative correlation between serum TMEM72 and eGFR ($P = 0.009$, $r = -0.347$), patients with higher concentration of serum TMEM72 had lower eGFR. On the contrary, serum TMEM72 was positively correlated with uACR. The expression of TMEM72 decreased gradually in human kidney tissue of different stage of DKD following the progression of disease. Co-localization of TMEM72 and NKCC2 in immunofluorescence staining indicated that TMEM72 was mainly expressed in the lysosomes of distal convoluted tubule. In vitro, the expression of TMEM72 increases with rising glucose concentration and initially rises but then decreases with prolonged glucose stimulation in HK-2 cells.
Conclusion: Our current study has revealed that TMEM72 was mainly expressed in the lysosomes of distal convoluted tubule, and it may correlated with the incidence of DKD. Serum TMEM72 may act as a novel participator in DKD by being involved in the renal tubular injury.

Key Words：diabetic kidney disease; Transmembrane protein 72; diabetic tubulopathy; mitophagy



Poster Presentation：Chronic Kidney Disease
Poster No.：B0200
Abstract Submission No.：E_APCN20251276

PPAR Signaling-mediated Lipid Metabolic Regulation by Micropeptide LSMEM1 Attenuates Renal Pathophysiology

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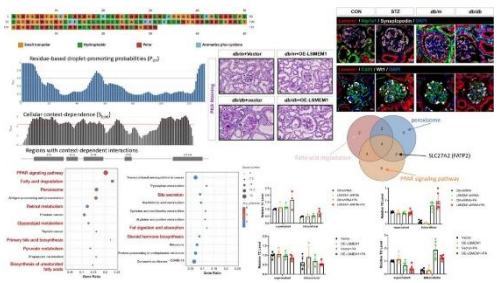
Background:
1) Small proteins or micropeptides play an important role in disease occurrence, cell signaling and metabolic regulation.
2) We aim to elucidate the function of the small protein LSMEM1 and dissect its implications in CKD.
Methods:
1) Patients: Analyzed LSMEM1 expression/localization in CKD patients via IHC/IF. Correlated LSMEM1 levels with BUN/Cre using ImageJ.
2) scRNA-seq: Identified LSMEM1-associated DEGs and biological processes.
3) In vivo/vitro: Assessed LSMEM1 expression in mice, generated KO/OE models, and investigated its role in DKD using RT-qPCR, WB, TSA, ELISA, Co-IP, etc.
Results:
A) Through analysis of the Nephroseq and GEO database we found that LSMEM1 expression was primarily localized to the renal cortex of the kidney and LSMEM1 mRNA levels were significantly higher in CKD patients.
B) LSMEM1expression levels were significantly higher in patients with CKD patients and mice (including *db/db* mice, STA-induced mice, UUO mice and FA mice) compared to normal controls as detected by RT-qPCR, WB, and immunohistochemical staining.
C) LSMEM1 levels were positively correlated with serum creatinine and blood urea nitrogen.
D) Through multiplex immunofluorescence labeling, we confirmed that LSMEM1 is primarily localized in proximal tubule cell (PT) and podocyte (Podo).
E) Of the scRNA-seq analysis, energy metabolism pathways and lipid metabolism-associated pathways were enriched with PPAR signaling, fatty acid degradation, and peroxisome function topping the list.
F) we measured lipid metabolism related indexes in the serum and kidney of mice, and the results showed that after Lsmem1 knock-out, the levels of the serum and kidney triglycerides, cholesterol, and low-density lipoprotein were increased, while the high-density lipoprotein level was decreased.
G) Through Venn diagram analysis of differentially expressed genes associated with the peroxisome proliferator-activated receptor (PPAR) signaling pathway and peroxisome-related pathways, we identified solute carrier family 27 member 2 (SLC27A2/FATP2) as a candidate gene.
H) We also used adeno-associated virus (AAV) overexpressing LSMEM1 to inject into db/db mice. In vitro, we constructed the lentivirus that including LSMEM1-sliencing and LSMEM1-overexpression to infect podocytes and renal proximal tube cells.



Conclusions:
The micropeptide LSMEM1 retards disease progression by reducing the PPAR signaling -mediated lipid droplet accumulation and may be a therapeutic target in CKD.