Oral Presentation (English)

December 14, 2024 (Saturday) 10:30 ~ 12:00 Venue：Room 1 (成杏廳)

【Oral-2】 Chair(s)：林水龍/ Shuei-Liong Lin、宋志建/ Chih-Chien Sung

10:30—10:42

1. Protective Effect of Combined use of Liraglutide and Empagliflozin on Tacrolimus-Induced Diabetes Mellitus and Nephrotoxicity in a Rat Model
Do Hyun Na[1,2], Sheng Cui[1], Xianying Fang[1], Hanbi Lee[1,2], Sang Hun Eum[1,3], Eun Jeong Ko[1,4], Yoo Jin Shin[1], Sun Woo Lim[1] and Byung Ha Chung[1,2]
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10:42—10:54

2. Mitochondrial AKT1 Signaling and Kidney Injury: Unraveling Mechanistic Insights into Metabolic Syndrome-Induced Renal Dysfunction
Hugo Y.-H. Lin[1,2,3], I-Ya Chen[4], Tzu-Ming Wang[1]
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[2.] Division of Nephrology, Department of Internal Medicine, Kaohsiung Medical University Hospital
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[4.] Graduate Institute of Clinical Medical Sciences, College of Medicine, Chang Gung University, Taoyuan, Taiwan

10:54—11:06

3. Effects of Vitamin K3 analog Phthiocol against Vascular Calcification in Chronic Kidney Disease
Tsung-Jui Wu[1, 2], Yi-Cheng Wang[3], Chung-Jen Lee[3,4], Bang-Gee Hsu[2, 3, 5]
[1]Division of Nephrology, Hualien Armed Forces General Hospital, Hualien, [2]Institute of Medical Sciences, Tzu Chi University, Hualien, [3] Division of Nephrology, Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Hualien, [4]Department of Nursing, Tzu Chi University, Hualien, [5]School of Medicine, Tzu Chi University, Hualien, Taiwan

11:06—11:18

4. Alleviation of Chronic Kidney Disease by Combinational Approach of Resveratrol and Cinnamic Acid in Rodents by Regulating Gut Microbiota and Inflammation
E. Yadav
Shalom Institute of Health and Allied Sciences, Sam Higginbottom University of Agriculture Technology and Sciences, Allahabad, India

11:18—11:30

5. SGLT2 Inhibitors vs. GLP-1 Agonists: Impact on Dementia and Outcomes in Type 2 Diabetes with AKD
Ying-Ru Chen[1], Jui-Yi Chen[2], Vin-Cent Wu[3,4]
[1] Taipei Medical University School of Medicine, [2] Chi Mei Hospital Department of Nephrology , [3] Division of Nephrology, Department of Internal Medicine, National Taiwan University Hospital, Taipei, Taiwan; [4] NSARF (National Taiwan University Hospital Study Group of ARF), and CAKS (Taiwan Consortium for Acute Kidney Injury and Renal Diseases), Taipei, Taiwan.

11:30—11:42

6. Efficacy of Novel Oral Anticoagulants in Acute Kidney Disease Patients with Incident Atrial Fibrillation

Chung-Te Chu[1], Yi-Hong Wu[1], Cheng-Chien Lai[1], Der-Cherng Tarng[1,2,3,4], Wei-Cheng Tseng[1,2,3,*]

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11:42—11:54

7. The Impact of SGLT2 Inhibitors on Mortality and Cardiovascualr and Kidney Outcomes in Patients with Type 1 Diabetes and Acute Kidney Disease

Chung-An Wang [1], Li-Chun Lin [2], Jui-Yi Chen [3], Wei-Jie Wang [4], Vin-Cent Wu [5]

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1

Protective effect of combined use of liraglutide and empagliflozin on Tacrolimus-induced diabetes mellitus and nephrotoxicity in a rat model

Do Hyun Na[1,2], Sheng Cui [1], Xianying Fang[1], Hanbi Lee[1,2], Sang Hun Eum[1,3], Eun Jeong Ko[1,4], Yoo Jin Shin[1], Sun Woo Lim[1] and Byung Ha Chung[1,2]
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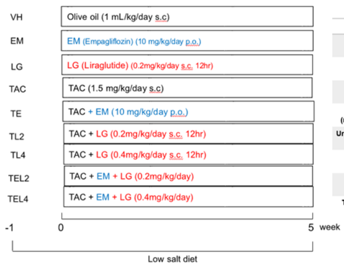
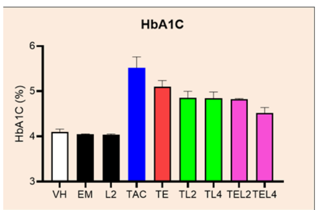
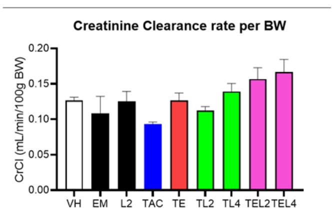
Background: Glucagon-like peptide-1 receptor agonists (GLP-1RA) and sodium-glucose co-transporter-2 inhibitors (SGLT2i) are important newer anti-diabetic drugs in the treatment of type II diabetes mellitus (DM). However, their use in tacrolimus (TAC)-induced DM remains undetermined. The aim of this study is to investigate the protective effect of the combined use of liraglutide (GLP-1RA) and empagliflozin (SGLT2i) on the TAC induced pancreatic and kidney injury.
Methods: Sprague-Dawley (SD) rats were divided into six groups, each containing six rats, and were treated with a low-salt diet and Tacrolimus (1.5mg/kg/day, subcutaneously) for 5 weeks. In addition to Tacrolimus, liraglutide (0.2mg/kg/day or 0.4mg/kg/day subcutaneously q12hrs), empagliflozin (10mg/kg/day, oral gavage), and a combination of both were administered for 5 weeks to evaluate their potential protective effects. The effect of liraglutide and empafliflozin was evaluated by assessing HbA1c, creatinine clearance rate and by measuring markers of oxidative stress, apoptosis. Also, Morphologic change of kidney and pancreas was observed.
Results: In the experimental SD rat model of TAC-induced DM and nephrotoxicity, combined use of GLP-1RA and SGLT2i significantly decreased blood glucose level, HbA1C and increased pancreatic islet size. In the kidney, combined use of those drugs improved renal function assessed by creatinine clearance, and decreased interstitial fibrosis and profibrotic cytokines as well. Increased oxidative stress by TAC was remarkably decreased with GLP-1RA or SGLT2i in serum, pancreatic and renal tissues..
Conclusion: Combined use of GLP-1RA and SGLT2i showed protective effect on TAC-induced DM and nephrotoxicity.

Key words:
Glucagon-like peptide-1 receptor agonists, SGLT2 inhibitors, Tacrolimus induced diabetes mellitus, Post-transplant diabetes mellitus, Kidney transplant



	VH	TAC	TAC+EM	TAC+LL	TAC+HL	TAC+EM+LL	TAC+EM+HL
BW (g)	401 ± 25	343 ± 9a	349 ± 11a	336 ± 2a	339 ± 5a	351 ± 10a	318 ± 4a
△BW (g)	197 ± 21	145 ± 7a	152 ± 10a	138 ± 5a	134 ± 4a	144 ± 10a	119 ± 3a
UV (ml/day)	16.5 ± 4.2	43.0 ± 11	41.2 ± 7.5	23.5 ± 6.5	26.3 ±4.8	32.5 ± 12.8	41.3 ± 7.1
CrCl (mL/min/100g BW)	0.13 ± 0.004	0.09 ± 0.003a	0.13 ± 0.01	0.11 ± 0.01	0.14 ± 0.01b	0.16 ± 0.02b	0.17 ± 0.02abd
Urinary glucose (mL/day/100g BW)	0 ± 0	182 ± 178a	413 ± 85a	213 ± 71a	306 ± 128a	741 ± 285a	754 ± 118a
HbA1C (%)	4.10 ± 0.06	5.52 ± 0.24a	5.10 ± 0.14a	4.85 ±0.15	4.84 ± 0.14ab	4.92 ± 0.10	4.60 ± 0.13b
AST (U/L)	121 ± 8	199 ± 4a	182 ± 23	177 ± 12	160 ± 5	106 ± 9b,c,d	101 ± 5b,c,d,e
TAC conc. (ng/mL	0.48 ± 0.21	0.50 ± 0.17	0.30 ± 0.10	0.57 ± 0.11	0.50 ± 0.10	0.75 ± 0.05	0.48 ± 0.21





2

Mitochondrial AKT1 Signaling and Kidney Injury: Unraveling Mechanistic Insights into Metabolic Syndrome-Induced Renal Dysfunction

粒線體 AKT1 訊號傳導和腎臟損傷：揭示代謝症候群引起的腎功能障礙的機制

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Background：
Metabolic syndrome (MetS) is associated with kidney diseases, but the etiology is inconclusive. We hypothesized that renal tubular mitochondrial AKT1 (mito-AKT1) signaling plays a mechanistic role in the pathogenesis of kidney injuries in MetS.

Methods：
We executed the study with 8-week C57BL/6 male mice fed a high-fat diet for four months, compared with mice fed a standard chow diet. To examine the role of mito-AKT1 translocation, the cell viability of HK-2 cells treated with heat shock protein 90 (Hsp90) was examined.

Results：
In our murine MetS model, body weight significantly increased. The kidney size as a percentage of body weight remained similar between the groups. For the glucose tolerance test, fasting glucose levels were significantly higher in MetS mice than those on a regular diet ($p<0.05$). From 15 to 120 minutes, glucose levels were consistently elevated. The intraperitoneal glucose tolerance test curve differed between three and six months, with the area under the curve (AUC) value being higher at six months ($p=0.013$). Fasting hyperinsulinemia and insulin resistance, as measured by the Homeostatic Model Assessment for Insulin Resistance, were also elevated. Regarding renal function, serum BUN and creatinine levels in MetS mice did not change significantly. However, proteinuria and urine KIM-1 levels were elevated, which indicated renal tubular injuries. Histological examination revealed significant increases in glomerulosclerosis index, tubulointerstitial fibrosis, tubular dilatation, and tubular vacuolation score. To further investigate the role of mito-AKT1 signaling during MetS in renal tubules, we examined the mito-AKT1 protein. We observed an increased accumulation of phosphorylated AKT1 ($p=0.030$) in the mitochondria of proximal tubules after MetS. This AKT1 translocation was confirmed using immunohistochemistry staining and western blot analysis of mitochondrial proteins. Cell viability significantly decreased in the group treated with palmitic acid and the Hsp90 inhibitor, compared to the vehicle-treated group ($p<0.01$).

Conclusions：
These findings shed new light on the mechanistic role of renal tubular mito-AKT1 in MetS-induced kidney injuries and may be used to develop new strategies for preventing and treating kidney diseases.

Key words：
mitochondria, AKT1, metabolic syndrome

3

Effects of vitamin K3 analog phthiocol against vascular calcification in chronic kidney disease

Vitamin K3 衍生物 phthiocol 對於慢性腎臟病血管鈣化之保護效果

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Background: Vascular calcification (VC) is a multi-factorial pathological deposition of calcium, involving the trans-differentiation of vascular smooth muscle cells (VSMCs) into osteo-/chondroblast-like cells. VC is found to be associated with cardiovascular morbidity and mortality, especially in patients with chronic kidney disease (CKD). Phthiocol, a hydroxy analog of vitamin K3, is known for anti-hemorrhagic activity and recently found anti-tumor activity owing to its quinone structure. We investigated the effects of phthiocol on inorganic phosphate (Pi)-induced VC in VSMCs and high phosphorus diet with induced vascular calcification in ApoE-knockout mice with 5/6 nephrectomy-induced chronic kidney disease.

Methods: In *vitro* experiments were carried out by using Pi-induced vascular calcification in mouse VSMCs, co-treated without or with different concentrations of phthiocol. In *vivo*, the study of vascular calcification was induced by an 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 into 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: Phthiocol treatment in VSMCs cultured in a high Pi medium suppressed reactive oxidative species production, ferroptosis, and subsequent cell death and calcification in a dose-dependent manner. Phthiocol 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 PI3K, Nrf2, or HO-1 significantly abolished the protective effect of phthiocol on Pi-induced apoptosis and calcification. 5/6 nephrectomy and high-phosphorus diet in ApoE$^{-/-}$ mice significantly increased blood TCH, TG, BUN, Cre, phosphorus, PWV levels, and calcium deposition in the aortic sections shown in Von Kossa stain, while decreased blood HDL and tGFR compared with the control group. Phthiocol treatment in the VCP group significantly decreased blood TCH, TG, BUN, Cre, phosphorus, PWV levels, and aorta calcification while increased blood HDL levels and tGFR, compared with the VC group.

Conclusions: Vitamin K3 analog phthiocol 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.

Key Words: Phthiocol, Vascular calcification, High-phosphorus diet, ApoE-knockout mice, Chronic kidney disease, PI3K/Akt pathway, Nrf2/HO-1 activation.

4

Alleviation of Chronic Kidney Disease by Combinational Approach of Resveratrol and Cinnamic Acid in Rodents by Regulating Gut Microbiota and Inflammation

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Abstracts

Background：

Chronic kidney disease (CKD) is a debilitating pathology with various causal factors, culminating in end-stage renal disease (ESRD) requiring dialysis or kidney transplantation. It is a worldwide epidemic; therefore, there is an urgent need for promising preventive approaches. The current study was designed to explore the effect of two bioactive polyphenols, resveratrol (RTL) and cinnamic acid (CA), against adenine-induced CKD in mice.

Methods：

Female C57BL/6 mice were utilized for the study and randomly divided into different groups: negative control, model control, and models treated with RTL, CA and RTL+CA combinations at two dose levels each. CKD was induced with adenine by injecting intraperitoneally for 4 weeks, while the experimental treatment was started 14 days before the administration of adenine injection and then continued until 10 weeks.

Results：

Significant inhibition in was observed in tubular dilation, tubulointerstitial atrophy, interstitial chronic inflammation, and acute kidney inflammation in CKD-induced mice in RTL+CA group in a dose-dependent manner, followed by individual treatment groups. Additionally, it significantly reduced the level of serum cystatin. CKD-induced damaging effects, including levels of inflammatory cytokines and circulating concentrations of biomarkers associated with kidney injury, along with a reduced cecum Clostridium leptum group, were also reversed by the RTL+CA group dose-dependently.

Conclusions：

RTL+CA combinational approach has the significant potential to ameliorate the development and further progression of CKD.

Key words：

Polyphenols, CKD

5

SGLT2 Inhibitors vs. GLP-1 Agonists: Impact on Dementia and Outcomes in Type 2 Diabetes with AKD
SGLT2 抑制劑 vs. GLP-1 促效劑：對伴有 AKD 的 2 型糖尿病患者癡呆症及預後的影響

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Background：

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

Methods：

This cohort study utilized data from the TriNetX Research Network. The study included patients without dementia, aged 18 or older, who had been detached from temporary dialysis and survived for at least 90 days following discharge from the hospital. Participants were grouped based on their use of either SGLT2i or GLP-1RAs, with data collection spanning from September 2, 2012, to January 1, 2024. Developiting dementia, all-cause mortality, major adversed cardiovascular events (MACE), and major adversed kidney events (MAKE),were assessed over a period of three months to five years post-discharge.

Results：

Among 10638 individuals who could withdraw from acute dialysis, 7842 patients were prescribed with SGLT2i, while another 2796 were GLP-1RAs users. After performing 1:1 propensity score matching, there were 2691 patients in each group. Patients prescribed SGLT2i demonstrated a significantly lower risk of dementia (adjusted hazard ratio [aHR] 0.516, 95% CI 0.338-0.788) and MAKE ([aHR] 0.751, 95% CI 0.607-0.93)compared to GLP-1RAs users. However, there was no significant difference in all-cause mortality ([aHR] 0.898, 95% CI 0.707-1.141)and MACE([aHR] 1.18, 95% CI 0.966-1.441) between the two groups.

Conclusions：

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

Key words：

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

6

Efficacy of Novel Oral Anticoagulants in Acute Kidney Disease Patients with Incident Atrial Fibrillation

新型口服抗凝血劑於新發生心房顫動的急性腎臟病病患之效用

Chung-Te Chu[1], Yi-Hong Wu[1], Cheng-Chien Lai[1], Der-Cherng Tarng[1,2,3,4], Wei-Cheng Tseng[1,2,3,*]

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Background：

Acute kidney disease (AKD) is a serious, potentially life-threatening condition, and links to higher chance of atrial fibrillation (Af). Novel oral anticoagulants (NOAC) have become an promising treatment to decrease morbidity and mortality in Af patients without kidney disease. Nonetheless, the efficacy of NOAC in AKD patients with incident Af remains obscure.

Methods：

We analyzed a longitudinal cohort of 2786 AKD patients from a tertiary medical center between 2011 and 2021. The definition of AKD is based on the Kidney Disease: Improving Global Outcomes guidelines. Afterwards, 987 AKD patients with incident Af were enrolled and categorized into NOAC and warfarin groups. Cox regression was used to estimate the hazard of NOAC and warfarin on study outcomes (all-cause mortality, major cardiovascular events [MACE: cardiovascular death, myocardial infarction, ischemic stroke, heart failure hospitalization], and end-stage renal disease [ESRD]).

Results：

During a median follow-up of 399 days, 207 patients died, 170 patients had MACE and 192 patients had ESRD. Kaplan-Meier survival analysis showed that The NOAC group had a lower chance of ESRD (p <0.001). Multivariable Cox regression further validated that NOAC users were associated a lower risk for ESRD as compared to warfarin users (adjusted hazard ratio: 0.65; 95% confidence interval: 0.461-0.911, p = 0.013). The risk of all-cause mortality was comparable between NOAC and warfarin users. The results were consistent in subgroup and sensitivity analyses.

Conclusions：

NOAC has a superior therapeutic efficacy for AKD patients with incident Af as compared to warfarin.

Key words：Acute kidney disease; Atrial fibrillation; Novel oral anticoagulants

7

The impact of SGLT2 Inhibitors on Mortality and Cardiovascualr and Kidney Outcomes in Patients with Type 1 Diabetes and Acute Kidney Disease
SGLT2 抑制劑對於第一型糖尿病合併急性腎病患者之死亡與心腎風險的影響

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Background：

Sodium-glucose cotransporter 2 inhibitors (SGLT-2is) have demonstrated benefits in improving cardiovascular and kidney outcomes in diabetic patients. However, the association of SGLT-2is with cardiorenal outocmes in patients with type 1 diabetes (T1DM) and acute kideney disease (AKD) remains understudied.

Methods：

This retrospective cohort study used global healthcare record from the TriNetX network included patients aged 18 years or older with type 1 diabetes who experienced AKD and discharged from hospitals between January 1, 2012, and December 31, 2023. Patients with dialysis re-initiation or sustained kidney impairment post-discharge were excluded. The cohort was stratified into SGLT-2i users (n=599) and non-users (n=16,091) and subjected to 1:1 propensity score matching. Primary outcomes included major adverse cardiac events (MACE), major adverse kidney events (MAKE) and all-cause mortality, with a follow-up period to a maximum of 3 years.

Results：

A total of 590 patients with T1DM (mean age: 61±13.5 years, 45.3% male) underwent SGLT-2is treatment during the acute kidney disease (AKD) period, matched with their counterparts. The use of SGLT-2is was significantly associated with a lower risk of MAKE (adjusted hazard ratio [aHR] = 0.62, 95% confidence interval [CI] = 0.39-0.98) and all-cause mortality (aHR = 0.45, 95% CI = 0.29-0.68) compared to non-users. However, no significant reduction was observed in the risk of major adverse cardiovascular events (MACE) (aHR = 0.80, 95% CI = 0.51-1.27).

Conclusions：

In this study, treatment with SGLT-2is was associated with improved kidney outcomes and reduced all-cause mortality in patients with T1DM and AKD, though no significant reduction in MACE were observed. Importantly, these findings highlight the potential benefits of SGLT-2is in enhancing renal recovery and survival in patients with T1 diabetes following AKD.

Key words：

Acute Kidney Disease, Sodium-Glucose Cotransporter 2 inhibitors, Type 1 Diabetes