論文發表注意事項

【口頭論文發表】

- 試片室：7 樓 701C 會議室及 701G 會議室外小房間
- 口頭報告者請務必於該場次開始前 30 分鐘將隨身碟自行攜帶送至試片室進行測試，以避免中途影響會議速度進行，請先行測試檔案與隨身碟讀取正常。
- 一般論文口頭發表，每題 12 分鐘(報告 10 分鐘，討論 2 分鐘)，請各演講者務必控制報告時間，演講時間結束後即開燈結束演講。
- 學會於90年新增『年會論文優秀論文獎』，口頭發表及壁報發表分別評分。優秀論文獎得獎名單於會員大會公佈並頒獎。
- 得獎公佈—會員大會
  時間：112 年 12 月 10 日(星期日)上午 11:30 至 12:00 (請得獎者務必在現場)
  地點：701B會議室

- Our Preview Room are located outside of conference rooms 701B and 701F
- ***Oral Presentation***

Presentation Time
12 Minutes: including 10 minutes of presentation and 2 minutes of Live Q&A
Presentation Specification
***All oral presentation must Present LIVE.** File Type: **PPT or PPTX** only File Name: Oral_AbstractID_Name (e.g: OralPresentation1_25_Lin)

Oral Presentation 4 (English)

December 9 (Saturday), 2023 09:00 ~ 10:30 Room 7 (701-F)

【Basic-3】 *Chair(s)：許育瑞/ Yu-Juei Hsu、黎思源/ Szu-Yuan Li*

09:00—09:12

1. The Effects of Hypoxia-Inducible Factors on Water Regulation in the Kidney
Ching-Chun Yang[1], Kai-Ting Huang[1], Szu-Yu Pan[1,2], Shuei-Liong Lin[1,2]
[1] National Taiwan University College of Medicine,[2] National Taiwan University Hospital

09:12—09:24

2. Nephroprotective potential of Gallic acid metformin against Streptozotocin induced diabetic nephropathy in Wistar rats via inhibition of DPP-4 and TGF-β
Vikas Kumar[1], Firoz Anwar[2]
[1]Department of Pharmaceutical Sciences, Sam Higginbottom University of Agriculture, Technology & Sciences, Prayagraj, India
[2]Department of Biochemistry, King Abdulaziz University, Jeddah, Saudi Arabia

09:24—09:36

3. Synergy of clones of IgG1/3 anti-PLA2R autoantibody leads to complement activation and podocyte damage
Kun-Hua Tu[1,3], Tsai-Yi Wu[3], Tai-Di Chen[2], Ji-Tseng Fang[1], Chi-Wei Yang[1], Cheng-Lung Ku[3]
[1]Department of Nephrology, [2]Department of Pathology, Chang-Gung Memorial Hospital, [3]Graduate Institute of Clinical Medical Sciences, Chang Gung University, Taoyuan, Taiwan

09:36—09:48

4. The Effect of Non-Decaffeinated Coffee and and Decaffeinated Coffee After Induction of High-Purine Diet on Kidney Superoxide Dismutase (SOD) Levels in The Hyperuricemic Rats (Rattus norvegicus)
Hilmi Ardian Sudiarto[1], Dwi Nur Ahsani[2]
[1]General Practitioner, Department of General Practitioner, Bendan General Hospital, Pekalongan, Indonesia
[2]Department of Histology, Faculty of Medicine, Universitas Islam Indonesia

Oral Presentation 4 (English)

December 9 (Saturday), 2023 09:00 ~ 10:30 Room 7 (701-F)

【Clinical-2】 *Chair(s)：許育瑞/ Yu-Juei Hsu、黎思源/ Szu-Yuan Li*

09:48—10:00

5. AST-120 improved uremic pruritus by lowering indoxyl sulfate and inflammatory cytokines in hemodialysis patients

Chia-Chao Wu[1], Ya-Chung Tian[2], Chien-Lin Lu[3], Ming-Ju Wu[4], Paik-Seong Lim[5], Yi-Wen Chiu[6], Ko-Lin Kuo[7], Shou-Hsuan Liu[2], Yu-Ching Chou[8], Chien-An Sun[9], Yi-Chou Hou[10] and Kuo-Cheng Lu[7]

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10:00—10:12

6. Prevalence of anxiety and depression in hemodialysis patients of end stage renal disease

Muhammad Usman Noor, Santosh Kumar, Abdul Manan Junejo, Sadia Rehman, Om Lal, Muhammad Tassaduq Khan

Departments of Biochemistry, Medicine and Nephrology, The Kidney Foundation, Jinnah Sindh Medical University, Jinnah Post Graduate Medical Center, Bahria University Medical and Dental College, Dow University of Health Sciences, Karachi, Pakistan

10:12—10:24

7. Risk Factors of Rehospitalization in Chronic Kidney Disease Patients: An Observational Study

Ruba Ishtiaq[1], Muhammad Tassaduq Khan[1], Amna Hamid[2], Beenish Hamid[3]

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【Oral Presentation 4】
【Basic-3】 Room 7 (701F)

1

The Effects of Hypoxia-Inducible Factors on Water Regulation in the Kidney
缺氧誘導因子對於腎臟水份調節的影響

Ching-Chun Yang[1], Kai-Ting Huang[1], Szu-Yu Pan[1,2], Shuei-Liong Lin[1,2]
楊晶淳[1], 黃楷婷[1], 潘思宇[1,2], 林水龍[1,2]
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Background：
Previous studies have shown wide-spread HIF-1α-dependent inflammatory and fibrotic lesions in the kidney of mice with von Hippel-Lindau gene (*Vhlh*) deletion in Hoxb7-expressing renal collecting duct (CD) epithelia. Interestingly, *Vhlh* deletion in Ksp1.3-expressing renal tubular epithelia has also been reported to cause HIF-1α dependent diuresis in mice.
Methods：
Tg(Hoxb7-Cre);Vhlh$^{F/F}$ (*Vhlh* KO), *Vhlh*;*Hif1a* DKO, and *Vhlh*;*Hif2a* DKO mice were bred to study the effect of HIF stabilization in Hoxb7-expressing renal CD epithelia. Littermate without *Hoxb7-Cre* transgene was used as the control to compare body weight, blood and urine biochemistry, gene/protein expression and histology in kidney.
Results：
Compared to littermate control, *Vhlh* KO mice exhibited higher 24-hour urine volume and lower urine osmolality which could be partially ameliorated by water deprivation. Water deprivation-induced, vasopressin-dependent urine concentration was not different between *Vhlh* KO mice and littermate control. *Vhlh* KO mice exhibited higher food intake and solute diuresis, but decreased body weight. Food restriction led to similar excretion of urine solutes, but *Vhlh* KO mice consistently exhibited higher urine volume and lower urine osmolality than littermate control. *Vhlh*;*Hif2a* DKO mice exhibited higher urine volume and lower urine osmolality, but no more solute diuresis. *Vhlh*;*Hif1a* DKO mice did not exhibit abnormality in urine volume and osmolality. Histologically, *Vhlh* KO mice exhibited substantial tubulointerstitial injury.
Conclusions：
Vhlh KO and hence HIF-1α/HIF-2α overexpression in renal CD epithelia led to HIF-1α-dependent decrease in renal concentrating ability. However, increase of solute diuresis was HIF-2α-dependent. Water deprivation-induced, vasopressin dependent urine concentration was not impaired in *Vhlh* KO mice. HIF-1α-dependent diuresis might be caused by destructed renal interstitium. But the mechanisms underlying HIF-2α-dependent solute diuresis need further study.
Key words：polyuria, Hif1a, Hif2a, Vhlh, vasopressin, aquaporin-2

2

Nephroprotective potential of Gallic acid metformin against Streptozotocin induced diabetic nephropathy in Wistar rats via inhibition of DPP-4 and TGF-β

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[2]Department of Biochemistry, King Abdulaziz University, Jeddah, Saudi Arabia

Background：

Dipeptidyl-peptidase IV inhibitors (DPP-4) have gain popularity day by day as anti-diabetic agents and now are broadly used in the treatment of type 2 diabetes with chronic renal dysfunction.DPP-4 inhibitors have potential to reduce the glucose level independent of the renal function either reduce the level of glycated albumin without inducing the hypoglycaemic effects. Studies suggest that DPP-4 exert the renal protective effect via maintain the incidence of albuminuria. The current experimental study was make attempt to explore the renal protective effect of gallic acid- metformin (GA-Met) against the STZ induced diabetic rats via inhibition of DPP-4 and TGF-β.

Methods：

GA-Met was scrutinizing against the DPP-4 inhibitor. GA-Met were also examined via Insilco study with the structure of DPP-4 to identify the critical interactions for its bioactivity. STZ was used for induction the type 2 diabetes and blood glucose level, biochemical, antioxidant, cytokines and inflammatory mediators were estimated.

Results：

DPP-4 assay, GA-Met was found as potential drug with IC50 value = 4.34 μM. GA-Met Insilco interacted with various residue of DPP-4 inhibitor. GA-Met significantly reduced the blood glucose level (67%) and increased the plasma insulin level (45.5%). GA-Met improve the interstitial fibrosis, tubulointestitial injury and inflammatory cell infiltration in animal tissue. GA-Met exhibited the significantly decreasethe level of TNF-α (45%), Il-1β (54.3%). IL-6 (56.1%), caspase-1 (43%), caspase-3 (40.4%), COX-2 (65%). PGE_2 (60.3%) and NF-kB (52.3%). Oxidative stress marker and the expression of transforminggrowth factor-β (TGF-β) in the renal tissue of diabetic rats were significantly ($P<0.001$) altered by GA-Met treated group rats.

Conclusions：

The current investigation suggests that GA-Met exert the renal protective effect against the STZ induced DN rats via inhibition of DPP4 and TGF-β.

Key words：

Diabetic nephropathy, DPP-4, Gallic acid-metformin, TGF-β, Inflammation

3

Synergy of clones of IgG1/3 anti-PLA2R autoantibody leads to complement activation and podocyte damage

IgG1/3 抗磷脂酶 A2 受體抗體之協同作用導致補體活化及足細胞損傷

Kun-Hua Tu[1,3], Tsai-Yi Wu[3], Tai-Di Chen[2], Ji-Tseng Fang[1], Chi-Wei Yang[1], Cheng-Lung Ku[3]

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Background：

Primary membranous nephropathy (pMN) is an autoimmune disease characterized by deposition of immune complexes with complement activation upon glomeruli. Phospholipase A2 receptor 1 (PLA2R1) has been identified as a major auto-antigen in ~70% of pMN patients. IgG1 and IgG3 antibodies are believed as the main subclass of immunoglobulin to activate complement system rather than IgG4. Interestingly, the IgG4 autoantibodies are the predominant subclass of anti-PLA2R1 in pMN. This raises questions about the role of different IgG subclasses in mechanism of pMN, and how PLA2R autoantibodies lead to podocyte damage via complement activation.

Methods：

We utilized a single-cell capture method to generate 16 anti-PLA2R monoclonal antibodies (mAbs). These mAbs recognize four major epitopes, especially in the N-terminal region. Using the CDC assay, we assessed the complement activation potential of these mAbs.

Results：

While IgG4 anti-PLA2R mAb could not induce complement activation, one specific clone (1E12) originated from IgG4 showed mild cytotoxicity of PLA2R-overexpressed podocytes when converted to the IgG1 subclass. Combinations of mAbs to CTLD1 and mAbs to any other domain produced a synergistic effect, resulting in robust CDC activation which can't be achieved by single mAbs. Notably, this synergy in complement activation was observed even when IgG1 mAbs were paired with IgG4 mAbs. In other aspect, IgG4 seems to diminish this synergy. When CysR and CTLD1 epitope-specific IgG4 mAbs were added, complement activation was vastly reduced as IgG4 concentration increased. Polyclonal IgGs from pMN patients validated the observations.

Conclusions：

This investigation indicates that complement-activated IgGs, especially those in synergy of the clones targeting the CysR and CTLD1 domain, play a pivotal role in the pathogenesis of pMN. IgG1/3 subclasses of anti-PLA2R1 antibody seems instrumental in mediating podocyte injury.

Key words：

Membranous nephropathy, PLA2R1, complement activation.

4

The Effect of Non-Decaffeinated Coffee and and Decaffeinated Coffee After Induction of High-Purine Diet on Kidney Superoxide Dismutase (SOD) Levels in The Hyperuricemic Rats (Rattus norvegicus)

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Background :
Nowadays, hyperuricemia is often associated with various diseases including kidney diseases. Hyperuricemia can be secondary to impaired glomerular filtration rate (GFR). In this case, hyperuricemia can precede the development of kidney disease. Previous experimental studies showed that hyperuricemia can cause significant kidney damage, characterized by ischemia and inflammation, this condition is accompanied by increased levels of oxidative stress. Superoxide dismutase (SOD), is one of the main intracellular antioxidants that plays an essential role in the inflammation process. Coffee is a beverage that is known to have an effect in reducing uric acid levels, but there were no studies proven that the decrease in uric acid levels comes from the caffeine content in coffee. So, this study aims to compare the effects of non-decaffeinated coffee and decaffeinated coffee on kidney superoxide dismutase (SOD) levels in the hyperuricemic rats (Rattus norvegicus). Also, this study aims to prove whether hyperuricemia is related to the progression of kidney disease or not (indicated by kidney SOD levels).
Methods :
This study used a quasi-experimental method with a post-test-only control group design. This research was conducted in the laboratory of nutrition, at Gadjah Mada University for 2 months. This research used 24 male Wistar strain rats aged 1-2 months with BW of 100-150 grams. Rats were divided into four groups: normal (N), control (C), treatment 1 (T1), and treatment 2 (T2). All groups were given fed ad libitum for 1 month. The control group (C) was given 700 mg/kg BW/day of beef broth (high-purine diet), the treatment 1 group (T1) was given 700 mg/kg BW/day of beef broth + 144 mg/200 g BW/ day of non-decaffeinated coffee, and the treatment 2 group (T2) was given 700 mg/kg BW/day of beef broth + 144 mg/200 g BW/ day of decaffeinated coffee. The normal group (N) was only given fed ad libitum. At the end of the research, rats were terminated. SOD levels in the kidney were measured. ANOVA with the bonferroni post-hoc test was used in statistical analysis.
Results :
The mean of SOD (%) in normal group (N), control group (C), treatment 1 (T1), and treatment 2 (T2) consecutively were 70.22 ± 2.41, 23.50 ± 4.60, 43.99 ± 5.43, and 59.84 ± 3.98. The result showed that non-decaffeinated coffee and decaffeinated coffee are both significantly increase the kidney SOD levels ($p<0.05$).
Conclusion:
The results of the study showed that both decaffeinated coffee and non-decaffeinated coffee could reduce uric acid levels, these results also indicate that the decrease in uric acid levels was not caused by the caffeine content in coffee, but by other ingredients. The decrease in kidney SOD levels in the control group (C) also shows that the increase in uric acid levels is followed by a decrease in kidney SOD levels, so it could be concluded that the condition of hyperuricemia is related to the progression of kidney disease which is characterized by a decrease in the kidney SOD levels.
Key words :
Hyperuricemia, Kidney Superoxide Dismutase, Non-Decaffeinated Coffee, Decaffeinated Coffee, Kidney Disease.

5

AST-120 improved uremic pruritus by lowering indoxyl sulfate and inflammatory cytokines in hemodialysis patients

AST-120 透過降低血液透析患者的硫酸吲哚酚和發炎細胞因子來改善尿毒搔癢症

Chia-Chao Wu[1], Ya-Chung Tian[2], Chien-Lin Lu[3], Ming-Ju Wu[4], Paik-Seong Lim[5], Yi-Wen Chiu[6], Ko-Lin Kuo[7], Shou-Hsuan Liu[2], Yu-Ching Chou[8], Chien-An Sun[9], Yi-Chou Hou[10] and Kuo-Cheng Lu[7].
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Background：
Pruritus is a common and distressing symptom that affects patients with chronic kidney disease. The concentration of protein bounded uremic toxin was associated with the uremic pruritus. The aim is to assess the efficacy of AST-120 for uremic pruritus in hemodialysis patients.
Methods：
The participants were enrolled and then divided into the AST-120 treatment group and control group with a ratio of 2:1. All participants underwent pre-observation screenings two weeks before the study with three visits. In the treatment phase (week 1 to week 4), the treatment group added 6g/day of AST-120 along with routine anti-pruritic treatment. Visual analog scale(VAS) and biochemical parameters were measured.
Results：
The VAS score began to be lower in the AST-120 treatment group after the 5th visiting ($p < 0.05$). The reduction in indoxyl sulfate (IS) at 5th week along with TNF-alpha. The reduction ratio of indoxyl sulfate correlated with reduction of parathyroid hormone.
Conclusions：
This study has demonstrated that the four-week treatment of AST-120 decreased the severity of uremic pruritus in patients with ESRD. The concentration of IS and TNF-alpha decreased in the AST-120 treatment group. The reduction of iPTH correlated with the reduction of IS in the AST-120 treatment.
Key words：
protein bounded uremic toxin, uremic pruritus, AST-120, indoxyl sulfate, end-stage renal disease.

6

Prevalence of anxiety and depression in hemodialysispatients of end stage renal disease

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Objective:

To determines the frequency of years and mean duration of dialysis of 3.22±1.42 depression and anxiety in patients undergoing hemodialysis.

Methodology:

This descriptive cross sectional study was done at neurology department of Jinnah Postgraduate Medical Center (JPMC) Karachi. Patients undergoing hemodialysis (HD) were assessed for depression and anxiety by HADS (Hospital anxiety and depression) scale.

Results :

The study had 103 patients undergoing HD with mean age of 54.30±4.95 years. Anxiety and depression was found to be 28% and 33%, respectively and had association with age and dialysis duration.

Conclusion:

Anxiety and depression are common in HD patients. Timely screening, early management and social support are imperative for favorable outcome. (Rawal Med J 202;46:844- 847).

Keywords: Depression, anxiety, hemodialysis, end stage renal disease.

7

Risk Factors of Rehospitalization in Chronic Kidney Disease Patients: An Observational Study

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ABSTRACT

Objective: The objective of the study was to determine the factors associated with early rehospitalization among patientswith chronic kidney disease.

Methods: This was a case-control study conducted at the Department of Nephrology, The Kidney Centre, Postgraduate Training Institute, Karachi, Pakistan. Each group (rehospitalization and no rehospitalization) had 63 subjects. The subjectswere deemed eligible if they were known cases of chronic kidney disease, above 30 years of age, either gender, cases with rehospitalization within 30 days of discharge from the hospital, and controls with no rehospitalization within 30 daysof discharge from the hospital. Odds ratio was calculated to observe the strength of association between factors andrehospitalization.

Results: A total of 126 patients were enrolled. The mean age of patients in cases and controls was 69.5 ± 6.7 and 62.0 ± 9.6 years, respectively. Diabetes mellitus and heart failure were more common in the cases cohort in comparison with controls (69.8% vs. 34.9% and 50.8% vs. 7.9%, respectively). Among cases, mean serum hemoglobin and albumin lev-els were statistically lower in contrast to controls (10.0 ± 0.8 vs. 12.2 ± 1.0 g/dL, $P < .0001$ and 3.0 ± 0.6 versus 3.9 ± 0.5 mg/dL, respectively, $P < .0001$). Serum creatinine level was significantly higher in cases than in controls (2.8 ± 0.4 vs. 1.7 ± 0.3 mg/dL, respectively, $P < .0001$). The multivariate association of comorbidities with rehospitalization of chronickidney disease patients was studied and found significant for diabetes mellitus (OR 7.07, CI 2.73-18.29, $P < .0001$) and heart failure (odds ratio 18.72, CI 5.72-61.25, $P < .0001$).

Conclusion: The study showed that serum hemoglobin and albumin were significantly lower in rehospitalized cases.Furthermore, diabetes mellitus and heart failure were observed as significant risk factors for early rehospitalization.

Keywords: Chronic kidney disease, complications, early rehospitalization, Pakistan