negative charge, as a component of electric membrane on podocyte, prevents albuminuria loss. Podocyte injury is a key manifestation of proteinuric glomerulonephropathies, which, if extensive, leads to progressive glomerulosclerosis and end-stage kidney disease. The aim of this study was to investigate the effect of podocalyxin expression induced by lopride on podocytes.

**Methods:** Incubated MPC cells were divided into two groups: control group; lopride group (100 mg/ml). The expression of podocalyxin on podocytes was detected by immunofluorescence. Cell apoptosis was determined by flow cytometry with Annexin V-FITC/PI double staining.

**Results:** In the lopride-treated group, expression of podocalyxin was decreased compared with control group (P < 0.05). Apoptosis was increased compared with control group (P < 0.05).

**Conclusion:** The reduction in podocalyxin expression induced by lopride on podocytes contributes to albuminuria loss and podocyte injury, which relates to CIN progressing to chronic kidney disease.

http://dx.doi.org/10.1016/j.hkjn.2015.09.108

**0199**
Effect of Rhododendron Molle G. Don on Renal Interstitial Fibrosis in UUO Model
J. Xiong, S. Jin, C. Zhang
Department of Nephrology, Union Hospital, Tongji Medical College, Huazhong University of Science & Technology, Wuhan, China

**Objective:** Rhododendron Molle G. Don (rhododendron) was found to be effective on the treatment of rheumatoid arthritis and chronic glomerulonephritis. The aim of this study is to explore the role of Rhododendron on renal interstitial fibrosis (RIF) and the potential mechanism.

**Methods:** Unilateral ureteral obstruction (UUO) was performed as RIF model. Mice were randomly divided into 6 groups (n = 8): sham-operation group (Sham), UUO group, UUO mice treated with low dose or high dose of rhododendron leaf (U+L, U+R), UUO mice treated with low dose or high dose of rhododendron root (U+r, U+r). All mice were executed after 7 days of operation, and renal tissues were collected for pathological examination. Tubulointerstitial damage index (TDI) was measured by Masson’s trichrome staining. The expression of TGF-β1, fibronectin (FN) and α-SMA in renal interstitial was analyzed by immunohistochemical method.

**Results:** TDI was obviously increased in UUO mice, which indicated UUO model was successful. The expression of TGF-β1, FN and α-SMA in interstitial was also increased in UUO mice. However, more severe interstitial damage and higher level of TGF-β1, FN and α-SMA was found in UUO mice treated with rhododendron root, whatever high dose or low dose. The results showed that rhododendron root was harmful for renal interstitial, and TGF-β1 signaling involved in this process. But there was no difference in TDI and fibrotic indexes in UUO mice, those were treated with or without rhododendron leaf.

**Conclusion:** Rhododendron root was reported to be useful to treat inflammatory injury mediated by immune complex. However, in this study we found rhododendron root might aggravate interstitial fibrosis in UUO model, and the side effect of rhododendron root on interstitial damage should be paid attention. The ingredients of rhododendron are complex and not entirely clear, so more examination and evidence should be analyzed before rhododendron is used in clinic.

http://dx.doi.org/10.1016/j.hkjn.2015.09.109

**0204**
Core Fucosylation Regulates Process of Pericyte-Myoﬁbroblast Transition
Yiyao Deng, Nan Wang, Hongli Lin
The First Affiliated Hospital of Dalian Medical University, Dalian, China

**Objective:** Renal interstitial ﬁbrosis (RIF) is a common pathological process of CKD. Unfortunately, there is no satisfactory therapy to reverse this process. Recently, increasing evidences show that pericytes are the major sources of myoﬁbroblast during RIF. Pericyte transition involved in multiple signaling pathway cross-talk, such as PDGF pathway, TGFβ pathway, and VEGF pathway, and vital protein of these pathways are posttranslational modiﬁed by Fucosyltransferase 8 (Fut8). So we considered that Fut8 may play an important role in this process.

**Methods:** Mice kidneys were diced, incubated at 37°C for 45 minutes with liberase and Dnase in Hank’s balanced salt solution. After centrifugation, cells were resuspended, and filtered by screen mesh. Then we used 42% percoll working solution to remove glomerulus and cell debris. Cells were marked by PDGFRα-PE, and isolated by FACS. Then cells were identiﬁed by immunofluorescence staining of E-cadherin, CD31, α-SMA, PDGFRβ and CD73. Passage 2 to passage 5 pericytes were used in experiments, and pericytes were stimulated by TGFβ1 for 24 h, 48 h for further observation. Mice UUO model was applied for in vivo observation.

**Results:** Comparing with or without percoll in pericytes isolation, we found former can obtain a higher sorting positive percentage. Cell identiﬁcation showed that E-cadherin−, CD31−, α-SMA−, PDGFRβ+ and CD73+. After addition of TGFβ1, increasing expressions of α-SMA, Fut8, LCA of pericytes were observed. But expression of α-SMA, Fut8, LCA were greatly reduced under TGFβ1 stimulation after knockdown of Fut8 by transiently transfected Fut8-siRNA. We also found increasing co-localized expression of Fut8, LCA, PDGFRβ, α-SMA in UUO model.

**Conclusion:** Fut8 plays a vital role in process of pericyte transition, and block its function of glycosylated modiﬁcation can alleviate pericyte transition. But the underlying mechanism of this phenomenon need further elaborate. Prospectively, it may hint a novel therapeutic target of RIF.

http://dx.doi.org/10.1016/j.hkjn.2015.09.110

**0215**
Targeted Disruption of the Prostaglandin E2 EP3 Receptor Attenuates 5/6 Nephrectomize Induced Renal Fibrosis
Jiahua Wu, Xu Chen, Xiaolan Chen, Yaping Fan
Department of Nephrology, Affiliated Hospital of Nantong University, Nantong, Jiangsu, China

**Objective:** To illuminate the role of prostaglandin E2 EP3 receptor on 5/6 nephrectomize induced renal fibrosis.
Methods: Wild-type (EP3+/+) and EP1 gene-deficient (EP1−/−) mice were divided into 4 groups: (1) EP3+/+ Sham-operated group; (2) EP3+/+ 5/6 nephrectomy group; (3) EP3−/− Sham-operated group; (4) EP3−/− 5/6 nephrectomy group. The serum levels of blood urea nitrogen (BUN), serum creatinine (SCr), and urinary osmolarity in mice were measured. Kidney tissues were taken and fixed in 4% phosphate-buffered paraformaldehyde, embedded in paraffin and cut into slices after eight weeks. Pathological changes of renal tissue were observed by HE and PAS-Masson staining. The expression of FN, Col1, COX2, TGF, CTGF, Erbin were detected by immunohistochemistry.

Results: Compared with the respective sham group, 5/6 nephrectomy model group had higher levels of serum creatinine and BUN but lower urine osmolarity. Whereas the changes of EP3+/+ 5/6 nephrectomy group were significantly lower than those in the EP3+/+ 5/6 nephrectomy group (P < 0.05). In 5/6 nephrectomy mice, the interstitial fibrosis including tubular atrophy, loss and dilation, inflammatory cell infiltration and interstitial matrix deposition was prominent. Compared with the respective sham group, 5/6 nephrectomy group mesangial cells proliferation and extracellular matrix significantly higher, whereas the changes of EP3+/+ 5/6 nephrectomy group were significantly lower than those in EP3+/+ 5/6 nephrectomy group (P < 0.05). Immunohistochemistry technique showed that, compared with the each Sham-operated group, the expressions of FN, COX1, COX2, TGF, CTGF and Erbin in renal tissues significantly higher in 5/6 nephrectomy group, whereas the FN values in EP3+/+ 5/6 nephrectomy group were significantly lower than those in EP3+/+ 5/6 nephrectomy group (P < 0.05).

Conclusion: Targeted disruption of the EP3 can attenuate the pathological state of 5/6 nephrectomize induced renal fibrosis, and it implying that EP3 may take an important role in the process of renal fibrosis.

http://dx.doi.org/10.1016/j.hkjn.2015.09.111

0246
PPARβ augments IL-1β-induced Expression of COX2 and Production of PGE2 in Human Mesangial Cells via Sirt1 Pathway

Rong Cao 1, Youfei Guan 2
1The First Affiliated Hospital of Shenzhen University, Shenzhen, Guangdong, China 2Dalian Medical University, Dalian, Liaoning, China

PPARβ, a ligand-activated nuclear receptor, plays important roles in the regulation of lipid and glucose metabolism, cell proliferation and differentiation. Growing evidence indicates that PPARβ/δ also exerts anti-inflammatory properties by suppressing proinflammatory cytokines production. Previously, our studies showed that PPARβ/δ was expressed in human mesangial cells. However, the role of PPARβ/δ in human mesangial cell inflammation has not been elucidated. Excessive production of prostaglandin resulting from increased COX-2 expression is involved in various pathophysiological conditions, such as cancer and inflammation. Recently, it has been reported that activation of PPARβ/δ up-regulates the expression of COX-2 and increases production of PGE2 in tumor cells. The present study aimed to investigate the potential role of PPARβ/δ in COX-2 expression and PGE2 synthesis in human mesangial cells, particularly in the presence of IL-1β. Our studies showed that PPARβ/δ was functionally expressed in human mesangial cells, and its expression was increased by IL-1β in human mesangial cells. Treatment with GW9642, a selective agonist of PPARβ/δ, or viral-mediated overexpression of PPARβ/δ significantly upregulated expression and increased cellular PGE2 content in human mesangial cells. Importantly, activation of PPARβ/δ or PPARβ/δ overexpression significantly augmented IL-1β-induced COX-2 expression and PGE2 production in human mesangial cells. In addition, PPARβ/δ increased the expression of Sirt1, while knockdown of Sirt1 gene or inhibition Sirt1 activity by nicotinamide and EX527 partly abolished the effect of PPARβ/δ on IL-1β-induced expression of COX-2 and production of PGE2 in human mesangial cells. Taken together, PPARβ/δ augmented IL-1β-induced expression of COX-2 and production of PGE2 in human mesangial cells, at least in part, via the Sirt1 pathway. More importantly, PPARβ/δ may represent a novel target for the treatment of renal inflammatory diseases.

http://dx.doi.org/10.1016/j.hkjn.2015.09.113

0258
Effect of Protein Phosphatase 2AC and Norcantharidin on Smad3 Linker Region Phosphorylation in HK-2 Cells

Ying Li 1, Z. Xiao 1, Z. Y. Zhao, Q. X. Hu, H. Liu, J. Li, S. B. Duan, L. Sun, Y. M. Peng, F. Y. Liu
1The Second Xiangya Hospital of Central South University, Changsha, China

Objective: We assessed the role of PP2Ac and NCTD on the phosphorylation of Smad3 linker region in human renal proximal tubule cell lines (HK-2 cells).

http://dx.doi.org/10.1016/j.hkjn.2015.09.112