negative charge, as a component of electric membrane on podocyte, prevents albuminuria loss. Podocyte injury is a key manifestation of proteinuric glomerulopathies, 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 lipomide on podocytes. 

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

**Results:** In the lipomide-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 lipomide on podocytes contributes to albuminuria loss and podocyte injury, which relates to CIN progressing to chronic kidney disease.

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**0215**

Core Fucosylation Regulates Process of Pericyte-Myofibroblast Transition

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**Objective:** Corefucosylation is a common pathological process of CRD. 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. 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 sorted by PDGFRα+ and α-SMA signal. We also found increasing co-localized expression of Fut8, LCA, PDGFRα+, α-SMA in UUO model. But expression of α-SMA, Fut8, LCA were greatly reduced under TGFβ1 stimulation after knockdown of Fut8 by transiently transfected Fut8 siRNA. 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, PDGFRα+, 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.

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**0220**

Targeted Disruption of the Prostaglandin E2 EP3 Receptor Attenuates 5/6 Nephrectomize Induced Renal Fibrosis

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**Objective:** To illuminate the role of prostaglandin E2 EP3 receptor on 5/6 nephrectomize induced renal fibrosis.

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