Conclusion: Rxra is tonicity responsive and capable of interacting with Nfat5 to negatively regulate Nfat5 activity and to exert important regulatory roles in osmoregulation, osmoadaptation and renal function.

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0156 Lrp6 Mediated Wnt Signaling Acts Through Primary Cilia Leading to Renal Defects
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Objective: Congenital anomalies of the kidney and urinary tract (CAKUT) are one of the most common birth defects. Our previous results indicate that conventional deletion of the key Wnt receptor of Lrp6 leads to a spectrum of renal hypoplastic and cystic kidney disorders, which are major phenotypes for CAKUT. Primary cilia are cellular organelle existing in surface and serving as signalling centre for multiple pathways. We found cilia are defect in Lrp6-/- kidney, but the underlying mechanism is unclear. We aim to discover the roles of Lrp6 in cilia-mediated pathway which disrupts cellular proliferation, differentiation and organogenesis in renal defects.

Methods: Lrp6 beta-geo mice were raise and mouse embryonic fibroblasts (MEFs), murine inner medullary collecting duct (mIMCD3) was purchased from ATCC and cultured in UC Davis and Nanfang Hospital. Scanning electronic microscope (SEM) and immunofluorescence staining were performed according to standard protocol.

Results: (1) The embryonic kidney samples from E18.5 Lrp6-/- mice were examined under scanning electronic microscope (SEM). Primary cilia in mutant kidneys exhibit extra slim/long (up), ungrown, short or curved cilia (bottom) compared with littermate control. The picture in big frame in upper-left is the magnification of the picture in small frame (unpublished preliminary data). (2) The active state of phosphorylated Lrp6 (Tp1479) is co-localized with cilia markers of acetylated-tubulin and g-tubulin at basal body in MEFs and mIMCD3 cells. Phosphorylated Lrp6 (green) is expressed in the basal body in control MEFs, but absent in Lrp6-/- MEFs. Phosphorylated Lrp6 (green) is co-localized with acetylated-tubulin (red) and basal body marker of g-tubulin (red) in mIMCD3 cells (unpublished preliminary data).

Conclusion: Lrp6 mediated Wnt signaling may act through primary cilia with undiscovered mechanism which cause genetic renal defects and ciliopathy in CAKUT.

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0161 Reversal of Epithelial to Mesenchymal Transition Following Relief of Unilateral Ureteral Obstruction In the Rat
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Objective: Renal fibrosis begins with renal tubular epithelial mesenchymal transition (EMT); the progression thereafter depends upon a number of fibrotic factors. Unilateral ureteral obstruction (UUO) is a well-described model of EMT. We used an improved reversible unilateral ureteral obstruction (RUO) model to investigate whether a progressive renal injury model of EMT could be reversed into the opposite direction, into mesenchymal-epithelial transition (MET) after relief of UUO in rats.

Methods: Rats were subjected to UUO or sham operation and the obstruction was removed five days later (or was left in place). Rats developed EMT after reversal of 2 or 4 weeks of ureteral obstruction as assessed by the expressions of fibrotic factors, EMT and MET markers in this post-obstructive model.

Results: We found a significant decrease in the kidney weight and renal cortical thickness in the RUO group compared with the sham groups. This rise in the RUO group was significantly reduced. The elevated level of TGF-b1, TGF-b receptor and core fucosylation in the UUO group was significantly reduced in the RUO groups. The EMT markers staining showed results parallel to those of TGF-b1 expression levels. In addition, UUO rats exhibited pronounced inflammatory and intrinsically proliferative cellular responses, and ultimately fibrosis. By comparison, RUO mice had more controlled and measured intrinsic and extrinsic responses to EMT with return to MET within several weeks after release of ureteral obstruction.

Conclusion: Our findings provide a model that allows investigation of the fibrotic factors during reversal of EMT that contribute to the development of fibrosis. EMT of the progressive renal injury could be actively reversed into MET and renal architecture is better maintained throughout injury and recovery from injury after relief of UUO in rats.

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0163 Effect of Podocalycin Expression Induced by Iopromide on Podocytes
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Objective: Contrast-induced renal tubular cells apoptosis represents a key mechanism of contrast-induced nephropathy (CIN). So most episodes of CIN are self-limiting and resolve within 1–2 weeks, but in some cases, reduced renal function progresses to chronic kidney disease. Albuminuria was observed after injection of contrast media in animal studies, which last until the function of renal tubular epithelial cell recover. Podocalycin, which has