

IN BRIEF

BIOPSY

The length of renal cilia during renal injury is associated with the extent of damage and rate of repair. Verghese *et al.* found that, in biopsy samples of human renal transplants in which acute tubular necrosis had occurred, cilium length had more than doubled at approximately 1 week after injury. In later biopsies, cilium length returned to normal, a trend that correlated with recovery of normal kidney function.

Original article Verghese, E. *et al.* Renal primary cilia lengthen after acute tubular necrosis. *J. Am. Soc. Nephrol.* **20**, 2147–2153 (2009)

DEVELOPMENT

Integrins are adhesion receptors that regulate interactions between cells and components of the extracellular matrix and may have important roles in organ morphogenesis by means of modifying cell shape, growth and motility. Zhang *et al.* have now shown that, in mice, $\beta 1$ integrins regulate ureteric bud branching morphogenesis by mediating growth-factor-dependent signals involved in this process during development.

Original article Zhang, X. *et al.* $\beta 1$ integrin is necessary for ureteric bud branching morphogenesis and maintenance of collecting duct structural integrity. *Development* **136**, 3357–3366 (2009)

TRANSPLANTATION

By applying protein microarray technology to pediatric renal transplant recipients, Sutherland and co-workers have identified 36 potentially immunogenic non-human-leukocyte-antigen antibodies. Protein kinase C (PKC) ζ had the strongest mean signal intensity and was chosen for additional analysis. Increased levels of antibodies against PKC ζ was associated with poor allograft survival. The authors suggest that PKC ζ is a marker of severe allograft injury, rather than being a pathogenic antibody.

Original article Sutherland, S. M. *et al.* Protein microarrays identify antibodies to protein kinase C ζ that are associated with a greater risk of allograft loss in pediatric renal transplant recipients. *Kidney Int.* doi:10.1038/ki.2009.384

GENETICS

Dysfunction of the electrogenic chloride–proton exchanger CIC-5—encoded by *CLCN5* and expressed in the proximal tubule and collecting duct—has been implicated in the pathogenesis of Dent disease. Grand *et al.* assessed the functional effects of *CLCN5* mutations in Dent disease and identified two types of mutant with distinct mechanisms of CIC-5 impairment. Type I mutants undergo complex glycosylation at the cell surface but have reduced electrical activity, whereas type II mutants do not induce currents, owing to defective N-glycosylation.

Original article Grand, T. *et al.* Novel *CLCN5* mutations in patients with Dent's disease result in altered ion currents or impaired exchanger processing. *Kidney Int.* **76**, 999–1005 (2009)