

*Nature Reviews Nephrology* 9, 433 (2013); published online 2 July 2013;  
 doi:10.1038/nrneph.2013.119;  
 doi:10.1038/nrneph.2013.120;  
 doi:10.1038/nrneph.2013.121;  
 doi:10.1038/nrneph.2013.122

## IN BRIEF

### POLYCYSTIC KIDNEY DISEASE

#### Role of SIRT1 in ADPKD pathophysiology

SIRT1 signalling is involved in the pathogenesis of autosomal dominant polycystic kidney disease (ADPKD) and may represent a new therapeutic target for this disease, say researchers. Zhou *et al.* found that Sirt1 levels were upregulated in a mouse model of ADPKD. Deletion or inhibition of Sirt1 delayed renal cyst formation. The researchers propose that Sirt1 regulates epithelial cell proliferation and death via pathways involving Rb and p53.

**Original article** Zhou, X. *et al.* Sirtuin 1 inhibition delays cyst formation in autosomal-dominant polycystic kidney disease. *J. Clin. Invest.* doi:10.1172/JCI64401

### LUPUS NEPHRITIS

#### Termination of a trial of ocrelizumab for lupus nephritis

An analysis of efficacy and safety data from a phase III, randomized controlled trial of ocrelizumab for class III/IV lupus nephritis has found that this treatment does not significantly improve renal response rates compared with placebo and is associated with a higher rate of serious infections in patients receiving mycophenolate mofetil. The trial had been terminated early because of the increased risk of infections observed in the treatment group.

**Original article** Mysler, E. F. *et al.* Efficacy and safety of ocrelizumab in active proliferative lupus nephritis: results from the randomized, double-blind phase III BELONG study. *Arthritis Rheum.* doi:10.1002/art.38037

### POLYCYSTIC KIDNEY DISEASE

#### Pathogenic role for microRNA cluster in PKD

New findings suggest that the oncogenic microRNA cluster, miRNA-17~92, promotes the growth of kidney cysts in mouse models of polycystic kidney disease (PKD). Patel *et al.* found that miRNA-17~92 is upregulated in mice with PKD. Kidney-specific overexpression of the cluster induced kidney cysts, whereas kidney-specific inactivation in a model of PKD slowed cyst growth, improved renal function, and prolonged survival. The researchers suggest that miRNA-17~92 mediates these effects by stimulating proliferation and by repressing *Pkd1* and *Pkd2*.

**Original article** Patel, V. *et al.* miR-17~92 miRNA cluster promotes kidney cyst growth in polycystic kidney disease. *Proc. Natl Acad. Sci. USA* doi:10.1073/pnas.1301693110

### STEM CELLS

#### Direct reprogramming of adult epithelial kidney cells

The reprogramming of one differentiated cell type to another can be achieved by enforcing the expression of key transcription factors. To investigate whether adult proximal tubule cells can be transcriptionally reprogrammed to become nephron progenitors, Hendry *et al.* used a combinatorial screening approach to identify a pool of six genes that activated a network of nephron progenitor genes in the adult proximal tubule (HK2) cell line. HK2 cells transduced with these genes underwent epithelial-to-mesenchymal transition and showed a differential capacity to contribute to the nephron progenitor compartment of a developing kidney *ex vivo*.

**Original article** Hendry, C. E. *et al.* Direct transcriptional reprogramming of adult cells to embryonic nephron progenitors. *J. Am. Soc. Nephrol.* doi:10.1681/ASN.2012121143