# **IN BRIEF**

# **GENETICS**

## Genetic links between diabetes and albuminuria

Loci that are associated with albuminuria in diabetes mellitus have been identified by GWAS meta-analysis of albuminuria traits in the general population. An association between *CUBN* and urinary albumin-to-creatinine ratio (UACR) was replicated, and gene-by-diabetes interactions were detected at the *HS6ST1* and *RAB38/CTSC* loci, where a genetic effect on the UACR was found in those with diabetes. *Rab38*-/- rats had higher urinary albumin concentrations and reduced levels of megalin and cubilin at the proximal tubule cell surface compared to controls, and the expression of *RAB38* was higher in the tubuli of patients with diabetic kidney disease than controls.

 $\begin{tabular}{ll} \textbf{ORIGINAL ARTICLE} Teumer, A. \it{et al.} Genome-wide association studies identify genetic loci associated with albuminuria in diabetes. \it{Diabetes} \hdots http://dx.doi.org/10.2337/db15-1313$ 

## **■** IMMUNOSUPPRESSION

### Modulation of BK polyomavirus replication

The effects of immunosuppressive drugs on BK polyomavirus (BKV) replication have been compared in a new study. Sirolimus inhibited viral replication up to 24 h postinfection, but not thereafter, and similar results were found for other mTOR inhibitors and ciclosporin A. Conversely, tacrolimus — a calcineurin inhibitor — activated BKV replication. The effects of tacrolimus and sirolimus were mediated through FKBP12 in renal tubular epithelial cells. The researchers propose that these data provide a rationale for clinical trials aimed at reducing the risk of BKV replication in renal transplantation.

**ORIGINAL ARTICLE** Hirsch, H. H. et al. BK polyomavirus replication in renal tubular epithelial cells is inhibited by sirolimus, but activated by tacrolimus through a pathway involving FKBP-12. Am. J. Transplant. http://dx.doi.org/10.1111/ajt.13541

## BASIC RESEARCH

#### TMEM107: a new ciliopathy transition zone gene

A bioinformatics-based analysis has identified *TMEM107* as a candidate transition zone (TZ) ciliary gene that is mutated in oral–facial–digital and Joubert syndromes. Functional analyses performed in *C. elegans* showed that TMEM107 contributes to ciliary composition and function in a redundant manner with NPHP-4, and regulates cilium integrity, TZ docking, and assembly of membrane-to-microtubule (Y-link) connectors. TMEM107 localizes to layer three of the 'MKS module' in the TZ, where it organizes recruitment of ciliopathy proteins.

**ORIGINAL ARTICLE** Lambacher, N. J. et al. TMEM107 recruits ciliopathy proteins to subdomains of the ciliary transition zone and causes Joubert syndrome. *Nat. Cell Bio.* http://dx.doi.org/10.1038/ncb3273

#### PROGENITOR CELLS

#### Promotion of nephron progenitor cell self-renewal

BMP, FGF, and WNT signalling regulate self-renewal of nephron progenitor cells (NPCs). A molecular analysis by Muthukrishnan *et al.* to delineate these pathways found that BMP7 activates TAK1 and JNK, resulting in JUN phosphorylation, *Myc* and *Ccnd1* transcriptional regulation, and NPC proliferation. JUN is regulated by BMP7, whereas its partner FOS is regulated by FGF9. The researchers show that BMP7 and FGF9 both regulate *AP-1* transcription, cell cycle progression, and NPC proliferation, and highlight the cooperation between these two NPC self-renewal pathways.

ORIGINAL ARTICLE Muthukrishnan, S. D. et al. Concurrent BMP7 and FGF9 signalling governs AP-1 function to promote self-renewal of nephron progenitor cells. Nat. Commun. http://dx.doi.org/10.1038/ncomms10027