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## IN BRIEF

### HYPERTENSION

#### Predictive biomarkers identified for renal denervation

In the search for potential biomarkers to predict the success of renal sympathetic denervation in patients with resistant hypertension ( $n=55$ ), Dörr *et al.* measured the serum levels of key factors associated with endothelial function: fms-like tyrosine kinase-1 (sFLT-1), intercellular cell adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1). Baseline levels of sFLT-1, ICAM-1 and VCAM-1 were all significantly higher in those who achieved a reduction in systolic blood pressure of  $>10$  mmHg 6 months after renal sympathetic denervation ( $n=46$ ) than in nonresponders, suggesting these factors might be useful clinical biomarkers.

**Original article** Dörr, O. *et al.* Soluble fms-like tyrosine kinase-1 and endothelial adhesion molecules (intercellular cell adhesion molecule-1 and vascular cell adhesion molecule-1) as predictive markers for blood pressure reduction after renal sympathetic denervation. *Hypertension* doi:10.1161/hypertensionaha.113.02266

### BASIC RESEARCH

#### Anoctamin 1—role in proton secretion and protein resorption

New research has examined the role of  $Ca^{2+}$ -dependent  $Cl^-$  transport in the kidney, focusing on the channel anoctamin 1 (Ano1). Immunohistochemistry revealed that Ano1 is predominantly expressed in the proximal tubular epithelium. Indeed, tubular Ano1 knockout led to proteinuria whereas selective knockout in podocytes did not have an effect on renal function, suggesting that Ano1 has a role in protein reabsorption rather than in the glomerulus. Low pH activated Ano1 currents, implicating Ano1-mediated  $Cl^-$  secretion in effective  $H^+$  transport by the vacuolar-ATPase in the kidney.

**Original article** Faria, D. *et al.* The calcium-activated chloride channel anoctamin 1 contributes to the regulation of renal function. *Kidney Int.* doi:10.1038/ki.2013.535

### CHRONIC KIDNEY DISEASE

#### Indices of insulin sensitivity put to the test

A study in  $>1,000$  men (mean age 70 years) has confirmed that insulin sensitivity indices (ISIs) perform similarly in those with or without chronic kidney disease. Of the ISIs, oral glucose tolerance tests performed better than fasting measurements compared with hyperinsulinaemic euglycaemic glucose clamp (HEGC; the gold-standard measurement of glucose disposal), which is not subject to renal function bias. Furthermore, neither HEGC nor the ISIs were associated with mortality in this study, regardless of kidney function. Accordingly, kidney function might be independent of insulin sensitivity-related outcomes.

**Original article** Jia, T. *et al.* Validation of insulin sensitivity surrogate indices and prediction of clinical outcomes in individuals with and without impaired renal function. *Kidney Int.* doi:10.1038/ki.2014.1

### DIABETES

#### ACE2 overactivity and hyperfiltration in diabetic nephropathy

Induction of diabetes in wild-type mice has revealed increased levels of angiotensin converting enzyme 2 (ACE2), which is involved in regulation of the renin–angiotensin–aldosterone system. Diabetes in these animals led to increases in tubular pressure, hyperfiltration and renal hypertrophy. ACE2-knockout mice and those given ACE2 inhibitors resisted this phenotype, and did not have increased creatinine clearance despite a high-protein diet—implicating ACE2 in diabetic nephropathy.

**Original article** Tikellis, C. *et al.* Angiotensin converting enzyme 2 and hyperfiltration associated with diabetes. *Am. J. Physiol. Renal Physiol.* doi:10.1152/ajprenal.00264.2013