IN BRIEF

TRANSPLANTATION

The –765G>C polymorphism in the gene promoter of cyclo-oxygenase 2 reduces promoter activity and prostaglandin E2 (PGE2) production, potentially influencing graft survival. Courivaud *et al.* assessed the functional effect of this polymorphism by analyzing PGE2 serum levels in renal transplant recipients. Patients with the polymorphism had significantly lower PGE2 levels and a higher risk of graft loss than patients without the polymorphism.

Original article Courivaud, C. *et al.* Influence of cyclooxygenase-2 (COX-2) gene promoter polymorphism –765 on graft loss after renal transplantation. *Am. J. Transplant.* **9**, 2752–2757 (2009)

HYPERTENSION

Angiotensin (Ang)-converting enzyme (ACE2) cleaves Ang II to form Ang-(1–7). Wysocki and colleagues examined whether human recombinant ACE2 (rACE2) could prevent hypertension caused by Ang II infusion in mice. After infusion, rACE2 degraded Ang II and normalized blood pressure. The researchers propose ACE2 as a potential therapeutic target for the treatment of hypertension.

Original Article Wysocki, J. *et al.* Targeting the degradation of angiotensin II with recombinant angiotensin-converting enzyme 2. *Hypertension* **55**, 90–98 (2010)

INTERSTITIAL DISEASE

Active vitamin D is thought to slow progression of chronic kidney disease. Tan and colleagues investigated whether treatment with paricalcitol and trandolapril, either individually or in combination, had protective effects in a mouse model of obstructive nephropathy. Both monotherapies reduced markers of fibrosis and inflammation. Combination therapy had additive effects in reducing renal scar formation.

Original article Tan, X. *et al.* Combination therapy with paricalcitol and trandolapril reduces renal fibrosis in obstructive nephropathy. *Kidney Int.* **76**, 1248–1257 (2009)

HYPERTENSION

Circadian clock malfunctions are linked to the pathogenesis of a variety of diseases. A new study in *Cry*-knockout mice, which lack core clock components and have high levels of aldosterone, links circadian clock malfunction with salt-sensitive hypertension. Doi *et al.* identified type VI 3β-hydroxyl-steroid dehydrogenase as a risk factor for hypertension that is linked to circadian functions and regulates aldosterone production.

Original article Doi, M. *et al.* Salt-sensitive hypertension in circadian clock-deficient *Cry*-null mice involves dysregulated adrenal Hsd3b6. *Nat. Med.* **16**, 67–74 (2009)