Nature Reviews Nephrology **7**, 676 (2011); published online 8 November 2011;
doi:10.1038/nrneph.2011.162;
doi:10.1038/nrneph.2011.163;
doi:10.1038/nrneph.2011.164;
doi:10.1038/nrneph.2011.165

IN BRIEF

PEDIATRICS

Improving growth in infants on peritoneal dialysis

The use of biocompatible peritoneal dialysis fluid, gastrostomy feeding and growth hormone is associated with improved linear growth in very young children with chronic kidney disease on peritoneal dialysis. Rees *et al.*, for the International Pediatric Peritoneal Dialysis Network registry, came to these conclusions following their analysis of 153 children in 18 countries who commenced chronic peritoneal dialysis at <2 years of age.

Original article Rees, L. *et al.* Growth in very young children undergoing chronic peritoneal dialysis. *J. Am. Soc. Nephrol.* doi:10.1681/ASN.2010020192

DIALYSIS

Explaining racial disparities in survival on dialysis

Survival is better in African Americans on dialysis than in white patients, even though in the general population African Americans have higher mortality and are at higher risk of renal failure. Crews *et al.* report that differences in inflammation levels might partly explain this paradox. Mortality rates were lower in African Americans, but median C-reactive protein (CRP) level was similar in the two groups. When patients were split into tertiles based on CRP level, however, mortality in the higher tertiles was lower in African Americans than in white patients.

Original article Crews, D. C. *et al.* Inflammation and the paradox of racial differences in dialysis survival. *J. Am. Soc. Nephrol.* doi:10.1681/ASN.2011030305

RENOVASCULAR DISEASE

Renin–angiotensin blockade in ARVD

All patients with atheromatous renovascular disease (ARVD) should be considered for treatment with renin–angiotensin blockade (RAB) unless absolutely contraindicated, state the authors of a recent study. Chrysochou *et al.* analyzed data from 621 individuals with ARVD and found that RAB was well tolerated in most patients receiving it, even in 78% of those with bilateral renal artery stenosis of ≥60% or occlusion. Patients receiving RAB were less likely to die over the 10-year study period than those not receiving such treatment.

Original article Chrysochou, C. *et al.* Dispelling the myth: the use of renin–angiotensin blockade in atheromatous renovascular disease. *Nephrol. Dial. Transplant.* doi:10.1093/ndt/gfr496

HYPERTENSION

How do calcineurin inhibitors cause hypertension?

Hypertension mediated by tacrolimus is largely caused by activation of the renal sodium chloride cotransporter (NCC), say researchers. In wild-type mice, tacrolimus caused salt-sensitive hypertension and increased levels of phosphorylated NCC, whereas in NCC-knockout mice, it did not affect blood pressure. The hypertensive response to tacrolimus was increased In NCC-overexpressing mice. NCC-blocking drugs reversed tacrolimus-induced hypertension in mice and increased fractional chloride excretion in human renal transplant recipients.

Original article Hoorn, E. J. *et al.* The calcineurin inhibitor tacrolimus activates the renal sodium chloride cotransporter to cause hypertension. *Nat. Med.* **17**, 1304–1309