Nature Reviews Nephrology **10**, 126 (2014); published online 28 January 2014;
doi:10.1038/nrneph.2014.12;
doi:10.1038/nrneph.2014.15;
doi:10.1038/nrneph.2014.13

IN BRIEF

DIALYSIS

Loss of mobility associated with mortality in elderly patients

Impaired mobility at the time of dialysis initiation is an important risk factor for short-term mortality in elderly patients. Now, new data from a Japanese retrospective study suggest that a decline in mobility after the initiation of dialysis is also associated with reduced survival in this population. Arai and colleagues divided 202 patients aged >75 years into subgroups on the basis of their mobility at the time of initiation of dialysis and during 6 months of follow-up monitoring. They report that 24.8% of patients had impaired mobility at dialysis initiation and 68.9% of patients showed a decline in mobility whilst on dialysis. Moreover, patients who were independently mobile at dialysis initiation but whose mobility subsequently declined, and those who had impaired mobility at the onset of dialysis, had increased risks of short-term mortality compared with patients who were independently mobile at dialysis initiation and at 6 months.

Original article Arai, Y. *et al*. Decreased mobility after starting dialysis is an independent risk factor for short-term mortality after initiation of dialysis. *Nephrology (Carlton)* doi:10.1111/nep.12202

ACUTE KIDNEY INJURY

Proximal tubular cells transiently acquire STC phenotype

Researchers have postulated that scattered tubular cells (STCs) might be the origin of regenerating tubular cells after acute kidney injury. However, new data suggest that STCs are not a fixed progenitor population. Using a transgenic mouse model, Berger and colleagues showed that when STCs were irreversibly genetically labelled before ischaemia reperfusion injury, the frequency of labelled cells did not change during recovery. However, when STCs were labelled during ischaemia reperfusion injury and recovery, the frequency of STCs significantly increased. These data suggest that STCs can arise from any surviving tubular cell. "[STCs] do not represent a fixed progenitor population but rather a phenotype that can be adopted by almost any proximal tubular cell upon injury," conclude the researchers.

Original article Berger, K. *et al*. Origin of regenerating tubular cells after acute kidney injury. *Proc. Natl Acad. Sci*. doi:10.1073/pnas.1316177111

GENETICS

Genotypes and phenotypes in *WT1*-associated nephropathy

The authors of a new study suggest that all children with steroid-resistant nephrotic syndrome should be screened for mutations in the *WT1* gene. Lipska and colleagues found that among 761 patients with this disease, those with mutations in *WT1* ($n=61$) more often presented with hypertension and chronic kidney disease at diagnosis and showed faster disease progression. Missense substitutions in *WT1* that affected DNA-binding residues were associated with diffuse mesangial sclerosis and early onset of steroid-resistant nephrotic syndrome with rapid progression to end-stage renal disease (ESRD). By contrast, truncating mutations in *WT1* were associated with late onset of steroid-resistant nephrotic syndrome and the highest risk of Wilms tumour, whereas intronic mutations were most likely to cause isolated steroid-resistant nephrotic syndrome, focal segmental glomerulosclerosis and slow progression to ESRD.

Original article Lipska, B. S. *et al.* Genotype–phenotype associations in *WT1* glomerulopathy. *Kidney Int.* doi:10.1038/ki.2013.519