

IN BRIEF

 KIDNEY CANCER**IMPRINT: no survival benefit of IMA901 in RCC**

The IMPRINT phase III trial shows that combination therapy with the multipeptide cancer vaccine IMA901 plus sunitinib does not improve the survival of patients with metastatic renal cell carcinoma (RCC) over sunitinib alone. In the trial, which included 339 HLA-A*02-positive patients with previously untreated metastatic or locally advanced clear cell RCC, median overall survival was 33.17 months in the sunitinib plus IMA901 group and was not reached (33.67–not reached) in the sunitinib monotherapy group. The researchers suggest that the magnitude of immune responses needs to be improved before the vaccine can be further developed for use in RCC.

ORIGINAL ARTICLE Rini, B. I. *et al.* IMA901, a multipeptide cancer vaccine, plus sunitinib versus sunitinib alone, as first-line therapy for advanced or metastatic renal cell carcinoma (IMPRINT): a multicentre, open-label, randomised, controlled, phase 3 trial. *Lancet* [http://dx.doi.org/10.1016/S1470-2045\(16\)30408-9](http://dx.doi.org/10.1016/S1470-2045(16)30408-9) (2016)

 ACUTE KIDNEY INJURY**Role of platelet activation and NETs in renal IRI**

Platelet activation and the formation of neutrophil extracellular traps (NETs) contribute to renal ischaemia–reperfusion injury (IRI), say researchers. Jansen *et al.* show that in mice, renal IRI led to activation of platelets in the proximity of necrotic cell casts. The antiplatelet agent clopidogrel reduced IRI-induced cell necrosis and inflammation. Further investigations indicated that the platelets were activated by extracellular DNA released by necrotic tubular epithelial cells, and that this activation in turn led to increased platelet–granulocyte interactions, the formation of NETs, renal inflammation and tissue injury.

ORIGINAL ARTICLE Jansen, M. P. B. *et al.* Release of extracellular DNA influences renal ischemia reperfusion injury by platelet activation and formation of neutrophil extracellular traps. *Kidney Int.* <http://dx.doi.org/10.1016/j.kint.2016.08.006> (2016)

 RISK FACTORS**Relationship between sodium intake and mortality**

New data indicate a long-term beneficial effect of reduced sodium intake on mortality. Among the participants of the phase I ($n = 744$) and phase II ($n = 2,382$) TOHP sodium reduction trials, the risk of death during a median post-trial period of 24 years was 15% lower in the intervention group than in the control group. The analysis showed a direct linear relationship between average sodium intake and total mortality, even at the lowest sodium levels. The TOHP trials used well-characterized measures of sodium intake estimated from urinary sodium excretion.

ORIGINAL ARTICLE Cook, N. R. *et al.* Sodium intake and all-cause mortality over 20 years in the trials of hypertension prevention. *J. Am. Coll. Cardiol.* **68**, 1609–1617 (2016)

 THROMBOSIS**New mechanism of thrombus formation in CKD**

Holy *et al.* suggest that carbamylated (c)LDL promotes thrombus formation in patients with chronic kidney disease (CKD). They found that LDL isolated from these patients (CKD LDL) had higher levels of carbamylation than LDL from healthy controls. In mice, administration of cLDL or of CKD LDL accelerated arterial thrombus formation compared with administration of native LDL. Moreover, cLDL and CKD LDL, but not native LDL, enhanced platelet aggregation in response to agonists via a mechanism involving phosphorylation of MAPK p38 and translocation of LOX-1 to the cell surface.

ORIGINAL ARTICLE Holy, A. N. *et al.* Carbamylated low-density lipoproteins induce a prothrombotic state via LOX-1. *J. Am. Coll. Cardiol.* **68**, 1664–1676 (2016)