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IN BRIEF

TRANSPLANTATION

Survival benefit of accepting a diabetic deceased donor kidney

Despite the increased mortality risk of recipients of diabetic versus nondiabetic deceased donor kidneys, for many transplant candidates, accepting a diabetic donor kidney confers a survival benefit compared with remaining on the waitlist, say researchers. The greatest survival benefit of diabetic donor kidney transplantation compared with remaining on the waitlist was seen for diabetic recipients at centres with long waitlist times, whereas candidates aged <40 years showed no survival benefit of accepting a diabetic donor kidney.

ORIGINAL ARTICLE Cohen, J. B. *et al.* Survival benefit of transplantation with a deceased diabetic donor kidney compared with remaining on the waitlist. *Clin. J. Am. Soc. Nephrol.* http://dx.doi.org/10.2215/CJN.10280916 (2017)

ONCONEPHROLOGY

New mouse model of clear cell renal cell carcinoma

A new autochthonous mouse model of clear cell renal cell carcinoma (ccRCC) could be used to investigate ccRCC biology and test new therapies. Combined deletion of *Vhl*, *Trp53* and *Rb1* in renal epithelial cells induced the development of ccRCC tumours that arose from the proximal tubule and had similar cellular and molecular features to human ccRCC, including recurrent mutations in genes associated with the primary cilium. Different mouse tumours responded differently to ccRCC therapies and to HIF-α inhibition, suggesting that the model could be a useful tool for the identification of molecular determinants of tumour sensitivity and resistance.

ORIGINAL ARTICLE Harlander, S. *et al.* Combined mutation in *Vhl*, *Trp53* and *Rb1* causes clear cell renal cell carcinoma in mice. *Nat. Med.* http://dx.doi.org/10.1038/nm.4343 (2017)

ACUTE KIDNEY INJURY

Targeting T_{reg} cells to protect the kidney

A hybrid cytokine created by linking IL-2 and IL-33 (IL233) might protect against acute kidney injury (AKI). In mice, IL233 increased the numbers of regulatory T (T_{reg}) cells in the blood, spleen and kidneys and prevented renal ischaemia–reperfusion injury (IRI) more efficiently than did IL-2 and IL-33. Treatment with IL233 also increased the proportion of innate lymphoid cells that expressed the IL-33 receptor (ILC2 cells) in the blood and kidneys. Adoptive transfer of IL233-treated T_{reg} cells or ILC2 cells protected mice against IRI, suggesting that IL233 enhances the protective effects of these cells in the setting of AKI.

ORIGINAL ARTICLE Stremska, M. E. *et al.* IL233, a novel IL-2 and IL-33 hybrid cytokine ameliorates renal injury. *J. Am. Soc. Nephrol.* http://dx.doi.org/10.1681/ASN.2016121272 (2017)

LUPUS NEPHRITIS

Role of NLRP3 inflammasomes in podocyte injury

New data implicate podocyte NLRP3 inflammasomes in the pathogenesis of lupus nephritis (LN). These inflammasomes were activated in biopsy samples from patients with LN and in lupus-prone NZM2328 mice. In the mice, NLRP3 inhibition reduced proteinuria, renal histological lesions and podocyte foot-process effacement. Sera from diseased NZM2328 mice activated NLRP3 inflammasomes in cultured podocytes via a mechanism involving production of reactive oxygen species. These findings are consistent with a role of NLRP3 activation in podocyte injury and the development of proteinuria in LN.

ORIGINAL ARTICLE Fu, R. *et al.* Podocyte activation of NLRP3 inflammasome contributes to the development of proteinuria in lupus nephritis. *Arthritis Rheum.* http://dx.doi.org/10.1002/art.40155 (2017)

CORRECTION

Targeting T_{reg} cells to protect the kidney

Ellen F. Carney

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In the original html and PDF versions of this article, IL-2 was incorrectly written as IL-22 in two places and IL-33 was incorrectly written as IL-23 in the first sentence. These errors have now been corrected.