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IN BRIEF

TRANSPLANTATION

A new biomarker for transplant rejection?

A need exists for biomarkers that can monitor transplant rejection. Now, Vallabhajosyula *et al.* show that, in a human-to-mouse xenogeneic islet transplant model, human exosomes (extracellular vesicles that are released by tissues) can be purified from the plasma of recipient mice. Moreover, islet rejection was associated with a marked reduction in the number of human exosomes. Importantly, donor kidney-specific exosomes were detected in five patients who underwent renal transplantation. The researchers propose that exosomes have potential as biomarkers of transplant rejection.

ORIGINAL ARTICLE Vallabhajosyula, P. *et al.* Tissue-specific exosome biomarkers for noninvasively monitoring immunologic rejection of transplanted tissue. *J. Clin. Invest.* http://dx.doi.org/10.1172/JCI87993 (2017)

EPIDEMIOLOGY

Sickle cell trait increases ESRD risk

The risk of end-stage renal disease (ESRD) is higher in people of African ancestry than in those of European ancestry, but whether sickle cell trait (SCT), which is relatively common in black individuals, is a risk factor for ESRD is unclear. Naik *et al.* analysed the presence of SCT and the incidence of chronic kidney disease and ESRD in >9,900 black individuals from the Reasons for Geographic and Racial Differences in Stroke cohort study. SCT was strongly associated with risk of progression to ESRD. The researchers suggest that this association might have public policy implications for the counselling and treatment of SCT carriers.

ORIGINAL ARTICLE Naik, R. P. *et al.* Sickle cell trait and the risk of ESRD in blacks. *J. Am. Soc. Nephrol.* http://dx.doi.org/10.1681/ASN.2016101086 (2017)

RENAL FIBROSIS

Pericytes activate complement in fibrosis

The complement pathway is activated during kidney fibrosis and may contribute to renal inflammation, but the underlying mechanisms are unclear. Using a mouse model of kidney injury, Xavier *et al.* show that platelet-derived growth factor receptor-β-positive pericytes (which are non-immune, interstitial cells) secrete C1q; this complement component is involved in activation of the classical pathway. Global deletion of C3, which is downstream of C1q, reduced fibrosis in mice, suggesting that the complement pathway might represent a novel therapeutic target for the treatment of renal fibrosis.

ORIGINAL ARTICLE Xavier, S. *et al.* Pericytes and immune cells contribute to complement activation in tubulointerstitial fibrosis. *Am. J. Physiol. Renal Physiol.* **312**, 516–532 (2017)

LUPUS NEPHRITIS

CD11b suppresses inflammation in SLE

Single-nucleotide polymorphisms (SNPs) in the *ITGAM* gene (which encodes the adhesion molecule CD11b) are associated with an increased risk of systemic lupus erythematosus (SLE). Faridi *et al.* show that *ITGAM* SNPs that reduce CD11b function correlate with elevated activity of pro-inflammatory IFN-1 in patients with SLE. Furthermore, pharmacological activation of CD11b protected lupus-prone mice from end-organ injury and reduced pro-inflammatory IFN-1 responses by suppressing Toll-like receptor signalling. The researchers suggest that CD11b could be a target for therapeutic strategies to treat SLE.

ORIGINAL ARTICLE Faridi, M. H. *et al.* CD11b activation suppresses TLR-dependent inflammation and autoimmunity in systemic lupus erythematosus. *J. Clin. Invest.* http://dx.doi.org/10.1172/JCI88442 (2017)