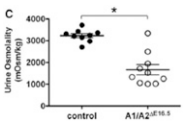
This Month's Highlights

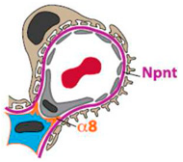
BASIC RESEARCH

Ascending Vasa Recta Are Lymph-Blood Vessel Hybrids



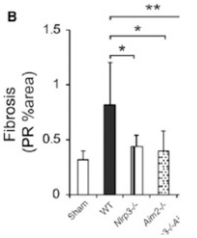
Interstitial fluid accumulates in the renal medullary interstitium, from which it is then returned to the systemic circulation. However, the renal medulla lacks lymphatic vessels, and the vessels involved in fluid return to the circulation have not been identified. In this issue, Kenig-Kozlovsky *et al.* demonstrate that the ascending vasa recta (AVR) are specialized hybrid vessels with properties of both blood and lymphatic vessels. Loss of angiopoietin/Tie2 signaling in late gestation leads to loss of the AVR and causes dilution and increased excretion of urine, as well as the formation of interstitial cysts in the medulla. Thus, the novel lymphatic-like features of the AVR appear responsible for fluid drainage in the medulla. *See Kenig-Kozlovsky et al., pages* 1097–1107.

Nephronectin Links Glomerular Basement Membrane and Mesangial Cells



Components of the glomerular basement membrane (GBM) contact the mesangial cells that provide structural stability to glomeruli, but the functional consequences of such contact are unknown. Here, Zimmerman *et al.* show that nephronectin (*Npnt*), a GBM component and $\alpha 8\beta 1$ integrin ligand, is produced by podocytes and required to maintain GBM-mesangial adhesions in mice. Deletion of *Npnt* from nephron progenitors increases mesangial cell number and causes mesangial sclerosis. These results reveal a receptor-ligand interaction between the GBM and mesangial cells *in vivo* and suggest that nephronectin is an endogenous regulator of mesangial cell number and matrix production. *See Zimmerman et al., pages* 1128–1140.

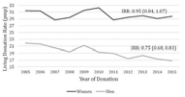
The Absent in Melanoma-2 Inflammasome in Kidney Disease



Inflammasomes are innate immune system pathways that contribute to kidney inflammation and fibrosis, yet the role of most inflammasomes in the kidney is unknown. Here, Komada *et al.* report that the absent in melanoma-2 (AIM2) inflammasome is expressed in several compartments of normal kidneys and upregulated in the tubular epithelium and inflammatory infiltrates during kidney disease. Studies in a mouse model of unilateral ureteric obstruction showed that the Aim2 inflammasome drives tubulointerstitial inflammation and fibrosis by stimulating the uptake of necrotic cell DNA by recruited macrophages. The therapeutic utility of targeting inflammasomes and the related effector pathways in kidney disease should be investigated. *See Komada et al., pages* 1165–1181.

CLINICAL EPIDEMIOLOGY

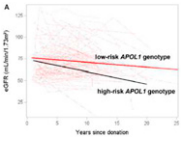
Sex and Living Kidney Donation Rate



Why have living kidney donation rates declined in the United States? In this issue, Gill *et al.* report the findings from their population-based analysis of the changes in living kidney donation in adults in the United States between 2005 and 2015. The rate of living kidney donation declined in men but remained the same in women, and income appeared to be an important factor in this discrepancy. Notably, living related kidney donations declined in both sexes, regardless of income. Although studies are needed to identify the reasons for the decline in living related donations, strategies to remove financial barriers and prevent discrimination against donors may help maintain living kidney donation as an option for patients in need of a transplant. *See Gill et al., pages* 1301–1308.

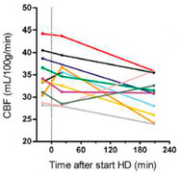
CLINICAL RESEARCH

APOL1 Status and Kidney Donor Outcomes



Does the *APOL1* high-risk genotype associate with postdonation kidney function in black donors? Doshi *et al.* examined this possibility in 136 black living kidney donors grouped by *APOL1* genotype: high-risk (two risk alleles) or low-risk (one or zero risk alleles). The *APOL1* high-risk genotype associated with lower predonation and postdonation renal function in donors. Two donors reached ESRD, both with high-risk *APOL1* genotypes. When donors were matched with nondonors by *APOL1* status, renal outcomes did not differ between the groups. *APOL1* high-risk genotype may be associated with worse renal outcomes in kidney donors, but these data require replication in a larger study before routine *APOL1* genetic testing is recommended. *See Doshi et al., pages* 1309–1316.

Hemodialysis and Cerebral Blood Flow



Previous studies have shown that the initiation of hemodialysis associates with significant cognitive decline and an increase in white matter lesions, but the underlying mechanisms are unknown. Polinder-Bos *et al.* examined the acute effect of conventional hemodialysis on cerebral blood flow (CBF) using $[^{15}O]H_2O$ positron emission tomography-computed tomography (PET-CT) in 12 patients aged ≥65 years. Their findings reveal a significant reduction in global and regional CBF occurs over a single hemodialysis session. Higher pH, body temperature, and ultrafiltration volume and rate were associated with lower brain perfusion. Research is needed to develop hemodialysis protocols that minimize cerebrovascular stress. *See Polinder-Bos et al., pages* 1317–1325.