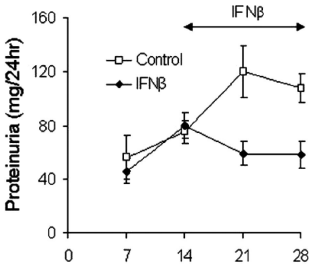
This Month's Highlights

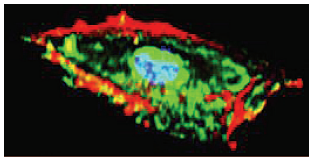
BASIC RESEARCH

Mechanism of IFN-β's Antiproteinuric Effect



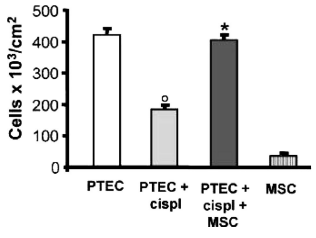
IFN-β, an immunomodulatory cytokine, has been shown to reduce proteinuria, improve renal function, and prolong survival in a mouse model of lupus. Satchell and colleagues report that IFN-β significantly reduces proteinuria in three rat models of glomerulonephritis, but this does not seem to be a result of reduced glomerular inflammation. Instead, IFN-β decreases albumin flux and increases electrical resistance across glomerular endothelial cells and podocytes. They suggest that IFN-β may hold promise for the treatment of proteinuric renal diseases, in part by tightening the glomerular filtration barrier. *See Satchell et al., pages 2875–2884.*

Glycocalyx Contributes to Glomerular Permselectivity



Glycocalyx coats the luminal surface of all endothelia. Singh and colleagues confirm the presence of a 200-nm coat of glycocalyx on conditionally immortalized human glomerular endothelial cells. Incubation with heparanase, which degrades a component of the glycocalyx, increases albumin flux across these cells but does not affect transcellular passage of water and solute. Therefore, glycocalyx seems to contribute to the selective permeability of the glomerular basement membrane, and heparanase may contribute to the pathogenesis of proteinuric renal disease by disturbing this barrier. *See Singh et al., pages 2885–2893.*

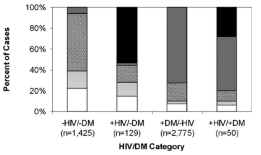
Renoprotective Stem Cells Secrete IGF-1



Administration of bone marrow–derived mesenchymal stem cells improves renal structure and function in mice with cisplatin-induced kidney injury. Coculture experiments by Imberti and colleagues demonstrate that IGF-1 produced by stem cells protects cisplatin-treated proximal tubule cells by promoting cellular proliferation and reducing apoptosis. In mice, this renoprotective effect was removed when the IGF-1 gene was silenced. Mesenchymal stem cells may be used to treat acute kidney injury in the future, and their beneficial effect may result, in part, from the release of IGF-1 in the damaged microenvironment. *See Imberti et al., pages 2921–2928.*

CLINICAL EPIDEMIOLOGY

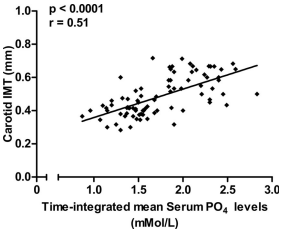
Race, HIV, and ESRD



Little is known about the risk of ESRD among patients infected with the HIV. Choi and colleagues analyzed data from >2 million U.S. veterans and report that diabetes and HIV confer similar increased risks of ESRD among blacks. Among whites, diabetes was associated with an increased risk of ESRD but HIV was not. In addition, the age- and sex-adjusted incidence of ESRD was nearly 10 times higher among HIV-infected blacks than HIV-infected whites. These findings should encourage new strategies to treat renal disease among HIV-infected blacks. *See Choi et al., pages 2968–2974.*

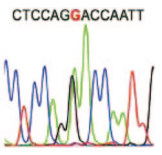
CLINICAL RESEARCH

PTH Levels Predict Vascular Damage in Children



Cardiovascular disease is the most common cause of death among young adults with chronic kidney disease. Shroff and colleagues report that children with ESRD and levels of PTH more than twice the upper limit of normal have greater carotid intima-media thickness, stiffer vessels, and increased cardiac calcification compared with age-matched controls. A strong dose-dependent correlation exists between vitamin D therapy and all vascular measures. Although causation cannot be claimed from this observational study, uncontrolled secondary hyperparathyroidism and exogenous vitamin D may contribute to vascular damage and calcification among children on dialysis. *See Shroff et al., pages 2996–3003.*

Thin Basement Membrane Nephropathy Isn't Always Benign



Classic teaching suggests that thin basement membrane nephropathy carries an excellent prognosis. Voskarides and colleagues diagnosed concomitant FSGS and thin basement membrane nephropathy in 20 renal biopsies from 13 families. In 10 of these families, the authors identified mutations in the *COL4A3/COL4A4* locus. Of 82 patients harboring mutations, 38% developed chronic renal failure, and 20% developed ESRD. Although the identified mutations have not been shown to cause FSGS directly, it can be concluded that "benign familial hematuria" is a misnomer for some cases of thin basement membrane nephropathy. *See Voskarides et al., pages 3004–3016.*