**BASIC RESEARCH**

**Pores Found in the Glomerular Filtration Barrier**

Study of the ultrastructure of the glomerular filtration slit during the past few decades has put forward the notion that podocyte foot processes form a zipper-like structure. Structural observations and analyses of solute clearance, however, have not been entirely consistent. Here, Gagliardini et al. used scanning electron microscopy to image the deepest regions of the filtration slits. They observed circular and ellipsoidal pores in the podocyte junctions with some very large pores present in proteinuric states. These results provide a unique look at the heteroporous nature of the glomerular filtration barrier. See Gagliardini et al., pages 2081–2089.

**Minocycline Protects against Osmotic Demyelination**

Osmotic demyelination syndrome (ODS) can follow rapid correction of hyponatremia, and activated microglia may promote its pathogenesis. In this issue, both Suzuki et al. and Gankam-Kengne et al. independently report that minocycline, which is a potent inhibitor of microglial activation, can protect against ODS and improve survival in rats that experience rapid correction of chronic hyponatremia. Although avoidance of rapid changes in serum sodium should remain the mainstay of care in humans, these data suggest a possible prophylactic use of minocycline in cases of severe hyponatremia. See Suzuki et al., pages 2090–2098, and Gankam-Kengne et al., pages 2099–2108.

**CLINICAL RESEARCH**

**Antibodies Impair Fibrinolysis in ANCA Vasculitis**

Anti-plasminogen antibodies are present in some patients with ANCA-associated vasculitis, but their functional consequence is not completely understood. In this issue, Berden et al. report that they identified anti-plasminogen antibodies in approximately 25% of patients in two independent cohorts of ANCA-associated vasculitis. Furthermore, these patients are more likely to harbor anti-tissue plasminogen activator antibodies as well. The presence of these antibodies associates with impaired fibrinolysis, and patients with these antibodies are more likely to exhibit glomerular fibrinoid necrosis, crescents, and impaired renal function, suggesting their functional consequence. See Berden et al., pages 2169–2179.

**Anti-Factor H–associated Atypical HUS Characterized**

Atypical hemolytic uremic syndrome (aHUS) is a rare form of thrombotic microangiopathy that can result from either genetic or acquired disorders. In this issue, Dragon-Durey et al. present a case series of 45 patients who presented with aHUS associated with anti–factor H antibody. Although they primarily observed this disease in children, they describe seven cases in adults as well. They found that activation of the alternative pathway of complement at disease onset portends poor prognosis and that early specific treatment may improve outcomes. The clinical details reported herein should improve the recognition of this form of aHUS. See Dragon-Durey et al., pages 2180–2187.

**BRIEF COMMUNICATION**

**Flow across Filtration Barrier Generates Voltage**

Electrical properties of the glomerular filtration barrier contribute to its selectivity, but how electrical potential differences are generated across the barrier is not well understood. Here, Hausmann et al. performed micropuncture of glomerular capillaries to determine whether flow across the glomerular filter generates electrical potential differences. Their experimentally based mathematical model suggests that perfusion pressure and, therefore, filtration generate potential differences sufficient to modulate the sieving coefficient of albumin. Their results extend our understanding of the fundamental properties of the glomerular filtration barrier. See Hausmann et al., pages 2053–2058.

**Magnesium Modulates Potassium Secretion**

Magnesium depletion can lead to refractory hypokalemia, but the mechanisms underlying the relationship between magnesium and $K^+$ homeostasis are incompletely understood. Here, Yang et al. demonstrate that intracellular magnesium blocks secretory $K^+$ currents through ROMK channels; therefore, magnesium depletion promotes $K^+$ loss. Furthermore, this magnesium-mediated inhibition of $K^+$ secretion increases as extracellular $K^+$ decreases, which appropriately reduces $K^+$ loss in the presence of $K^+$ deficiency. Their experiments help to explain changes in $K^+$ transport during states of $K^+$ or magnesium depletion. See Yang et al., pages 2109–2116.