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Response to "JC polyoma virus and kidney disease"

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We appreciate Dr. Brennan and colleagues' novel observation that shedding of urinary tract JC polyomavirus (JCV) associates with reduced rates of acute renal allograft rejection.[1] Bacterial urinary tract infections trigger acute rejection and non-JC viral infections may do the same. This finding further supports protective effects of urinary tract JCV on CKD and *APOL1* gene-JCV interactions reducing risk of non-diabetic nephropathy.[2;3] These diverse clinical scenarios suggest that JCV exhibits a commensal relationship with urothelial and kidney cells. JC viruria is not likely indicative of true infection, but colonization. This is akin to protective GI tract bacteria that inhibit growth of pathologic strains. As such, asymptomatic urinary tract JCV shedding could reflect health. Absent JCV, pathologic viruses may infect urothelial cells, ascend to and infect kidney cells, and cause chronic renal dysfunction or acute rejection. All studies support that a single urothelial viral strain inhibits growth of others.[1–3] Relative to the GI tract, the urine virome remains understudied. Known and novel viruses may reside in the urinary tract and be readily detectable in urine. Next generation sequencing in urine could identify intruding viruses that "move into the neighborhood when JCV is away".[4] Pathogenic viruses might underlie gene-virus interactions, CKD, and acute rejection. Kidney disease and acute rejection therapies may emerge from identification of these unwelcome guests.

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