Journal of NeuroVirology, 10(suppl. 2): 36, 2004

ISSN: 1355-0284 print / 1538-2443 online
DOI: 10.1080/13550280490469798

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Characterization of BK virus infectious entry

S Eash[1] and WJ Atwood[2]

[1]Graduate Program in Molecular Biology, Cell Biology and Biochemistry and [2]Department of Molecular Microbiology & Immunology, Brown University, Providence, Rhode Island, USA

Polyomavirus-associated nephropathy (PVAN) occurs in approximately 5% of renal transplant recipients and results in graft loss in 50% to 70% of these cases. The disease is caused by the reactivation of the common human polyomavirus BK (BKV) in the transplanted kidney. The early events in BKV productive infection are unknown. We are focused on elucidating the mechanisms of BKV internalization and intracellular transport in the target cell. Our data reveal that BKV entry into permissive Vero cells is slow, requires cholesterol, and is independent of clathrin-coated-pit assembly. Inside the cell BKV co-localizes with the caveolae-mediated endocytic marker Cholera toxin subunit B, but not with the clathrin-dependent endocytic marker transferrin. BKV infectious entry is sensitive to pH elevation in acidic organelles. We are currently using dominant negative caveolin-1 mutants to establish a role for caveolae in BKV internalization. Selective pharmacological disruption of cytoskeletal components will help us determine which elements of the cellular transport system aid BKV translocation from the periphery to the nucleus of the infected cell.