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Early events controlling infection of cells by human polyomaviruses

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Virus receptor interactions are important determinants of tropism, spread, and pathogenesis in the infected host. JC Virus (JCV) and BK Virus (BKV) are ubiquitous human polyomaviruses and are responsible for significant morbidity and mortality in immunosuppressed patients. The mechanisms by which JCV and BKV target specific cells and tissues in the human host are not understood. Our laboratory has characterized the cellular receptor for JCV as an N-linked glycoprotein containing terminal alpha 2–6 linked sialic acid. We recently demonstrated that the tissue distribution of alpha 2–6 linked sialic acid overlaps with the known tropism of JCV for oligodendrocytes, astrocytes, and B lymphocytes. Furthermore, a chimeric virus containing the early region of SV40 fused to the late region of JCV maintains the host range of JCV, suggesting that virus receptor interactions are an important determinant of tropism. This seminar will review these data and discuss new data from our studies on both JCV and BKV.