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Polyomaviruses and human neoplasia

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A number of studies in the last twenty years have reported the detection of polyomaviruses including JCV, BKV, and SV40 in various human tumors. These observations are alarming in light of laboratory data establishing the transforming ability of the polyomavirus early gene product, T-antigen, in cell culture and the tumorigenecity of this protein in experimental animal models. One area which has received extensive attention relates to the involvement of JCV with brain tumors, particularly medulloblastoma, and more recently, colorectal cancer. Our results show that the association of JCV T-antigen with p53 and pRb, the two main tumor suppressors that control cell proliferation, leads to their inactivation. In addition, T-antigen by dysregulating important signaling pathways such as IGF-1 and Wnt, promotes uncontrolled cell proliferation. Finally, T-antigen exhibits the ability to interfere with DNA repair and results in increased levels of genomic instability and aneuploidy. I will review the various mechanisms that are involved in polyomavirus induced transformation.