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HIV-associated salivary gland disease: a role for BK birus

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HIV-associated salivary gland disease (HIV-SGD) is disfiguring and causes significant morbidity in the HIV population. Evidence detailing the epidemiology of HIV-SGD suggests the involvement of a viral opportunist in its pathogenesis, yet the specific etiology of HIV-SGD remains unclear. To determine the role for an opportunistic virus as the etiologic agent of HIV-SGD, we hypothesized that HIV-SGD was a manifestation of primary infection or reactivation with a DNA tumor virus, BKV, during immune suppression. The central hypothesis of this work is that viral pathogenesis is essential to the development of salivary gland disease. Results show for the first time that polyomavirus, BKV, is associated with HIV-SGD. BKV DNA, RNA, and protein were consistently detected in salivary gland biopsies and in the peripheral blood and oral fluids from HIV-SGD patients and not in control subjects. To confirm the in vivo findings, an in vitro model was created whereby parotid and submandibular salivary gland cells were productively infected with BKV, demonstrating each part of the viral life cycle. Salivary gland tropism was confirmed and the BKV receptor on salivary gland cells was defined. BKV transmission and pathogenesis is not well understood. Importantly, these studies suggest a role for BKV in HIV-SGD and that BKV transmission may occur via the oral route. The long-term goal of this project is to make critical strides toward understanding the etiology of SGD in order to go beyond the ineffective palliative treatment that is currently the standard of care.

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