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Accelerated progression to AIDS in macaques coinfecting with simian immunodeficiency virus and human herpesvirus 6A

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Although HIV-1 is the necessary and sufficient causative agent of AIDS, genetic and environmental factors markedly influence the pace of disease progression. Clinical and experimental evidence suggests that human herpesvirus 6 (HHV-6), a cytopathic T-lymphotropic agent, may act as an accelerating factor in the progression of HIV disease, although conclusive *in vivo* evidence has yet to be attained. To evaluate the effect of HHV-6A on the course of AIDS in a relevant model system, we infected pig-tailed macaques (*M. nemestrina*) either with HHV-6A (strain GS) or with a pathogenic SIV strain (smE660), or with both viruses. Extensive longitudinal virologic, immunologic and clinical follow-up demonstrated that HHV-6A coinfection dramatically accelerated the progression toward full-blown AIDS. Rapid disease development in coinfecting animals was associated with an early depletion of both CD4+ and CD8+ T cells. Simultaneous replication of both viruses was documented in coinfecting lymph node tissue. These data establish a new animal model for the study of HHV-6 infection and provide the first conclusive *in vivo* evidence that HHV-6A acts as a cofactor in the progression of primate immunodeficiency virus disease.