Oral presentation

Open Access Accelerated progression to AIDS in macagues coinfected with simian immunodeficiency virus and human herpesvirus 6A Paolo Lusso^{*1}, Richard W Crowley², Mauro S Malnati¹, Maurilio Ponzoni¹,

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from 2006 International Meeting of The Institute of Human Virology Baltimore, USA. 17-21 November, 2006

Published: 21 December 2006

Retrovirology 2006, 3(Suppl 1):S62 doi:10.1186/1742-4690-3-S1-S62

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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 coinfected animals was associated with an early depletion of both CD4+ and CD8+ T cells. Simultaneous replication of both viruses was documented in coinfected 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.