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INDUCTION OF HHV-8 LYTIC CYCLE REPLICATION BY CYTOKINES PRODUCED BY HIV-1-INFECTED T-CELLS.

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HHV-8 is a recently described γ 2-herpesvirus which is consistently identified in Kaposi's sarcoma (KS). While HHV-8 infection appears to be necessary, it may not be sufficient for development of KS without involvement of other essential co-factors. One potentially important co-factor is HIV-1. HIV-1 infected cells produce HIV-1 related proteins and cytokines, both of which have been shown to promote growth of KS cells *in vitro*. While HIV-1 is not absolutely necessary for KS development, KS is the most frequent neoplasm in AIDS patients, and AIDS-KS is recognized as a particularly aggressive form of the disease. To determine if HIV-1 could participate in the pathogenesis of KS by modulating HHV-8 replication (rather than by inducing immunodeficiency), HIV-1 infected T-cells were co-cultured with the HHV-8 infected cell line, BCBL-1. The results demonstrate soluble factors produced by HIV-1 infected T-cells or in response to HIV-1 infected T-cells induce HHV-8 lytic cycle replication, as determined by production of lytic phase mRNA transcripts, viral proteins, and detection of progeny virions. By focusing on cytokines produced in the co-culture system, these studies demonstrate several cytokines known to be important in the growth and proliferation of KS cells *in vitro*, particularly Oncostatin M, scatter factor, and IFN- γ , induce HHV-8 lytic cycle replication when added individually to BCBL-1 cells. These results suggest cytokines may play an important role in the initiation and progression of KS through reactivation of HHV-8 replication. Thus, HIV-1 may participate more directly than previously recognized in KS by promoting the lytic replication and hence the local HHV-8 viral load.

Keywords: AEGIS, Herpesvirus 8, Human, HIV-1, Virus Replication, T-Lymphocytes, Sarcoma, Kaposi, Cytokines, Acquired Immunodeficiency Syndrome, Cell Line, Viral Proteins, Infection, AIDS-Related Opportunistic Infections, *in vitro*, Human, virology, AIDS

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