72

Cation Influence on Survival and Lymphoma in Murine Immunodeficiency Disease (MAIDS): Effects of Lithium and Magnesium In Vivo. VS Gallicchio, ML Cibull, NK Hughes, KF Tse, and H Gaines. Hematology/Oncology Division, Departments of Medicine, Clinical Sciences and Pathology, University of Kentucky Medical Center and Department of Veterans Affairs, Lexington, KY, USA.

MAIDS induced with the LP-BM5 strain of MuLV is a disease that shows many similarities to human HIV-infection. The monovalent cation lithium (Li^+) influences immunohematopoietic cells, immune function, and is an antagonist to Mg^{2+} dependent reactions. We report here results of in vivo studies investigating the effect of Li^+ and Mg^{2+} treatment in MAIDS-infected mice. Viral control, Li^+ and Mg^{2+} treated C57BL6 mice were monitored for survival and MAIDS pathology. Virus-infected mice were grouped to receive Li^+ daily as follows: (1) 48-hrs before virus; (2) at the same time as virus; and (3) 5-weeks post-viral inoculation. Mg^{2+} was given 5-weeks post-virus. After 22-weeks of observation percent survival was as follows: virus controls - 20%; Mg^{2+}-5 weeks post-virus - 40%; Li^+-5 weeks post-virus - 80%; Li^+-at the time of virus - 85%; and Li^+-48 hrs prior to virus - 100% survival. The extent of lymphoma in these Li^+/Mg^{2+} treated mice was significant as measured by splenomegaly (gms): virus control, 0.81 ± 0.24; Mg^{2+}, 1.32 ± 0.14; Li^+-5 weeks post-virus, 0.48 ± 0.03; and Li^+ at the time of virus, 0.28 ± 0.1 (P value <0.01). At 22 weeks all Li^+-48 hrs treated mice are still alive. These data suggest cations play an important role in MAIDS pathogenesis and indicates Li^+ may be efficacious in altering the pathogenesis of this retroviral disease.

73

LACK OF INVOLVEMENT OF TRANSACTIVATING HERPESVIRUSES IN ACUTE SIV INFECTION IN MONKEYS

D. Böttiger[1] and B. Öberg[1,2]
[1]Dept of Virology, KI, Stockholm and [2]Medivir AB, Huddinge, Sweden

There are several and conflicting reports on the transactivation of HIV by herpesviruses. The acute infection of cynomolgus monkeys with simian immunodeficiency virus (SIV) offers an in vivo system where the influence of replicating herpesviruses on SIV replication can be studied.

Four different inhibitors of herpesviruses, phosphonoformic acid (PFA), phosphonoacetic acid (PAA), acyclovir (ACV) and ganciclovir (GCV) were given s.c. to monkeys starting 8 hours before inoculation with SIV. Treatment was given every 8 hours for 10 days and the appearance of viral antigen in plasma was followed. PFA, which is an inhibitor of both herpesviruses and HIV/SIV, caused a delay in the appearance of antigens while the other inhibitors did not affect the SIV infection. These results indicate that transactivation by a herpesvirus is not significantly influencing the acute SIV infection in vivo.

The involvement of a transactivating herpesvirus in the late HIV/SIV infection remains to be studied.