II-20

Inhibition of HSV-2 Replication in Vero Cell by Spleen and Lymphnode Cells of Mice Infected With HSV-2 and Treated With Interferons. Y.-G. Man. Department of Cell Biology, Virus Research Institute, Hubei Medical College, Wuhan, China.

In order to determine whether IFN-s treatment could make the cellular immune system more effective to against HSV-2 infection and develop a simple technique to quantitate the spread of HSV-2 infection in vitro, C57BL/6J mice were infected intravaginally with HSV-2 and treated with a recombinant mouse a/β or r interferon. 3 and 7 days after virus infection, the spleen and lymphnode of mice were aseptically removed. A single cell suspension was prepared and added onto the HSV-2 infected vero monolayers at a various ratios of E:T. After 48 hours of incubation at 37°C, under a microscope, almost all the vero cells in the absence of effector cells showed HSV-2 induced cytopathological effect. But the infected monolayer was still confluent. In contrast, most of vero cells in the presence of effector cells showed no pathomorphological changes. But many holes were found in the infected monolayers. In the margin of hole, many effector cells surrounded the target cells. When these infectious cultures were harvested and titered, virus yield decreased, in a dose dependent manner, with increasing effector cells. The immunized cells were more effective to against HSV-2 replication than that of unimmunized cells. The spleen cells of mice infected with HSV-2 and treated with r interferon were found to have very strong inhibitory activity in which the virus yield was reduced by 4.13 Logs at a ratio of E:T = 100:1. This inhibitory effect was neither due to a nonspecific toxicity of soluble factors nor a metabolic effect or the generation of interferons.

II-21

Effect of 9-(1,3-dihydroxy-2-propoxymethyl) guanine (DHPG) on Cell Mediated Immune (CMI) Response of Normal and Murine Cytomegalovirus (MCMV) Infected Mice. J. Shelby, E.R. Kern and J.R. Saffle. Univ. of Utah Sch. of Med., Salt Lake City, Utah, USA.

DHPG has recently been shown to inhibit human lymphocyte blastogenesis in vitro, raising the possibility of immunosuppressive effects in vivo. To assess the effect of DHPG on CMI response, a delayed type hypersensitivity assay (DTH) was used in BALB/c mice. The mice were sensitized on days 0 and +1 with 25 ul of 0.25% dinitrofluorobenzene (DNFB) applied to the shaved abdomen. One footpad was challenged with DNFB on day +4 and measured 24 h later. The size difference from the unchallenged footpad was determined and expressed as % suppression compared with controls. Intraperitoneal (i.p.) injections of DHPG were given twice daily to normal and MCMV (10^5 pfu, i.p.) infected mice. The DTH response was suppressed by 95% and 42% in normal mice given 40 or 15 mg/kg of DHPG on days -5 to +4 ($p<0.05$), but not on days -1 to +4. Untreated mice given MCMV on day -3 showed a 31% suppression ($p<0.01$), while a normal response was seen in mice infected with MCMV on day -7. In contrast, the response of mice infected with MCMV on day -7 and treated with DHPG (15 mg/kg, days -5 to +4) was inhibited by 36% ($p<0.01$). The DTH response in mice infected on day -3 and treated with 40 or 15 mg/kg of DHPG on days -1 to +4 was suppressed by 93% and 25% respectively ($p<0.01$). A normal CMI response was observed in mice infected on day -3 and given 5 mg/kg of DHPG. These results indicate that the inhibition of the DTH response in normal and MCMV-infected mice was dependent on both concentration and duration of DHPG treatment.