

Effect of Zn-complexes on HSV-2 Infection *in vitro*

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The inhibitory effect of complexes of Zn(II) with picolinic and asparaginic acids [Zn(pic)₂ and Zn(asp)₂] on HSV-1 infection was previously reported by us (J. Chemotherapy, 1993, 5, 1, 3-9).

However, HSV-2 infection in HEL cells was also affected. Thus, after the action with 10, 1 and 0.1 μM of an each complex the infectious titre was reduced with 1.3, 1.2 and 1.1 lg units respectively. Furthermore, a sharply increased amount of empty viruses and L-particles in the presence of both complexes was detected by electron microscope. The prophylactic effect of Zn(pic)₂ and Zn(asp)₂ expressed in 24 hrs supplemented cells was detected by a reduction of infectious titre with 90% and 99% respectively. In addition, a ligand-dependent effect was manifested by: 1) Restoration of viral infectivity after influence with 10, 1 and 0.1 μM of Zn(pic)₂ with 10%, 20% and 50% respectively, while after the action with Zn(asp)₂ the viral titre was reduced with 20%, 75% and 100%; 2) Irreversible inhibition of the free virus being in a contact with Zn(asp)₂ for 2-6 hrs and a reversible effect of Zn(pic)₂ after 30 min contact; 3) Absence of gB, gC and gE after influence with Zn(asp)₂ only was obtained by immunoblot test;

Effect of human recombinant and recombinant hybrid alpha-interferons on the replication of herpes simplex virus type 1 in human fibroblast and neuroblastoma cells. S. Chatterjee¹, P. Burns¹, J.D. Gangemi² and R.J. Whitley¹. ¹Department of Pediatrics, University of Alabama School of Medicine, Birmingham, AL, ²Clemson University Biomedical Alliance, Clemson, SC; USA.

Pretreatment of human fibroblast and human neuroblastoma cells with human recombinant alpha-interferon (IFN) B or D or recombinant hybrid alpha - IFN B/D demonstrated that alpha-IFN B significantly inhibited the release of infectious herpes simplex virus type 1 (HSV-1) from treated cells. However, IFN B was more effective in human fibroblast cells than in human neuroblastoma cells. Immunoblot analysis showed that IFN B inhibited the expression of HSV-1 nucleocapsid proteins and glycoproteins in human fibroblast cells. In contrast, IFN B had no significant effect on the expression of nucleocapsid proteins, although the expression of glycoproteins was reduced in IFN B-treated human neuroblastoma cells. Pretreatment of neuroblastoma cells with staurosporine, a selective inhibitor of protein kinase C (PKC) prior to the addition of IFN B, blocked the inhibitory effect of IFN on the replication of HSV-1 in treated cells. In contrast, staurosporine had no effect on the inhibitory action of IFN B in human fibroblast cells. Consistent with this observation, addition of IFN B also resulted in an expression of PKC specifically in IFN-treated human neuroblastoma cells. These results suggest that the expression of PKC is an important step in the IFN B-treated cells of neuronal origin.