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Characterization of S-Adenosylmethionine Decarboxylase Induced by Human Cytomegalovirus. E.L. White, G. Arnett, J.A. Secrist III, and W.M. Shannon.* Southern Research Institute, Birmingham, AL USA.

Infection of human diploid embryonic lung (MRC5) cells by human cytomegalovirus (HCMV), strain AD169, increased the activities of two key enzymes in the synthesis of polyamines: S-adenosylmethionine decarboxylase (E.C. 4.1.1.50) and ornithine decarboxylase (E.C. 4.1.1.17). The initial peak of S-adenosylmethionine decarboxylase activity occurred at 15 hours postinfection, which was 10 hours before the ornithine decarboxylase activity peaked. S-Adenosylmethionine decarboxylase was purified from HCMV-infected and control uninfected MRC5 cells. No difference was found between the two enzymes in their stability to heat or effect of pH on activity. Both enzymes were activated only by putrescine. The K_m for S-adenosylmethionine for the virus-induced enzyme was 1.7 times higher than the K_m for the control enzyme. The most dramatic difference observed was in the effect of high salt concentration on enzyme activity. S-Adenosylmethionine decarboxylase from HCMV-infected cells was unaffected by 0.8 M NaCl; whereas, the enzyme from uninfected cells was inhibited by 50% at 0.45 M NaCl and was significantly inhibited at a concentration of 0.8 M NaCl. Thus, different forms of S-adenosylmethionine decarboxylase exist in infected and uninfected MRC5 cells. The early induction of S-adenosylmethionine decarboxylase after HCMV infection, occurring before the induction of ornithine decarboxylase, and the the stability of the HCMV-induced enzyyme relative to the host cell enzyme at high salt concentrations favor the proposition that HCMV encodes its own S-adenosylmethionine decarboxylase. **Partially supported by NIAID Contracts N01-AI-42555 & N01-AI-72642.**

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Co-infection of Herpes and Pox Viruses in BSC-1 Cells in the Presence of Antiviral Drugs. E. Katz. Department of Virology, Hebrew University - Hadassah Medical School, Jerusalem, Israel.

Co-infection in BSC-1 cells, of herpes simplex virus type 2, a virus which has a nuclear phase during its growth, and vaccinia virus, a virus growing in the cytoplasm of the cells, results in a significant inhibition of the two viruses. The inhibition of herpes virus takes place also when infection of vaccinia virus is carried out in the presence of isatin-β-thiosemicarbazone (28μM), an efficient inhibitor of pox viruses. This suggests that the inhibition caused by vaccinia virus is a result of a step preceding virus maturation. Adsorption of herpes virus, its penetration and induction of viral thymidine-kinase activity, are greatly suppressed following vaccinia virus infection. On the other hand, herpes virus at a multiplicity of infection of 2 plaque forming units per cell, inhibits vaccinia virus growth by 98%. This inhibition takes place also in the presence of acyclovir (100μM), a drug known to efficiently block herpes virus replication. This may indicate that a step preceding herpes virus DNA synthesis is involved in the inhibition of vaccinia virus by herpes virus infection.