60

ACTIVITY OF SEVERAL INHIBITORS OF S-ADENOSYL HOMOCYSTEINE HYDROLASE AGAINST AFRICAN SWINE FEVER VIRUS.

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We have investigated the activity of several inhibitors of S-adenosyl L homocysteine (SAH) hidrolase against African swine fever virus (ASFV) replication in Vero cells. The following compounds proved effective in inhibiting ASFV replication: 3-deazaneplanocin A, 6-β'-fluoroaristeromycin, 4',5'-unsaturated 5'-fluoroadenosine and the 9-(trans-2'-trans-3'-dihydroxycyclopentyl) derivatives of adenine and 3-deazaadenine. Their 50% inhibitory concentration for virus yield was 0.01, 0.01, 0.03, 0.3 and 0.1 µg/ml, and their 50% cytotoxic concentration for cell viability was 50, 1, 20, 200 and 200 µg/ml, respectively. Thus, the selectivity index of these SAH hydrolase inhibitors varied from 100 and 5000. SAH hydrolase inhibitors have proved specifically active against those viruses that encode their own methyltransferases. These enzymes are involved in methylation reactions required for virus replication. The anti-ASFV activity of the SAH hydrolase inhibitors may be attributed to the inhibition of methylation reactions responsible for the 5'-capping of viral mRNA.

61

AURINTRICARBOXYLIC ACID: AN INHIBITOR OF HERPES SIMPLEX TYPES I AND II, VACCINIA AND AFRICAN SWINE FEVER VIRUSES REPLICATION IN VERO CELLS.

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The action of the triphenylmethane dye aurintricarboxylate (ATA) has been tested as inhibitor of the replication of herpes simplex type I and II, vaccinia and african swine fever (ASF) viruses growing in Vero cells. The concentration required to inhibit these viruses is far below the toxicity for the cells. ATA inhibit protein synthesis of the viruses shown by polyacrilamide gel eletroforesis.

Experiments on addition time of the compound indicate that while both herpes are inhibited preferentially when the dye is added very early in infection vaccinia and ASF viruses are less inhibited at this time. The greatest inhibition was found between one and two hours after infection for vaccinia virus and four to six hours for ASF virus. This fact suggests two different mechanisms of inhibition.