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EDITORIAL

This issue is almost entirely devoted to antiviral studies with chemical substances. One of the recently developed antiviral agents, acyclovir, offers a great promise for the treatment of herpes simplex virus (HSV) infections. Its spectrum of activity, mechanism of action, efficacy in animal models and early clinical trials are reviewed by Brigden et al. These authors also address the problems of latency and resistance that are innately associated with the chemotherapy of HSV infections.

Two other antiherpes agents, [E]-5-(1-propenyl)-2′-deoxyuridine and 5-(1-propenyl)-1-β-D-arabinofuranosyluracil, are described by Stening et al. Although these compounds are less effective than the corresponding 5-(2-bromovinyl) derivatives, they may act through a similar mechanism of action, that is specific phosphorylation by the herpes virus-induced thymidine kinase, followed by a selective inhibitory effect at the viral DNA polymerase level. A therapeutic effect was noted with [E]-5-(1-propenyl)-2′-deoxyuridine when applied topically to guinea pigs with a cutaneous HSV type 1 infection.

Phosphonoacetic acid (PAA) and phosphonoformic acid (PFA) were evaluated by Kern et al. for their topical effects on genital HSV type 1 and 2 infections in mice. Both drugs significantly altered the course of the infection, and, although PAA was found more effective than PFA in the treatment of HSV type 2 genital infection, the results suggested that PFA should be followed up for the topical treatment of mucocutaneous HSV infections including genital herpes.

Round and Stebbing describe their experience with poly(C,S^4U_{10}), a single-stranded copolymer containing 9% cytidine residues and 91% 4-thiouridine residues. This compound proved effective in the treatment of influenza A virus infections in hamsters and ferrets. The anti-influenza activity of poly(C,S^4U_{10}) may at least in part be mediated by an inhibition of the virus-associated RNA replicase.

The antiviral potentials pf quercetin, a flavonoid, are addressed by Veckenstedt and Pusztai. This compound protected mice against a lethal encephalomyocarditis virus infection. Since silica abolished the protective activity of quercetin, it was believed that macrophages played an important part in the mechanism of antiviral action of the compound. They could do so by preventing the spread of virus through the organism.

Along the same line, Hirsch and Griffin found that mice, when growing older, acquired resistance to Sindbis virus infection, which seemed to be associated with an accelerated clearance of infectious virus from the blood stream. As for the role of macrophages in the study of Veckenstedt and Pusztai, the mechanism(s) responsible for the more rapid decline of infectious Sindbis virus from the blood of older mice deserve further investigation.

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Finally, McGraw et al. describe a new adamantane derivative, *N,N'*-bis(ethylene)-*P* (1-adamantyl)-phosphonic diamide, that inhibits Rous sarcoma virus replication in much the same manner as reported for the parent compound, 1-adamantanamine hydrochloride.

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