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EDITORIAL

Dear Reader,

For its first anniversary, Antiviral Research publishes a double issue containing papers from nine contributors.

Five papers deal with experimental herpesvirus infections. In two of them, Armerding and colleagues describe the effect of cyclosporin A on herpesvirus infection in mice. Cyclosporin is a fungus product which has received considerable interest in the field of tissue transplant rejection, as it seems to trick the recipient host into becoming tolerant against the transplant. Acquisition of immunity against certain virus infections (e.g. influenza) seems not to be affected by the drug, while primary immunization against other viruses, in casu herpesvirus, seems to be severely reduced, resulting in increased severity of the infection. Importantly, cyclosporin does not interfere with existing specific immunity against herpesvirus. In their second paper the authors study the mechanism by which cyclosporin prevents immunization against the virus; they present evidence supporting the hypothesis that cyclosporin affects macrophages and natural killer (NK) cells. They also express the view that the drug inhibits the phagocytic response of macrophages to herpesvirus and change NK cells (or pre-NK cells?) so that they do not respond to the interferon induced by the virus infection, and thereby fail to be activated. The type of study presented by Armerding et al. supports the view that biological response modifiers (of which cyclosporin is a rather negative example) are a reality and may have great clinical relevance.

The other three papers on herpesvirus deal with the direct antiviral effects of what have now become 'classical' antiherpes compounds, adenine arabinoside analogues and 5′-substituted deoxypyrimidine nucleosides. Various substances, including the promising 5′-bromovinyldeoxyuridine, are tested either in in vitro systems or in a rabbit encephalitis model.

Koff and colleagues describe inhibition of a togavirus in human leukocytes by combined treatment with ribavirin (an inhibitor of inosine-monophosphate dehydrogenase) and mercapto-tetrahydro-furylpurine (an inhibitor of hypoxanthine-guanine phosphoribosyltransferase). Their experiments suggest that it may be useful to test this combination in in vivo model systems of Dengue virus infections.

Eriksson et al. elaborate on previous findings that certain, but not all pyrophosphate analogues (of which the antiherpes compounds phosphonoformate and phosphonoacetate are the prototypes) are inhibitors of retroviruses.

The last two papers of this issue deal with interferon. Both papers compare certain biological activities of different types or subtypes of interferon. This is a field of growing

interest. The multiplicity of biological actions attributed to interferon, on the one hand, and the multiplicity of molecular variants of interferon which seem to exist in all animal species, on the other hand, strongly support the concept that each molecular variant has a defined role in regulation of host response to foreign substances. The first of the two papers points to the existence of specific cellular receptor systems for specific interferon variants. The second paper points to the different pharmacokinetic behaviour of different molecular types of interferon.

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