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could conceivably be linked to the general decrease in RNA synthesis.

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Methylation of Ribonucleic Acid in Cells Infected with Herpes Simplex Virus

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A decrease both in the synthesis and in the methylation of rRNA and tRNA has been found after infection of mammalian cells with poliovirus (Grado, Friedlinder, Ihl & Contrevas, 1968) and foot-and-mouth-disease virus (Vande Woude, Arlinghaus & Polatnick, 1967). Tumours induced by SV40 (Mittelman, Hall, Yohn & Grace, 1967), adenovirus 12 (Macfarlane & Shaw, 1968; Macfarlane, 1969), murine leukaemia virus (Silber, Goldstein, Berman, Decker & Fried, 1967) and Marek's-disease virus (Hacker & Mandel, 1969) possess increased activities of tRNA methylases that apparently (Baguley & Staehelin, 1968) give rise to quantitative and qualitative changes in a tRNA substrate. It has been shown (Low, Hay & Keir, 1969) that the DNA of herpes simplex virus is not methylated at the polynucleotide level.

We have examined the synthesis and methylation of rRNA and tRNA in baby-hamster kidney (BHK 21) cells after infection with herpes simplex virus. Incorporation of radioactive uridine and methionine into tRNA between 4 and 8 h after infection was inhibited by one-third and one-half respectively compared with uninfected cells. The synthesis and methylation of rRNA was inhibited to an even greater extent.

At 4 h after infection the activity of tRNA methylase had fallen to 80% of the control value both in exponentially growing cells and in cells incubated with 5% of the normal serum concentration. Such cells before infection exhibit markedly decreased activities of tRNA methylase.

Inhibition of RNA methylation after infection with herpes simplex virus is not due to the presence of an *S*-adenosylmethionine-cleaving enzyme and

Evidence for the Induction of a New Deoxycytidine Kinase in Cells Infected with Herpes Virus

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After the infection of animal cells by herpes virus a number of enzymes show elevated activities, and there is evidence that some of these are virus-coded (Keir, 1968). As part of a survey of enzymes of nucleic acid metabolism in virus-infected cells, we have studied the deoxyribonucleoside kinases of baby-hamster kidney (BHK) cells before and after infection with herpes virus.

It was found that the kinase activities towards deoxyadenosine and deoxyguanosine were heat-stable and detectable in approximately equal amounts before and after infection. The phosphorylating activities towards deoxycytidine and deoxythymidine, however, showed lower specific activities in uninfected cells than the purine kinases and were much more heat-labile. In virus-infected cells both activities rose about 15-fold by 12 h after infection.

The induction of deoxythymidine kinase has already been described (Kit & Dubbs, 1963; Klemperer, Haynes, Shedden & Watson, 1967). The following observations show that the increment in deoxycytidine kinase is due to the development of a distinct activity: (i) deoxycytidine stabilizes the virus-induced deoxycytidine but not deoxythymidine kinase against heat inactivation; (ii) deoxycytidine 5'-triphosphate at 20 μ M completely inhibits the deoxycytidine kinase activity but has no effect on deoxythymidine kinase.

Experiments with puromycin and actinomycin D showed that protein and RNA synthesis were