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Characterization of Proteins Induced by *Herpesvirus saimiri*: Comparative Immunoprecipitation and Analysis of Glycosylation

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Marmosets of the genus *Saguinus* (*S. nigricollis, S. fuscicollis,* and *S. oedipus*) are highly susceptible to tumor induction by *Herpesvirus saimiri* and die from a rapidly growing neoplastic disease [1–3] following viral infection. Owl monkeys (*Aotus trivirgatus*) show a similar course of disease. The appearance of tumors in these animal is often delayed and about 20% of the owl monkeys do not develop tumors [4, 5]. *Herpesvirus saimiri*-infected New Zealand White Rabbits (NZWR) show a disease pattern similar to the primate tumors; the incidence of neoplastic disease after infection with *H. saimiri* ranges from 20% to 75% in different studies [6–8]. The specificity of antibodies in the various experimental and natural hosts (*Saimiri scireus*) was determined by immunoprecipitation of viral polypeptides obtained from owl monkey kidney (OMK) cells infected with *H. saimiri* in the presence of labeled precursors. Whereas we describe the unglycosylated virus-induced proteins involved in another report [9], we include in this report the description of the glycoproteins in *H. saimiri*-infected cells.

In this study, we infected OMK cells with *H. saimiri* (strain 11) and an attenuated mutant of *H. saimiri,* originally isolated by Schaffer [10]. At various times (6–8 h, 15–17 h, and 24–26 h after infection), virus-induced cell proteins were labeled with ^{35}S-methionine (20 μCi/ml) and the viral proteins were immunoprecipitated with various sera from infected animals (the sera from the natural host *Saimiri sciureus* and from infected owl monkeys were a gift from L. Falk, New England Primate Center). A class of early proteins was obtained from infected cells by treatment with azetidine (Sigma); they were precipitated with the same sera.

The protein profiles obtained after precipitation with sera from *H. saimiri*-infected experimental hosts (owl monkeys, white lip marmoset, and NZWR) differed from those obtained with sera from the natural hosts. Proteins precipitated with sera from *Saimiri sciureus* were mostly late proteins and components of the virion. Sera from the experimental hosts precipitated a limited number of proteins (p 152, p 127, p 115, p 80, p 55–57, p 53, and p 50). Three of them (p 115, p 80, and p 55–57) were not found with sera of the natural hosts. p 115 and p 55–57 are already synthesized at an early stage after infection; the synthesis of p 115, however, is inhibited by treatment with azetidine and thus may belong to a second group of early proteins. p 127 and p 152 are components of the viral capsid. Natural and experimental hosts of *H. saimiri* revealed distinct profiles, whereas some similarities existed among the various types of experimental hosts. The reason for the observed differences may be the expression of viral polypeptides in the various host cells or host-specific differences in the immune system. No difference could be detected between the patterns obtained with the oncogenic *H. saimiri* 11 and the attenuated strain (*H. saimiri* 11 att.).

In a second line of experiments, we characterized glycosylated proteins produced in *H. saimiri*-infected OMK cells. Two different methods were used: (1) in vivo labeling with ^{14}C-glucosamine in a medium containing fructose instead of glu-

cose and followed by immunoprecipitation; (2) in vitro labeling of the glycoproteins separated on SDS-polyacrylamide gels with ^{125}I-labeled lectins. Both experiments showed a similar pattern of glycosylated viral proteins produced in infected cells and can be used interchangeably. Seven glycosylated proteins could be identified (p 152, p 140, p 127, p 88, p 67, p 53, p 50) with both methods.

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