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NOTES

Effects of Ultraviolet Irradiation on the Transforming and Plaque-forming Capacities of Simian Adenovirus SA7

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Ultraviolet (UV) irradiation of SV40 and polyoma virus (R. I. Carp and R. B. Gilden, Virology **27**:639, 1965; V. Defendi, F. Jensen, and G. Sauer, p. 645, *in* J. S. Colter and W. Paranchych [ed.], *The Molecular Biology of Viruses*, Academic Press, Inc., New York, 1967) and adenovirus type 12 (Z. Gilead and H. S. Ginsberg, J. Bacteriol. **92**:1853, 1966) inactivated their capacity to induce a new complement-fixing antigen (T antigen) at a rate that was one-third to one-fourth the rate of inactivation of virus infectivity. In addition, the capacity of polyoma virus to transform cultured cells was more resistant to inactivation by UV- and γ-irradiation than was the capacity to initiate plaques (T. L. Benjamin, Proc. Natl. Acad. Sci. U.S. **54**:121, 1965; C. Basilico and D. di Mayorca, Proc. Natl. Acad. Sci. U.S. **54**:125, 1965; R. Laterjet, R. Cramer, and L. Montagnier, Virology **33**:104, 1967). The experiments reported here demonstrate that the radiation-sensitive region of the virus responsible for transformation by an adenovirus is also smaller than the region responsible for plaque formation.

The source and passage history of the simian adenovirus, SA7, and the methods for the preparation of cell cultures, virus assays, and transformation of hamster embryo cells have been described elsewhere (B. Casto, J. Virol. **2**:376, 1968). The SA7 virus used in this study was propagated in BSC-1 cells and had a titer of 5×10^7 plaque-forming units (PFU)/ml in primary African green monkey kidney cell cultures. In the two experiments reported here, 3- and 5-ml virus samples, contained in 60- or 100-mm plastic petri dishes (Falcon Plastics, Inc., Los Angeles, Calif.), respectively, were irradiated on a rotary shaker at a distance of 10 cm from the UV-light source (two Sylvania G15T8 lamps). Immediately after irradiation, the samples were frozen at -60 C; on the following day, they were tested for infectivity (plaque formation in LLC-MK_2 cells) and transforming capacity (in hamster embryo cell cultures).

The inactivation of SA7 virus infectivity followed first-order kinetics and remained linear throughout the irradiation period. A comparison of the inactivation curves of the transformation capacity (focus-forming units or FFU) and the infectivity (PFU) are presented in Fig. 1. Analysis of the two slopes reveals that the inactivation rates for PFU and FFU were approximately 3.5 and 1.7% per second, respectively. In a second experiment, the rates were 5.7 and 1.9% per second. The calculated FFU/PFU slope ratios for the two experiments were 0.485 and 0.333.

Since undiluted virus from the various irradiation periods was used in the transformation assay, the possibility existed that the greater resistance to inactivation demonstrated by the FFU curve could be attributed to multiplicity effects, although in the two experiments in the transformation assay the input multiplicity of infective virus, prior to inactivation, was only 2 PFU/hamster embryo cell. Table 1 shows the results of an experiment in which virus samples from the 0-, 10-, and 20-sec inactivation periods

TABLE 1. *Effect of dilution of UV-irradiated samples on the numbers of adenovirus (SA7)-transformed cell foci*

Virus dilution	Irradiation (sec)		
	0	10	20
Undiluted	85[a]	39	19
1:2	39	24	8
1:4	13	6	5

[a] Total number of foci observed 3 weeks after inoculation and transfer of three 60-mm plates of hamster embryo cell culture.

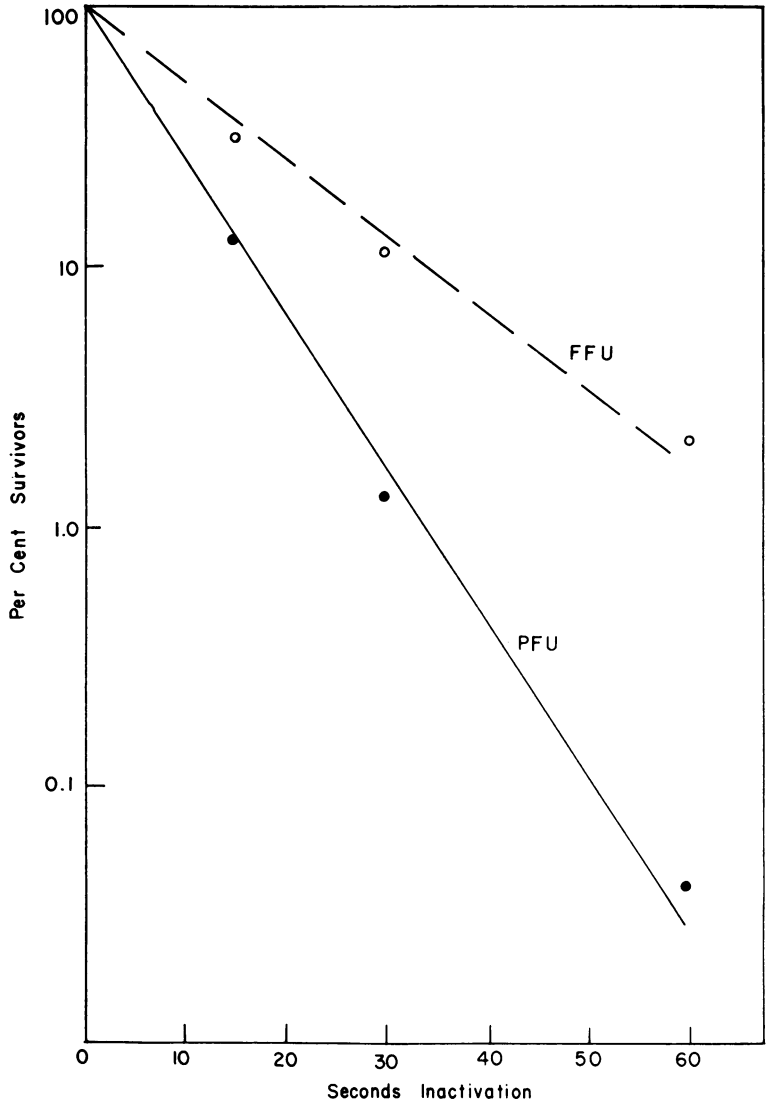

FIG. 1. *Effect of UV irradiation on the transforming capacity and virus infectivity of simian adenovirus, SA7. The virus suspension was irradiated at a distance of 10 cm from the UV-light source (two Sylvania G15T8 lamps) with continuous rotation. The points represent the surviving fraction of PFU or FFU (expressed as per cent) at the indicated times, as compared to the nonirradiated control. Each point on the FFU curve is based on the number of foci arising from four plates (a total of 356 foci at time 0).*

were diluted twofold and tested by the transformation assay. As can be seen, reasonable linearity occurred with dilution. Therefore, virus multiplicity effects did not appear to be responsible for the increased survival of the transforming ability. These results strongly suggest, as has been found with polyoma virus (*see* previous references), that the relative irradiation target size of the viral genome, which is responsible for adenovirus transformation, is less than half of that which is necessary to code for virus replication.

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