

Failure of Inhibition of Polyoma Virus Replication by Distamycin A

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Distamycin A, Polyoma Virus

Distamycin A caused no significant inhibition of polyoma-virus growth or capsid and mRNA formation in embryonic mouse cells.

Distamycin A (DA), an antibiotic from *Streptomyces distallicus*¹, is known to interfere with the replication of some large DNA viruses, e.g. vaccinia virus², Epstein Barr virus³, and herpes simplex virus⁴ (own unpublished results). However, the reports on DA action on polyoma virus (PV), a small DNA virus, are contradictory in the literature: Verini and Ghione² were not able to find a significant effect, whereas later Castro *et al.*⁵ reported inhibition of PV yield by the factor 10² and 10^{7.5} in the presence of 20 and 100 μ M of DA, respectively.

We wish to report on some investigations of DA action on PV functions. DA was used in a concentration of 200 μ M (Boehringer Co, lot 7943305). PV was a wild type (obtained from Dr J. Žemla, Bratislava) and the cells used were secondary mouse embryo cells (MEC).

Two types of experiments were performed.

1. MEC were infected with PV (5 pfu per cell) and DA was continuously present (medium once

changed at 36 h p.i.) until cells and virus were harvested at 70 h p.i. We assayed for PV yield (hemagglutinin and infectivity) and capsid formation (immunofluorescence test) (Table I, columns A).

2. For investigation of the DA effect on the current virus-specific RNA formation in PV infected cultures the DA was not added until 60 h p.i., as described for experiments with cytosine arabinoside and SV40 and PV^{7,8}. Total RNA was labelled one hour later for a two hour period by addition of 5,6-(³H)-uridine (40 μ Ci per ml) to the DA containing medium. RNA was isolated and 20 μ g were hybridized with denatured PV DNA on nitrocellulose filters as described elsewhere⁶ (Table I, columns B).

We observed no significant inhibition of PV growth and protein synthesis (Table I, A). The partial inhibition of total RNA formation in DA treated cells and also the finding that DA does not specifically interfere with the PV specific transcription (Table I, B) are in agreement with results in human fibroblasts infected with herpes simplex virus type 1 (A. Burger, G. Brandner, unpublished results).

In summary, our results provide no evidence for a significant inhibition by DA of PV replication. Thus, we are able to confirm the findings of Verini and Ghione².

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DA [μ M]	A: Continuous presence of DA			B: DA present 60–63 h p.i.	
	PV infectivity [TCID ₅₀ per ml]	HA [titer]	PV capsid [% positive cell nuclei]	Spec. radioact. of total RNA [cpm \times 10 ⁻⁴ per μ g]	PV specific RNA [% of total RNA radioact.]
0	10 ^{5.0}	1 : 200	80–90	2.8	0.22
200	10 ^{4.5}	1 : 400	80	1.3	0.27

Table I.
Effect of DA on PV replication.

- ¹ F. Arcamone, S. Penco, V. Nicoletta, P. Orezzi, and A. M. Pirelli, *Nature* **203**, 1064–1065 [1964].
- ² M. A. Verini and M. Ghione, *Chemotherapy* **9**, 144–157 [1964].
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- ⁴ G. H. Werner, P. Ganter, and Y. de Ratuld, *Chemotherapy* **9**, 69–79 [1964].

- ⁵ A. Castro, G. Carrera, and G. Russo, *Giorn. Batteriol. Virol. Immunol.* **63**, 713 [1970].
- ⁶ G. Brandner and N. Mueller, *Cold Spring Harbor Symp. Quant. Biol.* **39**, 305–308 [1974].
- ⁷ G. Brandner and N. Mueller, *FEBS Letters* **42**, 124–126 [1974].

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