Experiments carried out in our laboratory (to be reported elsewhere) suggest that the effect of mestranol in synthetic grit is avian specific, for grit force fed to rodents was voided within a few hours without a significant effect on their fecundity.

This work was supported by the US Bureau of Sport Fisheries and Wildlife, the Massachusetts Division of Fisheries and Game, the University of Massachusetts and the Wildlife Management Institute.

BERNARD C. WENTWORTH
Massachusetts Cooperative Wildlife Research Unit,
University of Massachusetts,
Amherst, Massachusetts 01002,

Received October 10; revised November 18, 1968.

[1] Davis, D. E., *Anat. Rec.*, **134**, 549 (1959).
[2] Elder, W. H., *J. Wildlife Management*, **28**, 556 (1964).
[3] Lofts, B., Murton, R. K., and Thearle, R. J. P., *J. Reprod. Fertil.*, **15**, 145 (1968).
[4] Wetherbee, D. K., Coppinger, R. P., Wentworth, B. C., and Walsh, R. E., *Univ. Mass. Exp. Stat. Bull.*, **543**, 1 (1964).
[5] Wentworth, B. C., Hendricks, B. G., and Sturtevant, J., *J. Wildlife Management*, **32**, 879 (1968).
[6] Snedecor, J. G., *J. Exp. Zool.*, **110**, 205 (1949).
[7] Van Tienhoven, A., *Poultry Sci.*, **36**, 628 (1957).

Infectivity associated with Simian Adenovirus Type SA7 DNA

SIMIAN adenovirus type SA7 (ref. 1) induces tumours in newborn hamsters[2] and transforms cells in tissue culture[3]. We have reported the induction of tumours in newborn and weanling hamsters by DNA isolated from SA7 virus[4]. We now describe cytocidal infectivity associated with this DNA.

DNA was extracted by a modified phenol procedure from virus that had been concentrated and twice banded in a CsCl gradient[5]. 20 μl. of betamercaptoethanol and 1 mg of papain were added to each ml. of virus previously dialysed against 0·15 M NaCl containing 0·01 M sodium phosphate, *p*H 7·0, and 0·001 M EDTA. After incubation for 10–12 min at 37° C, 20 mg/ml. solid sodium dodecyl sulphate was added and incubation was continued for 7 min at 37° C. This mixture was extracted with aqueous phenol (containing 0·2 per cent 8-hydroxyquinoline) at room temperature. After two re-extractions with fresh phenol, the DNA preparations were dialysed against 0·015 M NaCl containing 0·0015 M sodium citrate. After dialysis the preparations were adjusted to 0·15 M NaCl and 0·015 M sodium citrate and stored at 4° C with a drop of chloroform.

The infectivity of the DNA extracts was determined: monolayer cultures of CV-1 cells[6] in T_{30} flasks were rinsed once with phosphate buffered saline and once with Eagle's BME (ref. 7) containing 0·01 M *tris* buffer, *p*H 7·4 (BME–*tris*). Each culture was then incubated for 30 min at room temperature with BME–*tris* containing 500 μg/ml. DEAE-dextran. This solution was removed and the DNA was added (0·5 ml. in BME–*tris*) and allowed to absorb for 30–45 min at room temperature. Five ml. of Eagle's MEM medium[8] containing 5 per cent foetal calf serum was added to each flask without removing the DNA inoculum. After 2 h at 37° C the medium was replaced with fresh medium. DNase (Worthington purified) was used in the presence of 0·005 M magnesium acetate. Inocula treated with DNase or antiserum were incubated for 30 min at room temperature before being added to the culture. Untreated DNA inocula were incubated in the same conditions.

The results of two typical experiments are summarized in Table 1. Nucleic acid derived from SA7 produced a cytopathic effect identical to that caused by intact virus. The infectivity of these preparations was completely destroyed following exposure to DNase. Treatment with SA7 type-specific antiserum had no effect on the infectivity. Heating at 56° C for 1 h did not destroy the infectivity, whereas intact adenovirus is rendered non-infectious in these conditions. The viral progeny produced in cultures with CPE ranged in titre from $10^{4\cdot6}$ to $10^{7\cdot3}$ $TCID_{50}$/ml. and has been identified as SA7 by serum neutralization tests.

Table 1. INFECTIVITY OF DNA EXTRACTS OF SA7

Experimental conditions	Infectivity No. + CPE/No. cultures inoculated	Average titre produced $TCID_{50}$/ml.
Experiment No. 1		
5 μg DNA	4/4	$10^{7\cdot3}$
5 μg DNA + 2 μg DNase	0/4	
Experiment No. 2		
5 μg DNA	6/6	$10^{4\cdot6}$
5 μg DNA heated at 56° C for 60 min	2/2	$10^{5\cdot4}$
5 μg DNA + SA7 antiserum*	6/6	$10^{5\cdot3}$
5 μg DNA + 2·5 μg DNase	0/6	

* DNA was treated with an equal volume of SA7 antiserum having a titre of 1 : 512.

These findings suggest that DNA extracted with phenol from SA7 virus is infectious. The DNA isolated from human adenoviruses is not[9].

J. PAUL BURNETT
J. A. HARRINGTON

Lilly Research Laboratories,
Indianapolis, Indiana 46206.

Received September 16; revised November 11, 1968.

[1] Malherbe, H., and Harwin, R., *S. Afric. Med. J.*, **37**, 407 (1963).
[2] Hull, R. N., Johnson, I. S., Culbertson, C. G., Reimer, C. B., and Wright, H. F., *Science*, **150**, 1044 (1965).
[3] Altstein, A. D., Sarycheva, O. F., and Dodonova, N. N., *Science*, **158**, 1455 (1967).
[4] Burnett, J. P., and Harrington, J. A., *Proc. US Nat. Acad. Sci.*, **60**, 1023 (1968).
[5] Burnett, J. P., Summers, A. O., Harrington, J. A., and Dwyer, A. C., *Appl. Microbiol.*, **16**, 1245 (1968).
[6] Jensen, F. C., Girardi, A. J., Gilden, R. V., and Koprowski, H., *Proc. US Nat. Acad. Sci.*, **52**, 53 (1964).
[7] Eagle, H., *Science*, **122**, 501 (1955).
[8] Eagle, H., *Science*, **130**, 432 (1959).
[9] Green, J., Pina, M., Kimes, R., Wensink, P. C., MacHattie, L. A., and Thomas, jun., C. A., *Proc. US Nat. Acad. Sci.*, **57**, 1302 (1967).

Role of the LDH-elevating Virus in Leukaemia Therapy by Asparaginase

THE dramatic cure of certain leukaemia and lymphomas in mice by the administration of the enzyme L-asparaginase[1–4] has led to an extensive clinical evaluation of this therapy in patients with leukaemia and other forms of cancer. Unfortunately, the results so far in human beings have not compared favourably with those obtained in mice, although significant temporary remissions have been obtained in a substantial number of patients[5–7]. It is important to examine the mechanism of action of asparaginase in the hope that the curative effects on disseminated neoplasms in mice might also be realized for other species.

Studies on the lactate dehydrogenase-elevating virus (LDH virus) have shown that one of its important biological characteristics is the induction of a moderate impairment in the protein clearance capacity of the infected host[8–10]. This has been studied chiefly as specific enzyme clearance from the blood of mice following intravenous or intraperitoneal injection of lactate dehydrogenase (LDH) or EC-2 asparaginase (ref. 8 and my unpublished work with H. A. Campbell and C. C. Stock).

We found that the clearance or disappearance rate of injected asparaginase in mice infected with LDH virus is reduced by a factor of five to ten, as compared with normal mice ($T_{1/2}$ = 2–4 h in normal mice, 20–30 h in infected mice), and that all tested mouse leukaemias or tumour lines routinely used for asparaginase therapy carried the LDH virus (ref. 11 and my unpublished results with H. A. Campbell and C. C. Stock). The unappreciated presence of a silent, benign virus has