Table 1. ANTIBODY RESPONSE IN HANDLED AND UNHANDLED RATS

	Primary response								Secondary response		
									Booster	Booster	Booster
Day	4		7		14		21	28	+4	+7	+14
	Total antibody	19S antibody	Total	19S	Total	19S					
Handled											
No. of animals	53	47/50	52	42	54	47	31	53	54	53	50
Mean titre	1 : 44	1 : 10	1 : 384	1 : 11	1 : 2816	1 : 5·5	1 : 9728	1 : 1152	1 : 3840	1 : 3072	1 : 1280
Mean dilution number	4·1	2·0	7·2	2·1	10·1	1·1	11·9	8·8	10·5	10·2	9·0
Unhandled											
No. of animals	54	40/43	54	49	52	44	26	54	51	51	54
Mean titre	1 : 24	1 : 5	1 : 272	1 : 8	1 : 1152	1 : 6	1 : 3840	1 : 768	1 : 3072	1 : 1408	1 : 832
Mean dilution number	3·2	1·0	6·7	1·6	8·8	1·2	10·5	8·2	10·2	9·1	8·3

consultation; and Suzanne Donohue for technical assistance. This work was supported by the Veterans Administration Hospital and a grant from the US Public Health Service. S. L. holds a US Public Health Service research scientist award.

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Received September 23, 1968.

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Differential Inhibition by Phagicin of DNA Synthesis in Cells infected with Vaccinia

CENTIFANTO[1,2] reported the isolation of an antiviral agent (phagicin), active against vaccinia and herpes simplex viruses, from cultures of *Escherichia coli* infected with λ bacteriophage. Its production is associated with the phage infection and does not take place in uninfected cells.

In the work reported here, phagicin was prepared from a strain of *E. coli* (K12 (λ−)) infected with λ-b2b5c (kindly supplied by Dr Centifanto). The technique for the production of phagicin[2] consists essentially of lysis of *E. coli* by the coliphage, followed by purification of the crude lysate by centrifugation and gel filtration on 'Sephadex' columns. Chick embryo cell cultures were grown in 50 ml. flat bottles in Eagle's minimal essential medium with 10 per cent heat-inactivated calf serum; these were inoculated with vaccinia virus (concentration 10^{-3} PFU/ml.). Phagicin was added to test for toxicity towards the host cells, the final concentration being slightly below that necessary for total inhibition of plaque formation. At this concentration the number of plaques was reduced by 94·6 per cent compared with that of uninfected controls. After 48 h with phagicin present, the chick embryo cells showed no morphological evidence of toxicity.

Using synthesis of DNA as a more sensitive indicator of possible cell damage, the experiment was repeated with a three-fold increase in concentration of phagicin. Tritiated thymidine (specific activity 3 Ci/mmole ; Radiochemical Centre, Amersham), was added 3 h after the phagicin to a final concentration of either 1·0 or 2·0 μCi/ml. Cultures were terminated by fixation 8 h after infection, and autoradiographs prepared according to the technique of Drown *et al.*[3]. Results showed no reduction in either the percentage of cell nuclei labelled or the intensity of their labelling. At the same time, the centres of viral DNA synthesis present in the cytoplasm of control culture cells were suppressed completely.

The results indicate a differential inhibition of viral DNA synthesis by phagicin at a concentration which has no effect on host-cell DNA synthesis. Tests were repeated, delaying the addition of phagicin up to 3·5 h after infection of the cells without loss of antiviral effect.

We thank the British Empire Cancer Campaign for Research and Hoechst Pharmaceuticals for financial support.

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Received July 29; revised September 10, 1968.

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Platelet Accumulation observed by Electron Microscopy in the Early Phase of Renal Allotransplant Rejection

THE rejection of skin allografts depends on an immunological response of the recipient[1] which can be transferred with whole cells[2]. The contribution of soluble antibody to rejection of solid tissue grafts remains an open question[3]. Although antibody may react directly with donor cells to produce a cytotoxic effect, other destructive mechanisms are equally possible. For example, Gardner, Guttman and Merrill[4] have observed an ischaemic response during rejection of rat kidney transplants. Indeed, interference with the blood supply leading to cell destruction in grafts undergoing rejection has previously been deduced from metabolic studies[5,6]. Such vascular blockades could be produced by platelet aggregates, as noted by Porter[7] in acute rejection of human kidney transplants. The work reported here shows that substantial numbers of platelets