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Propagation of Measles Virus in Human Carcinoma Cells. (22643)

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Because they could not regularly be reproduced, early claims of the propagation of measles virus in tissue cultures have not met with wide acceptance. However, Enders and Peebles(1) reported that an infectious agent could be isolated from the throat or blood of measles patients and readily grown in cultures of human kidney cells.

It seemed desirable to find other cells in which the virus may be propagated since human kidneys are not readily available and monkey kidney cells, although susceptible, frequently carry a latent agent which may cause lesions similar to those of measles virus. To this end, Dr. George Foley put at our disposal the KB strain of human epidermoid carcinoma cells, originally isolated by Dr. Harry Eagle(2). These cells grow profusely in a relatively simple and well defined medium containing 10% horse serum(3,4).

In tubes to which a suspension of these cells were added, confluent sheets of growth were seen after 2 or 3 days. At this time, the cells were inoculated with the fluid from the 23rd passage in human kidney cell cultures of a strain (Edmonston) of measles virus(1).

Daily microscopic examination of the cultures revealed on the 4th or 5th day, 6 to 10 areas of "giant cell" or syncytial formation comparable to those described by Enders and Peebles(1) in cultures of human renal cells. By the following day, however, these "lesions" had fallen off the glass and the clear area was quickly overgrown by cells of normal appearance. Two to 3 days later, (Fig. 1), a second but larger group of giant cells appeared both in the same areas and elsewhere throughout the cell sheet. This process of simultaneous cell destruction and multiplication continued until complete destruction of the culture by the virus at the end of 5 weeks.

During this period, culture fluid from the fast growing yet infected primary culture was used repeatedly to start subcultures in which the same changes were observed and in which the cells underwent the same fate as those of the primary culture within about the same period. Only in the final stages were acidophilic inclusion bodies seen in several of the cells that were comparable to those described as occurring abundantly in normal human and monkey renal cells(1). Further evidence that these lesions were caused by measles virus was afforded by the specific fixation of complement using as antigens fluids from infected cultures in the presence of measles convalescent serum. Only those fluids, however, taken from cultures that were inoculated with the virus 3 to 4 weeks previously were shown to fix complement under these conditions.

From these observations it is concluded that the measles virus may be propagated in

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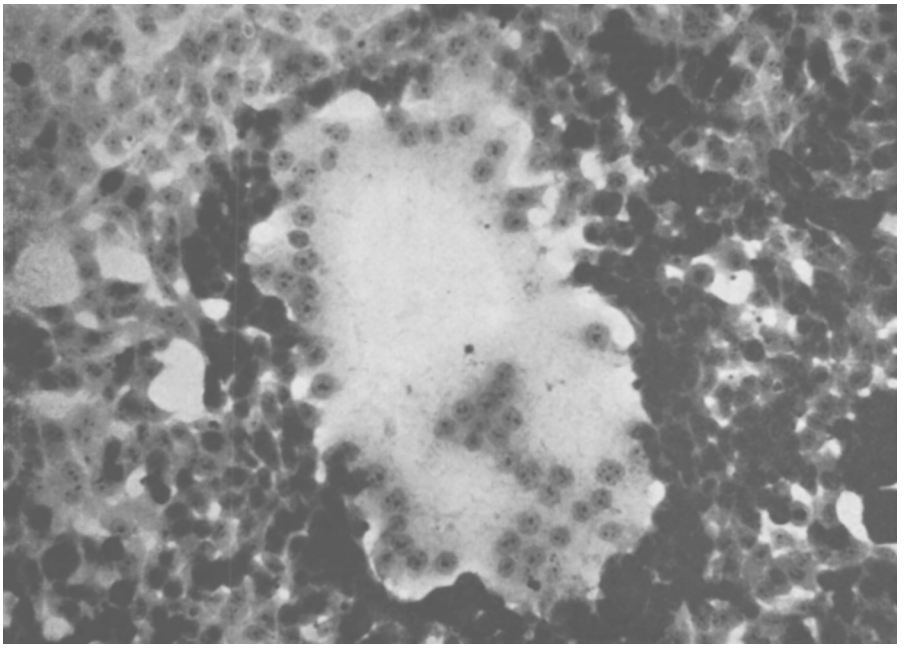

FIG. 1.

the KB strain of human carcinoma cells.

1. Enders, J. F., and Peebles, T. C., PROC. SOC. EXP. BIOL. AND MED., 1954, v86, 277.

2. Eagle, H., *ibid.*, 1955, v89, 362.

3. ———, *J. Exp. Med.*, 1955, v102, 37.

4. ———, *ibid.*, 1955, v102, 595.

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Lipid Metabolism in Kidney Undergoing Compensatory Hypertrophy.* (22644)

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Previous studies involving "regenerating" liver in rats(1), and the prostate and seminal vesicle tissue of non-castrated and castrated rats with and without testosterone treatment (2), suggested that there was an increased P^{32} uptake in all of the Schmidt-Thannhauser (3) fractions that was associated with cellular hypertrophy. Neither of these conditions can be considered as a hypertrophy, pure and simple, however, since the hypertrophy seen in the first 24 hours in regenerating liver is, in a sense, a preparative state for an ensuing cellular hyperplasia in that it occurs prior to the appearance of mitosis, while that in the prostates and seminal vesicles occurred in the

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