over electron microscopy is that particles can be counted in infected yolk-sac suspensions without purification or concentration. The relationship between particle counts and infectivity for chick embryos and HeLa cells is being investigated, using several strains of virus.

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Correlation between the Surface Structure of Viruses and Latency

By the use of DEAE-cellulose chromatography, we have endeavoured to obtain in their pure form various factors developed by adenoviruses during their propagation in tissue culture cells[1–4]. HeLa or human amnion cells were inoculated with adenoviruses of types 1, 2, 3, 5, 7*a*, 9, 10, 11 and 15. On the 3rd–5th day the culture fluids were collected and were dialysed against 0·005 *M* borate buffer solution, *p*H 8·10. The dialysed fluid was then introduced into a DEAE-cellulose column (1 cm. diam., 10 cm. high) which was previously balanced by 0·005 *M* borate buffer solution, *p*H 8·10. Fractionation was carried out by adding 0·005 *M* borate buffer solution, *p*H 8·10, containing sodium chloride, to the column in linearly increasing concentrations of 0–0·8 *M*. 5-ml. fractions were collected and analysed with the view of detecting hæmagglutinins (by the reaction of hæmagglutination with the erythrocytes of rats and monkeys), also an early cytopathogenic factor[5,6], the complement-fixing antigen and an infectious virus.

Certain results obtained by these experiments are illustrated in Fig. 1. Further detailed results will be published elsewhere.

Attention is directed to the fact that adenoviruses of types 3, 7*a*, 9, 10 and 15, which cause acute diseases in human beings, can be eluted from the DEAE-cellulose column with smaller concentrations of sodium chloride (0·2–0·3 *M*) than types, 1, 2 and 5, which act as the latent ones eluted at 0·45 *M*–0·55 *M* sodium chloride. The intermediate position is occupied by adenovirus, type 11. The observed regularity testifies to the difference in the structure of proteins which form the surface of adenoviruses, causing latent and acute infections respectively. One may imagine that the proteins forming the membrane of latent adenoviruses contain a greater number of groups which are negatively charged at *p*H 8·10 than the proteins forming the surfaces of 'acute' adenoviruses.

The characteristics instrumental in developing the latent nature of some infections caused by definite viruses are not known. What is known, however, is that the viruses which cause latent infections sometimes lead to the development of acute diseases. The viruses which are characterized by causing acute infections alone, never behave as the latent viruses. It can therefore be assumed that the cause of latency lies not in the properties of the cells affected in this or that case but in the virus itself.

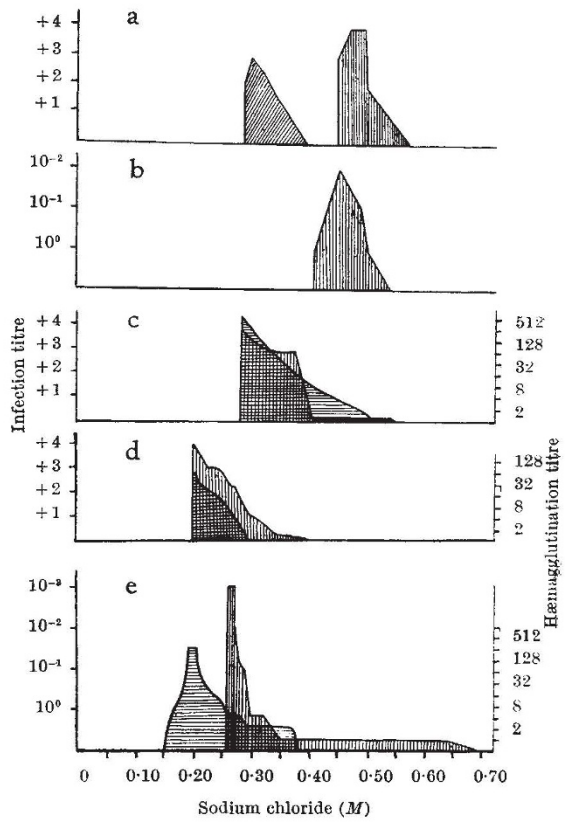

Fig. 1. Fractionation of infected cultural fluids of adenoviruses of different serotypes on DEAE-cellulose. Vertical shading, virus; horizontal shading, hæmagglutinin; oblique shading, early cytopathogenic factor. *a*, Serotype 5 (similar to type 2); *b*, serotype 1; *c*, serotype 11; *d*, serotype 7*a* (similar to type 3); *e*, serotype 10 (similar to types 9 and 15)

In attempting to interpret the possible significance of this regularity we focused our attention on the ability of polyanions to hamper the course of the infectious process when tissue cultures were inoculated with polio virus[7]. The question arises whether latent viruses do not contain similar polyanionic structures in their very elementary particles (unlike the 'acute' ones)—a phenomenon which is actually reflected in the greater number of anions on their surface. Experiments in this direction are now in progress.

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