The preparations of the dermo-muscular tissue of human embryos were treated with the virus within 20 min at 4° C; residual virus was removed by triple washing in the cold Hanks's solution; then Medium 199 with 10 per cent serum and ^{14}C-adenine, 0·01 μc./ml., were added and preparations were incubated at 37° C. After exposure for 3 h, cells were washed with Hanks's solution, fixed in Carnoy's solution, covered with γ-globulin labelled with iodine-131, exposed for 20 min and then washed again. Then they were covered with film, type *MR*, and exposed with silica gel in the dark at 4° C for 7 days. Developed preparations were stained with methylene blue.

Fig. 1 demonstrates some results of the method of the double radioactive labelling. ^{14}C-adenine as nucleic acid precursor is presented as small points randomly scattered while γ-globulin labelled with iodine-131, blocking viral antigen, forms large autographs and conglomerates. So far as carbon-14 and iodine-131 have different energies of decay, it is possible to observe simultaneously the reproduction of viral nucleic acid and protein as autographs of different size in the same cell.

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Effect of Actinomycin *D* on Measles Virus Growth and Interferon Production

ACTINOMYCIN *D* combines *in vitro* with DNA in mammalian cells and results in impairment of DNA synthesis and of DNA-dependent RNA synthesis[1,2]. This results in uncoupling DNA from cellular protein synthesis.

Certain animal riboviruses (polio, mengo and Newcastle disease viruses (NDV)) have been shown to replicate in actinomycin *D* treated cultures, indicating their independence of cellular nucleic acid function[2,5].

Interferon production in cell cultures occurs in response to treatment of cells with foreign nucleic acids[6]. Because actinomycin *D* inhibits cellular RNA synthesis and hence protein synthesis, it would be expected to inhibit production of interferon.

Bukrinskaya and Zhdanov[7] demonstrated a decrease in the latent period of Sendai virus production in actinomycin *D* treated chick fibroblasts. They attempted to explain their results by suggesting that actinomycin *D* may have inhibited messenger-RNA of the cell, resulting in early direction of cellular enzymatic systems to virus production.

Treatment of chick fibroblasts with actinomycin *D* prior to inoculation with Chikungunya virus resulted in inhibition of interferon production accompanied by enhancement of viral growth[8,9]. Actinomycin *D* has also been shown to cause enhancement of growth of Semliki forest virus (SFV) and NDV (ref. 10).

This communication reports preliminary investigation of the effects of actinomycin *D* on production of interferon and virus titres in measles-infected chick fibroblast cultures.

*H.Ep.*2 cells infected with the 'Edmonston' strain of measles virus were disrupted by three cycles of freeze-thawing. This fluid, which after centrifugation and membrane filtration contained $10^{3\cdot7}$ plaque producing doses/ml., was used as inoculum for chick embryo fibroblast monolayers maintained at 37° in medium 199 with 5 per cent fœtal calf serum. After 8 days the whole cultures were disrupted as described and assayed for measles virus by plaque production in *H.Ep.*2 cell monolayers and, after high-speed centrifugation (120,000*g*), for interferon. The end-point of interferon titration was expressed as the reciprocal of the dilution causing 50 per cent inhibition of plaque formation by Sindbis virus in chick fibroblast monolayers.

Both yield of infectious virus and interferon production were compared from normal cultures and from cultures of cells pretreated with actinomycin *D* at a concentration of 0·01 μg/cm² of monolayer (0·1 μg/ml.) for 4 h.

Normal cultures yielded fewer than 10 infectious particles/ml. with an interferon titre of 12 while actinomycin-treated cells yielded $10^{2\cdot7}$ infectious particles with undetectable interferon production. These results support the view that actinomycin allows increased virus yield by inhibiting interferon production. Thus monolayers of a cell type normally showing a high degree of 'resistance' to measles virus and to which this virus must normally be 'adapted' by repeated passage are rendered more susceptible by suppression of interferon production.

Further experiments are now being carried out to characterize this interferon and examine production of measles interferon in other cell systems.

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Lipid in a Purified Preparation of Tomato Spotted Wilt Virus

ELECTRON micrographs of tomato spotted wilt virus (TSWV), when stained by uranyl acetate or lead hydroxide, revealed the presence of an envelope surrounding an aggregate of several heavily staining spheroidal particles[1]. The envelopes stain only lightly with these reagents, suggesting that the envelopes contained lipid. Accordingly, a purified preparation of the virus was examined to test this hypothesis. By direct extraction with organic solvents the preparation was shown to contain about 20 per cent extractable lipid. Thin-layer chromatography of these extracts revealed a number of spots which gave a blue colour when treated with a molybdenum-based stain[2]. Although lipid has long been known to form part of some animal viruses there is no record of the presence of a lipid in a plant virus.

The virus was a sample of strain *E* of TSWV. The virus was extracted from systemically infected leaves of *Nicotiana glutinosa* by homogenizing them in the buffer solution at *p*H 7, removing non-viral material by alternate cycles of low- and high-speed centrifugation followed by two cycles of sucrose density-gradient centrifugation as described by Best and Palk[1]. The virus was identified in the light-scattering band of the density-gradient tubes by its infectivity and characteristic size and shape under the electron microscope[1]. After removing the sucrose and buffer salts the virus was freeze-dried and then stored over phosphorus pentoxide.

The dry virus was extracted for lipid by the micro-extraction method described by Thomas[3], using the

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