was also inoculated. After a further 3 days each of the hydrocarbon cultures was transferred again to a fresh flask containing the same mineral salt solution and hydrocarbon. The two successive transfers to media containing hydrocarbon were required in order to reduce the amount of glucose transferred to a negligible proportion and to rule out the possibility of glucose being carried forward with the cells and being used as nutritional source. It was found that the cell counts after 3 days incubation in hydrocarbon medium of the second transfers were the same following thirty-eight transfers on glucose as after only one transfer (that is, 10^9 cells/ml.). Furthermore, hydrocarbon and glucose were equally suitable as carbon sources, because cell densities on media containing either were the same. The ability to attack the hydrocarbon readily was retained by the bacteria throughout the whole series of glucose transfers, which suggests that the ability to ferment hydrocarbon is constitutive and not adaptive.

I am grateful to Sir Cyril Hinshelwood for the suggestion on which these experiments are based, and to Miss C. W. Baxter for technical assistance.

KERSTIN M. FREDRICKS

Imperial Chemical Industries, Ltd.,
Petrochemical and Polymer Laboratory,
Runcorn Heath, Cheshire.

[1] Fredricks, K. M., *Nature*, **209**, 1047 (1966).

Interference not mediated by Interferon

AUTOINTERFERENCE or autoinhibition is the phenomenon in which reduced multiplication of a virus results when a host is inoculated with a high concentration of virus. It has often been encountered in tissue culture cells with animal viruses. Members of the myxoviruses and arboviruses groups provide many examples[1–4]. These viruses can also induce interferon in the cells they infect. Interferon can be demonstrated in undiluted or low dilutions of culture fluids infected with virus used as inocula. Autointerference, therefore, has usually been attributed to the presence of interferon carried along with the inoculum. The present communication deals with an example of autointerference which results from the infection of chick embryo (CE) cells by Western equine encephalomyelitis (WEE) virus. The results showed that interferon was not involved in this instance.

The methods for the growth of WEE virus and the titration of its infectivity in CE cultures have previously been reported[5]. CE cultures were prepared according to the method of Dulbecco and Vogt[6]. Preliminary experiments indicated that cultures receiving 1,000 plaque-forming units (P.F.U.) per cell of WEE virus produced only about 10 per cent as much infectious virus as cultures infected with 0·1 P.F.U./cell. Actinomycin *D* completely inhibits the action of interferon in CE cells[7,8]. Cultures were incubated with actinomycin (5 μg/ml. for 2 h) and then infected with various concentrations of WEE virus. Control cultures which received no drug were included for comparison. Virus yields were determined 20 h later. The addition of 1,000 P.F.U./cell resulted in a reduction of virus yields from about 5,700 P.F.U./cell to about 700, just more than an eight-fold reduction. The decrease in the yield of virus from cultures receiving high doses of virus was not influenced by pre-incubation of the cultures with actinomycin *D*. This rules out the possibility that the autointerference found here was due to any interferon present in the inoculum.

Proof that the interference observed here was due to virus particles rather than interferon was also obtained with the semi-purified WEE virus. WEE virus was partially purified by high speed differential centrifugation. Virus can be separated from interferon by high speed centrifugation. Similar reductions in the amount of infectious virus produced by CE cells were observed when such virus preparations were used to infect the cells at a multiplicity of 1,000.

These results suggest that the autointerference observed here was due to the virus particles themselves.

Table 1. INFLUENCE OF MULTIPLICITY ON THE YIELD OF WEE VIRUS IN CE CELLS

Concentration of virus added (P.F.U./cell)	Yield of virus (P.F.U./cell) Control Exp. 1	Control Exp. 2	Treated with actinomycin *D* Exp. 1	Treated with actinomycin *D* Exp. 2
1,000	720	658	650	600
100	1,160	*	950	*
10	2,240	2,356	1,080	1,124
1	4,400	4,251	2,800	2,625
0·1	5,600	5,880	6,000	5,775

* Not done.

CE cell cultures 24 h old, containing 3×10^6 cells per culture were used. Cultures were treated with actinomycin *D* by adding 2 ml. of Eagle's medium containing 5 μg/ml. of the drug and incubating for 2 h at 37° C. They were infected by adding 0·5 ml. of WEE virus diluted appropriately and incubating for 1 h at 37° C. The cultures were then washed three times with phosphate buffered saline containing bovine serum albumin and 5 ml. of Eagle's medium were added to each culture. They were then incubated at 37° C. The cultures were collected 20 h after the addition of virus by scraping the cells into the medium. Samples for infectivity titrations were taken after freezing and thawing the infected cells three times.

This ruled out conclusively the involvement of interferon. A physical particle present in vesicular stomatitis virus (VSV) populations obtained after multicyclic growth in CE cells has been held responsible for the autointerference in VSV infection[9]. We do not know whether similar particles are present in WEE virus. Density gradient analyses of crude WEE virus stocks have yielded two peaks of virus specific materials[10]. The faster sedimenting peak accounted for the bulk of the infectivity. Both peaks contain haemagglutinin. Two types of physical particles have been observed in Sindbis virus populations, one accounting for the mature infectious virus and the other possessing low infectivity associated with a high haemagglutinin[11]. Experiments are in progress to ascertain the role of these particles in the observed interference.

This investigation was supported by a grant from the National Institute of Allergy and Infectious Diseases, U.S. Public Health Service.

T. SREEVALSAN
R. Z. LOCKART

Department of Microbiology,
University of Texas,
Austin, Texas.

[1] Von Magnus, P., *Acta Pathol. Microbiol. Scand.*, **28**, 278 (1951).
[2] Cooper, P. D., and Bellett, A. J. D., *J. Gen. Microbiol.*, **21**, 485 (1959).
[3] Chambers, V. C., *Virology*, **3**, 62 (1957).
[4] Lockart, R. Z., *Virology*, **10**, 198 (1960).
[5] Lockart, R. Z., *J. Bact.*, **85**, 556 (1963).
[6] Dulbecco, R., and Vogt, M., *J. Exp. Med.*, **99**, 183 (1954).
[7] Taylor, J., *Biochem. Biophys. Res. Commun.*, **14**, 447 (1964).
[8] Lockart, R. Z., *Biochem. Biophys. Res. Commun.*, **15**, 513 (1964).
[9] Hackett, A. J., *Virology*, **24**, 51 (1964).
[10] Sreevalsan, T., and Lockart, R. Z. (unpublished observations).
[11] Mussgay, M., and Rott, R., *Virology*, **23**, 573 (1964).

Host Induced Change in the Morphology of a Powdery Mildew Fungus

BLUMER'S[1] work showed that the powdery mildews produce two types of conidiophore, those of the *Erysiphe polygoni* DC. type, producing one mature conidium in a day, and those of the *Erysiphe cichoracearum* DC. type, producing a chain of between two and eight mature conidia in a day. Childs[2] later referred to these two powdery mildews as the non-chain forming and the chain forming types respectively. He showed that the former had a single peak period of spore liberation between