self-contradictory, although the 'enteron' does include the pharynx. This difficulty emphasizes the need for reconsidering the classification and nomenclature of the small ether-resistant viruses and the adoption of a system which will embrace the non-human enteroviruses and the 'respiratory enteroviruses'.

I thank Dr. A. P. Waterson of the School of Pathology, University of Cambridge, for carrying out the electron microscopy.

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[1] Plummer, G., and Kerry, J. B. (in preparation).

Apple Virus Inactivation by Heat Therapy and Tip Propagation

The presence of many viruses latent in apple varieties and rootstocks has been shown by the use of indicator plants such as Virginia Crab, Spy 227, and *Malus platycarpa*. Three of these viruses which on *M. platycarpa* produce symptoms of line pattern (= chlorotic leaf spot), dwarfing and scaly bark have been shown to be widely distributed. No virus-free source was found of many of the apple varieties and rootstocks grown commercially in England.

Experiments were therefore started in 1958 to try to inactivate these viruses by heat therapy, a method which had proved successful in similar work with some strawberry and raspberry viruses.

A cabinet was used in which the air temperature was controlled at 37° ± 2° C. and young apple trees were grown at this temperature for 10, 15, and 20 days. Buds from the extension shoots, produced while the trees were in the cabinet, were afterwards retested on *M. platycarpa*, using the double-budding method of virus indexing. The results shown in Table 1 suggested that the viruses in the deep-seated tissues were not inactivated by any of these treatments, for although some initial improvement was seen, this was not maintained and systemic reinfection was rapid. At the end of the test year the virus content of the treated plants was the same as that of the controls.

Table 1

	Heat treatment (days)	No. of clones treated	No. of clones virus-inactivated
No tip propagation	10	12	—
	15	9	—
	20	7	—
	20	22	7
Tip propagation	23	25	10
	30	9	4

A method of isolating the growing tip from the heat-treated plant as soon as possible after treatment was therefore developed. The extreme tip (1 cm.) of the young shoot was wedge-grafted into a young apple seedling of the same diameter. The tiny graft was held in position by self-adhesive crepe bandage and enclosed in a small polythene bag. Propagation is possible in this way at any time of the year but most success was achieved with grafts made in the spring, when about 90 per cent grew.

The results after heat treatment and tip propagation are also shown in Table 1.

It can be seen that the introduction of tip propagation after heat treatment proved successful in more than one-third of all the clones treated. Success is not related to the duration of the heat treatment, since it appears possible to produce healthy clones after 20 days, yet some still carry infection after 30 days.

The important factor seems to be the growing-away from the virus rather than the complete inactivation of all the virus present. It is therefore necessary to produce very rapid growth under conditions unfavourable to virus multiplication and to isolate the young tip as soon as possible after treatment. Tips from control plants not heat-treated were still infected when re-tested.

Some of the apparently healthy clones may become re-infected from deep-seated virus at a later date. It is also possible that the virus concentration had been reduced to such a level that no symptoms appeared on the indicator plants. Since a fairly long period (1½ years) has elapsed, however, since heat treatment, neither of these possibilities seems likely; nevertheless periodic tests will be necessary to confirm freedom from infection.

As a clone can be rapidly built up from one individual by vegetative propagation, the heat treatment and tip propagation need be successful in only one individual of each variety and rootstock. The clonal descendants from such an individual should remain free from these viruses indefinitely, if contact with infected material is avoided, since there is, as yet, no evidence to indicate their spread in the orchard by other means.

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Use of Goose Cells in Hæmagglutination Tests with Louping ill Virus Antigen

Porterfield[1] recommended the use of goose red blood cells for hæmagglutination and hæmagglutination inhibition (H.A.I.) tests with arbovirus antigens. Such tests are susceptible to non-specific inhibition by lipids present in normal animal serum[2,3]. These non-specific inhibitors are widely distributed[4], but little is known of their nature or mode of action.

Porterfield and Rowe[5] investigated a number of purified phospholipid fractions of human red cells and found that the inhibiting activity against several Group *B* arbovirus antigens was associated with certain choline-containing phospholipids related to lecithin. Lecithin itself extracted from hen's eggs was a powerful inhibitor, but they were unwilling to accept the explanation that lecithin itself was the active component, in view of the fact that one fraction without activity in hæmagglutination inhibition was the richest in lecithin by chemical tests, and egg lecithin had only 0·5 per cent of the activity of other fractions.

In the course of carrying out hæmagglutination inhibition tests on sheep serum[6] with a louping ill virus (Group *B*) antigen we noted that the goose cells used in the tests became inagglutinable when egg laying commenced in April 1961, and remained so during the egg-laying period, agglutinability returning some time after egg laying had ceased. The goose cells from this bird again became inagglutinable on October 5, 1961, when egg laying again occurred. At the same time non-specific inhibitor, normally present in goose and gander serum, was no longer detectable in the goose (Table 1). These inagglutin-