

changes of nutrient fluid. Virus has been demonstrated, by mouse inoculation, in the fluids removed during the course of the experiment, including that of the 17th day. The calculated multiplication of the virus was approximately 10^3 times. This finding suggests that multiplication occurred in this tissue which, from the embryologic point of view, is more mature.

To compare the increase of virus in nervous tissue with that in tissue of the intestine and the extremities, cultures of embryonic brain were prepared. The multiplication of the virus in this medium has been comparable to that in the other types. Thus in one experiment carried out contemporaneously with the series of embryonic intestinal cultures mentioned above, the calculated multiplication of virus was of the order of 10^{12} times.

No evidence was obtained which indicated that an agent other than the Lansing strain of virus was propagated in any of the three types of tissue. Mouse infectivity tests for the presence of virus in the supernatant fluids of uninoculated control cultures were negative. Aerobic and anaerobic cultures of supernatant fluids yielded no growth of bacteria.

On microscopic examination of fragments of the three types of tissue, removed after about 30 days of cultivation, differences have been observed in cell morphology between those derived from inoculated and uninoculated cultures. Many of the fragments from uninoculated cultures contained cells which appeared to be viable at the time of fixation, as indicated by the normal staining properties of the nuclei and cytoplasm. In contrast, the nuclei of the majority of the cells in fragments from inoculated cultures showed marked loss of staining properties. Since the amount of material which has been studied is as yet relatively small, one cannot conclude that the changes observed in the inoculated cultures were caused by the virus.⁶

It would seem, from the experiments described above, that the multiplication of the Lansing strain of poliomyelitis virus in the tissues derived from arm or leg, since these do not contain intact neurons, has occurred either in peripheral nerve processes or in cells not of nervous origin.

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Insect Vectors of Phony Peach Disease¹

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Phony peach is a virus disease of economic importance in the southeastern United States (1). While the disease attacks other species of the genus *Prunus*, its most serious effects result from heavy infections in commercial peach orchards, particularly in the South Atlantic and Gulf Coast States from South Carolina to Louisiana. The disease has caused the loss of more than 1,500,000 peach trees during the period from 1929 to 1947, inclusive.

Since 1936 the Bureau of Entomology and Plant Quarantine, in cooperation with the Bureau of Plant Industry, Soils, and Agricultural Engineering, has been conducting research in an effort to find the insect vector, or vectors, of phony peach. Following an extensive survey of insects associated with peach orchards in areas where the annual disease incidence was high, in other areas where few new cases occurred each year, and finally, in areas where there appeared to be no local spread, a test program utilizing the most likely insect suspects was undertaken.

Results of the survey, together with data derived from experimental transmission of the disease, suggested the probability that phony peach was being spread by one or more stem-feeding Cicadellidae (leaf hoppers) belonging to the subfamily Tettigellinae. Although 53 species of insects, including members of several different families of Homoptera and a number of species of Cydnidae (Hemiptera), have been used in transmission tests, most attention for the last four years was given to four species of leaf hoppers.

From 1939 through 1944 the work was conducted at Chattanooga with field-grown peach trees that were unprotected from possible natural infection. During the course of these tests three of the experimental trees developed symptoms of the disease. One had been subjected to inoculation by *Cuerna costalis* (F.), one by *Graphocephala versuta* (Say), and one by *Homalodisca triquetra* (F.),² all members of our selected group of suspects. One case of phony peach occurred in one of the many check trees that were maintained.

In 1945 the work was transferred to Fort Valley, Georgia, where it was necessary to grow the experimental trees in a large screened cage because of the certainty of considerable natural spread in unprotected trees. Insects

¹ The diagnoses on which this report is based were made by Lee M. Hutchins and L. C. Cochran, of the Bureau of Plant Industry, Soils, and Agricultural Engineering, and by L. D. Christenson and William F. Turner, Bureau of Entomology and Plant Quarantine, of the United States Department of Agriculture.

² The leaf hoppers actually used in each of these and all other tests were identified by P. W. Oman and J. S. Caldwell, Division of Insect Identification, Bureau of Entomology and Plant Quarantine.

collected on peach trees, weeds, and wild and cultivated shrubs were confined in cheesecloth bags on branches of phony trees. After 4 or 5 days the bags with their contained insects were transferred to test trees growing in the large cage. The bags were left on these test trees for 30 or more days, unless all the insects had died earlier. At the end of the test period the bags were carefully removed in such a manner that none of the insects escaped within the large screened cage. The test trees were Elberta June-buds in their second year from seed. In order to provide fully adequate controls, alternate trees were left as checks. Transmission tests were made from April to October. During the following winter the test trees and checks in the cage were dug and replanted in isolated plots in areas where there were no commercial orchards and where phony disease was rare or unknown.

Phony disease has a long incubation period in peach trees, apparently from about 18 months (or two growing

TABLE 1

Species	Total No.	Phony peach transmissions	
		Definite	Probable
<i>H. triquetra</i>	203	8	8
<i>C. costalis</i>	64	1	2
<i>O. undata</i>	26	3	0
<i>G. versuta</i>	20	2	0

seasons) to as much as 3 years. Although a few of the trees used in the 1945 tests showed suspicious symptoms in September 1947, it was not until the spring of 1948 that reliable diagnoses could be made. At that time 7 trees were positively phony, and 8 more were almost certainly so. Of the 7 positive cases, two had been inoculated by *G. versuta*, three by *H. triquetra*, and two by *Oncometopia undata* (F.). All of the 8 trees that were almost certainly diseased had been inoculated with *H. triquetra*. None of the check trees showed any symptoms of the disease.

In contrast with the 1945 series, a number of the trees used in the 1946 experiments showed positive symptoms in July 1948. At that time seven definite cases of phony peach were noted. One of these trees had been inoculated by *C. costalis*, one by *O. undata*, and five by *H. triquetra*. At least two other trees inoculated by *C. costalis* are almost certainly diseased. All check trees appeared to be normal.

The totals for definite and probable insect transmissions of phony peach obtained in the test series of 1945 and 1946 are presented in Table 1. One check tree was provided for each test tree, a total of 313 in all. All check trees are still normal.

The definitely positive results represent from 2 to 28% successful transmission by the different species. Although all these insects are general feeders, three species are known to be associated with peach trees at certain seasons. For the fourth one, *C. costalis*, a natural association is doubtful, but the insect survives for periods of

a month or more when confined on peach. This species has thus far been the least successful of the four in transmitting the disease. All four species belong to the subfamily Tettigellinae, and it appears quite possible that other members of this group may be able to transmit phony peach even though they may be of no real importance as vectors.

In 455 experiments involving the use of the remaining 49 species of insects that were tested, all results were definitely negative.

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Inhibition of the Adenosinetriphosphatase Activity of Preparations of Myosin¹

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During the past year we have been investigating the inhibition of the adenosinetriphosphatase activity of the myosin complex by extracts of the posterior pituitary

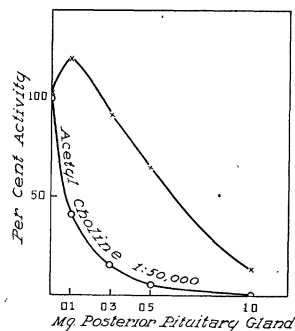


FIG. 1. ATPase activity in the presence of extract of posterior pituitary gland and acetylcholine. The percentages of the control values are plotted. Activity was determined by analysis for inorganic phosphate after 5-min incubation at 30° C in 1 M KCl buffered at pH 9.0 with 0.1 M NaHCO₃-Na₂CO₃. One mg of CaCl₂, 10 mg of ATP (sodium salt), and a solution of twice-precipitated myosin (0.2 mg of protein nitrogen) containing 0.001 M NaCN were present in a total volume of 5 ml. Acetylcholine and the soluble material of the desiccated posterior pituitary gland were added as indicated. The incubation was terminated by the addition of trichloroacetic acid to make a final concentration of 5%.

gland. Our interest in this casual observation was due to the finding that the inhibition was greatly reinforced by the addition of acetylcholine. Although a considerable mass of data has been accumulated, truly definitive experiments have not been devised, and it is considered

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