MSU Extension Publication Archive

Archive copy of publication, do not use for current recommendations. Up-to-date information about many topics can be obtained from your local Extension office.

Virus Diseases of Plants Michigan State University Agricultural Experiment Station Circular Bulletin L.O. Kunkel, Departmext of Animal And Plant Pathology, The Rockefeller Institute For Medical Research, Princeton, New Jersey Issued November 1947 19 pages

The PDF file was provided courtesy of the Michigan State University Library

Scroll down to view the publication.



Circular Bulletin 208

November 1947

Virus Diseases of Plants

What They Are and How They Differ from Fungus Diseases

By L. O. Kunkel

Michigan State College

Agricultural Experiment Station

East Lansing

FOREWORD

The material presented in this bulletin is from an address by Dr. L. O. Kunkel of the Rockefeller Institute for Medical Research before the Annual Fruit Growers' Conference, held at Michigan State College, January 13, 1947.

The information proved so helpful to fruit growers present, in explaining the complexities of viruses and in detailing some of the extensive and valuable research work that has been done and is being done in this field, that they requested its publication and distribution. It presents the more important established facts regarding the nature of virus diseases of plants, with special reference to horticultural crops. It should be of great value not only to fruit growers of Michigan but also to florists, nurserymen, vegetable growers, and general farmers.

Virus Diseases of Plants ... What they are and how they differ from fungus diseases

By L. O. KUNKEL

DEPARTMENT OF ANIMAL AND PLANT PATHOLOGY, THE ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH, PRINCETON, NEW JERSEY

Received for publication Feb. 3, 1947

It is evident from the title assigned me that you wish a discussion of what virus diseases are, and I assume you also wish a discussion of what viruses are. You perhaps have gained the impression that scientists are now agreed as to the nature of viruses and that it should be possible to discuss them just as we discuss the parasitic fungi. Fungi, as you know, are nongreen plants that live as parasites and saprophytes. They are organisms that can be seen by the unaided eye or under ordinary light microscopes. No one questions these statements.

The viruses, on the other hand, are extremely minute bodies that vary considerably both in size and in shape and cannot be seen under ordinary light microscopes. Tobacco mosaic virus, as you know, is a rod-shaped body (15). Tomato bushy stunt virus and the virus of southern bean mosaic are spherical or almost spherical bodies (17). Under the electron microscope these viruses look remarkably like bacteria, only they are smaller than bacteria. They do not swim around like some bacteria do, and are not known to multiply by dividing as do the bacteria.

One good way of acquainting you with some present ideas regarding viruses is to quote from recent literature. "With the exception of virus activity, the properties of some of the smaller viruses are quite similar to the properties of ordinary protein molecules, whereas at the other extreme, with respect to size, the properties of the viruses are more nearly like those of accepted living organisms," says Stanley (19). "The generalisation that 'plant viruses are simple nucleoproteins' appears to lack foundation and the more speculative deductions from it should be accepted with caution," says Pirie (16). These authors have contributed much to our knowledge of the chemical and physical properties of viruses, but as the quotations indicate, they seem uncertain as to what viruses really are. However, statements that are far more positive than those just quoted are to be found in recent literature, and it may be well to consider some of these before discussing special types of virus behavior.

The clonal variety of potato known as King Edward carries particles which, if transferred to other clones by grafting, produce disease. "What is a stable and presumably useful cell protein with one plant genotype acts as a destructive agent with another. Just, in fact, as plasmagenes do," argues Darlington (3). Then he says "The same principle applies to the origin of the viruses causing the Rous sarcoma and presumably mammary cancer in mice. Since they are transmitted, the one only by injection, and the other only by injection or through the milk, they can scarcely have arisen otherwise than from the cell proteins of the fowl, or the mouse, in which we find them." Also, he says, "Now grafting is not a natural process but a human invention and a very recent and restricted one. Any virus which can be transmitted only by grafting must therefore have arisen from grafting, that is to say, from the invasion of one plant by the proteins of another." Some further statements by Darlington are revealing. "There is therefore nothing surprising in the fact that reproductive particles can suddenly appear in the cytoplasm by the action either of the mutafacient nucleus or of external carcinogens, nor again that such particles may either be transmissible or only transplantable." "The plasmagene is a protein which can be made outside the nucleus and comes to be inherited through the egg. The virus is a similar protein which is capable of being acquired later. It is a protein which prospers through being in the wrong organism and gets there by infection. Both classes are, of course, immensely heterogeneous. In addition, both are continually arising de novo, rapidly evolving, as their conditions change and partly by direct action of those conditions." Now you know what viruses are and how they arise, if you believe Darlington's arguments are sound.

It would seem unprofitable to quote such statements if it were not for the fact that some of our foremost scientists seem to give them serious consideration. In discussing the possibility that viruses may have arisen by retrogressive evolution from some parasitic organisms, Beadle (2) cites Darlington and says "An alternative view that must also be given serious consideration is that viruses arise directly by mutation from nucleoproteins of the host without going

through the entire retrogressive evolutionary sequence outlined above." In discussing viruses in relation to the cancer problem, Rous (18) says "They increase in quantity only when associated with cells, none having been cultivated in artificial media despite energetic efforts in this direction. Some reside permanently in certain animals or plants, e.g., the virus of King Edward potato plants, which causes them no perceptible trouble, though capable of killing plants of other varieties. How they get from one living organism to another is still uncertain in many instances, but not a few do so by means of intermediate hosts, the mosquito, for example, in the case of yellow fever, the leaf hopper in that of aster vellows, the earthworm in swine influenza. Some viruses are so large and chemically so complex as to warrant the view that they are the products of retrograde evolution in the sense in which Darwin used the term, mere residua, like the tapeworm, of what they once were, their functioning parts lopped off one by one. so to speak, when their host cells had taken over their duties. Others are so extremely minute that it is difficult to see how the needed equipment for life can be comprised within them, and a few have been purified by chemical procedures, with the result that they crystallized out. On looking at the virus crystals and thinking of what they can do one wonders again what life is. Indeed the worker with viruses has continually a sense of uneasiness as to what next, for they are like something out of folklore, like the 'wee people' who play mischievous tricks on the Irish peasantry, prodigies of the imagination."

Rous points out that only a few cancerous growths have been shown to be caused by viruses, and considers this a "meagre yield for a generation of effort." He says "The single chicken virus thus far studied for durability, that responsible for an exceedingly malignant sarcoma, has proved remarkably fragile; it becomes inactive within a few hours after separation from the neoplastic tissue unless special steps are taken to preserve it. How then can it get from fowl to fowl? Not through the egg, test has excluded that possibility, and normal fowls do not develop the sarcoma when kept for years in the same cage with birds carrying it. If the virus passes at all from one individual to another, it must be in some devious, highly-conditioned way, and more would be required of any virus responsible for the rare human tumors of which new examples appear now and then at wide distances of time and space."

It is with these quotations in mind that I wish to discuss a few virus diseases of plants from the point of view of a plant virologist.

You probably are acquainted with the disease known as Abutilon mosaic. A man by the name of Thompson took an Abutilon plant having this disease from the West Indies to England. There it was propagated by means of cuttings and was introduced into the trade in 1868 (1). The plant became popular as an ornamental, being used in border plantings. It was soon discovered that if a twig from one of these chlorotic Abutilons was grafted to a healthy Abutilon plant the latter soon developed chlorosis. The disease passed from the chlorotic plant into the healthy plant. Then it was recognized that the original plant which had come to be known under the species name of Abutilon Thompsoni was in reality a sick plant of the wellknown species Abutilon striatum Dicks. These diseased Abutilon plants have been grown beside healthy Abutilon plants in England and other European countries during the past 75 years. There is no record that the disease ever passed spontaneously to a healthy plant in Europe. Diseased and healthy Abutilon plants have been grown side by side in this country for many years. We grew 400 diseased and healthy Abutilon striatum plants in rows in which the healthy plants alternated with diseased plants in a garden at Yonkers, New York, some years ago. The disease did not spread to a single healthy plant. We were justified in concluding, and in fact did conclude, that if this virus ever passes in nature it must be by some devious, highly-conditioned way, but we never doubted that it passed.

Some years ago while visiting in the Island of Haiti, I observed that a virus disease closely resembling Abutilon mosaic was spreading among Sida plants of the species Sida rhombifolia L. This led me to suspect that here in the general region from which the original Abutilon Thompsoni plant was obtained there was an insect capable of transmitting the virus of this disease. Later it was learned that Sida mosaic also was present in Florida. When affected Sida twigs were grafted to healthy plants of Abutilon striatum, a disease was obtained in the latter that closely resembled Abutilon mosaic (7)as is shown in Fig. 1. Insects found feeding on diseased Sida plants in Florida were secured and tested for ability to transmit Sida mosaic, but no vector was discovered. During the past year Silberschmidt (14) has reported that a Sida mosaic, prevalent in Brazil and closely resembling Abutilon mosaic, is transmitted by a white fly. The work will have to be confirmed, but it seems probable that the 75-year-old mystery of how Abutilon mosaic passes in nature has been solved.

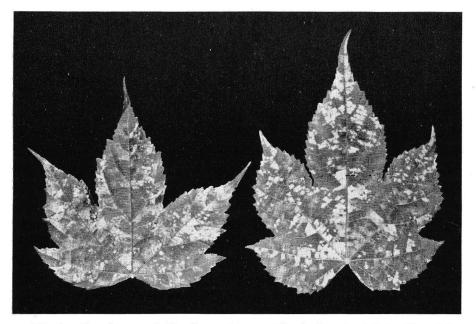


Fig. 1. Two leaves of Abutilon striatum. The leaf at the left is affected by Sida mosaic from Florida; that at the right by Abutilon mosaic. (Photograph by J. A. Carlile.)

The disease which puzzled Baur (1) because it seemed to be transmissible only by grafting apparently gets along well and spreads rapidly in the presence of its insect vector. In the absence of such a vector it does not pass except by grafting. As the years go by, more and more vectors of plant virus diseases are found, and the pattern of the behavior of these viruses becomes more and more clear.

You will recall that we do not know how some of the most important virus diseases of peach are spread. Peach rosette has been known and studied for more than 50 years. In some seasons it appears in considerable abundance in certain localities, but in others it appears in only a few trees of the many thousands that grow in the area of its occurrence. Until recently it had been transmitted only by grafting, that recent and restricted invention of man. We now know that is can be transmitted readily by the dodder species, *Cuscuta campestris* (11). Most species of plants, as you well know, can be grafted only on closely related plants. It is easy to graft peach on plum and plum on peach, but you cannot successfully graft an aster scion on a peach stock. There is no mechanical difficulty in making

such a graft. Aster scions often live for more than a month when inserted in peach trees, but they all die eventually. However, species of the genus Cuscuta are not so restricted. These plants are able to graft themselves onto hundreds of different species. This grafting is their speciality, the method by which they live. Cuscuta campestris can live and thrive on such widely different plants as tomato, alfalfa, cranberry, carrot and aster. Figure 2 shows it growing on cranberry and tomato. Now if this dodder is established on a healthy young carrot plant and allowed to grow over onto and parasitize a rosetted peach tree, the peach rosette virus will be transmitted to the carrot plant. Dodder transmitted rosette from the peach to the carrot plant shown in Fig. 3. Once the dodder has picked up the rosette virus it readily transmits this to tomato, Vinca rosea and other herbaceous plants. This was a big surprise, because we had supposed peach rosette was specific for the peach and other species of Prunus. No one had ever heard of it in herbaceous plants before. However, this knowledge that peach rosette virus will go to herbaceous plants may be of value in the search for a vector. Peach rosette probably is not

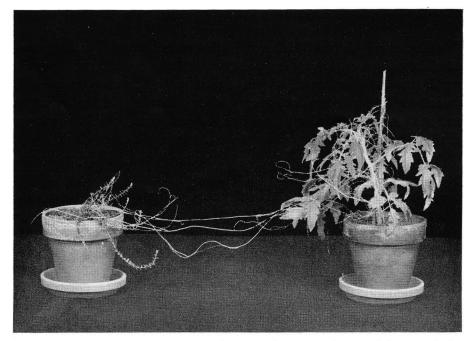


Fig. 2. Cuscuta campestris on cranberry and tomato plants. (Photograph by J. A. Carlile.)

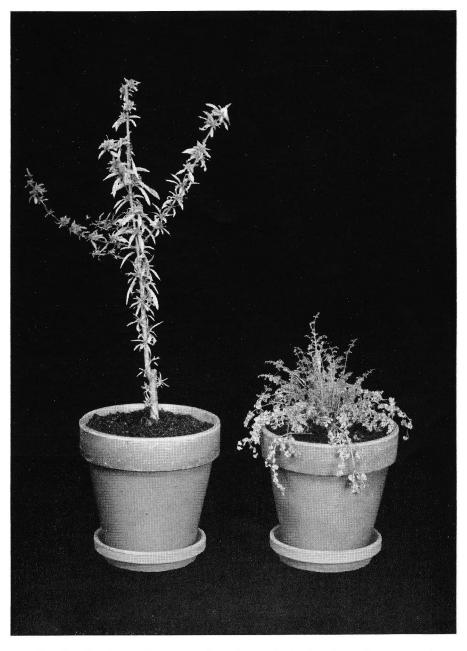


Fig. 3. Peach rosette on peach and carrot plants. The disease was transmitted to the peach by budding and to the carrot by dodder. (Photograph by J. A. Carlile.)

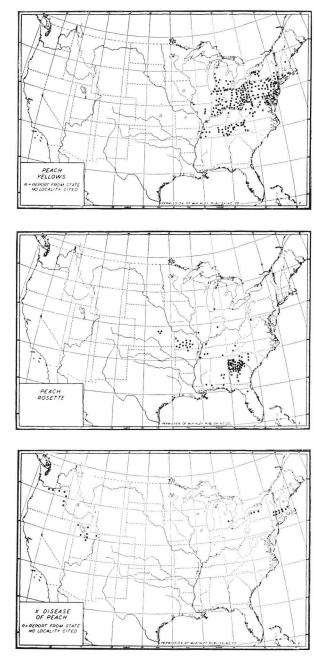
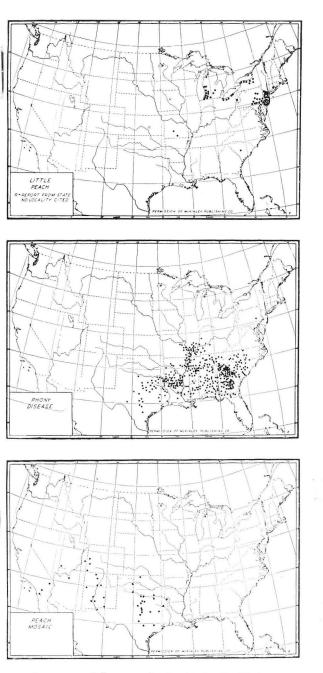


Fig. 4. Distribution of six peach diseases in th



United States. (Photographs by J. A. Carlile.)

primarily a peach disease. It may be a disease of some wild herbaceous plant, perhaps a weed in which peach growers have no interest whatever. In the future we will be on the lookout for anything that resembles peach rosette in any plant in the area in which peach rosette occurs. Also, we will be on the lookout for insects that may be found feeding on such plants. It is among these that we may expect to find the vector of peach rosette, for if peach rosette were spread by a peach insect surely that fact would have been established long ago.

Let us now return to a consideration of dodder transmission of plant viruses. The parasite, in grafting itself onto different plants, is doing essentially what man does when he grafts one plant onto another. Thus we see that grafting is not as Darlington seems to think, an invention of man. Both man and the dodder parasite are able to transmit a good many different viruses including peach rosette as recounted above. While the parasite readily transmits peach rosette to plants like the carrot and the tomato, it is unable to transmit rosette to healthy peach trees. This is a surprising fact for which we have no explanation at present.

Up to this time peach rosette virus has not been transmitted to peach except by grafting. Should we therefore conclude that it must have arisen by grafting, as Darlington would be inclined to do? Grafting probably has had nothing to do with its origin and little to do with its spread. The fact that in some years rosette develops in only a few trees of the many thousands in its area of prevalence does not justify the conclusion that it must be arising de novo, or that it does not pass spontaneously in nature. In my opinion proof that any virus causes a disease prevalent in nature, whether the disease be a chlorosis, a necrosis, a mild stunting only discernible with difficulty, or an overgrowth of one kind or another, or whether in the hands of the experimenter it be transmitted by the slightest contact or only by grafting, should be taken as prima facie evidence that the virus concerned does pass in nature. We need to learn much more about peach rosette virus before we can fit it into the pattern of behavior presented by some other yellows viruses, but I have no doubt that when more is known it will fit into that pattern.

Although there may be no good reason for believing that viruses have arisen through the invasion of certain plants by the proteins of other plants, or through mutations in the genes or plasmagenes of normal plants, are there any well-known facts that make such modes of origin seem unlikely? There are a number of different virus diseases of peach prevalent in the United States. Let us look at some maps that show the distribution of a half a dozen of them (Fig. 4). You will note that peach yellows is eastern and northern in its distribution. It does not occur in the peach-growing regions of Georgia or in California where large numbers of peach trees are grown. Little peach has much the same distribution. The two diseases are believed to be caused by closely related viruses. Peach rosette and phony disease of peach are eastern and southern in their distribution. Insofar as I am aware, except for one report of rosette, neither disease occurs in Michigan and neither occurs in California. X-disease of peach is northern in distribution and spans the country from east to west. It does not occur in New Jersey or in Georgia. Peach mosaic, on the other hand, is southern and western in distribution. Now if these diseases had arisen through grafting or by mutation of normal peach proteins why should they have such restricted distribution? Grafting is practiced throughout the country. If they arise through mutations in normal proteins we would have to assume that such mutations are rare. Otherwise we should expect to find all of them occurring throughout the country. You might answer that they have arisen all over the country but that they flourish and spread only where conditions are favorable. Such an explanation of their present distribution may seem reasonable, but we know that some viruses have remained absent for many years from areas in which they later flourished. For instance, we know that sugar cane has been grown in the Hawaiian Islands since 500 A.D. In all of the time that elapsed from that date down to recent times no sugar cane mosaic appeared in those Islands. If the virus of this disease could arise de novo by mutations from normal proteins in sugar cane cells, why did it not arise in the 1400 years during which sugar cane was grown there before the disease appeared? A vector was prevalent in those Islands and conditions for spread were favorable as was proved when sugar cane mosaic virus inadvertently came in on some sugar cane cuttings imported from another country. There is a virus disease of corn in the Hawaiian Islands known as vellow stripe. It is transmitted by the leaf hopper Peregrinus maidis (5). Peregrinus maidis is prevalent in the southern part of this country. In the east, at least, it ranges up to about as far as the Mason and Dixon line. As everyone knows, many acres of corn are grown south of that line. If the virus of vellow stripe of corn can arise from normal proteins why does it not appear in that region?

If it should arise there we have good reason to believe that it would be spread by the corn leaf hopper. Aster vellows which is prevalent throughout the United States does not occur in England, although aster plants are grown there. In fact no plant virus disease of the yellows type occurs in England. The stunt disease of rice is and has for a long time been prevalent in Japan (4). It does not occur in the rice fields of the United States, but it should if it could arise de novo in rice cells. We must concede that, in spite of the fact that a few virus diseases of plants are world wide or almost world wide in their distribution, this does not hold for the generality of plant virus diseases. The present distribution of most plant virus diseases does not coincide with the distribution of the plants they affect. Hence, any hypothesis based on the assumption that viruses are presently arising de novo by mutation or otherwise seems ill-founded; for even though a virus disease might not be able to spread in any given region, it would be seen there if it appeared at all frequently in any important crop plant. Although we do not know how plant viruses have arisen, we do know that most of them are restricted in their distribution. It seems fair to assume that they have arisen in the countries where they now occur, if indeed those countries existed in the ancient times when, in all probability, the viruses first appeared. The geographic distributions of plant viruses are characteristically local, like the geographic distributions of most plant species that have not yet been widely distributed by man. The distributions of plant virus diseases do not coincide with the distributions of plant proteins which are, of course, found whereever the species of the plants with which they are associated occur. Therefore, I am inclined to believe that plant viruses have arisen through a process of evolution similar to that by which plants and animals have arisen, rather than by mutations in normal plant proteins. Insofar as I am aware there is no evidence whatever to support the view that plant viruses are of recent origin.

You will recall Rous' statement that the single chicken sarcoma virus which has been studied for durability has proved remarkably fragile. He wonders how such a fragile entity can get from fowl to fowl, and even seems to doubt that the sarcoma virus passes in nature. It is in this connection that I wish to describe some experiments which indicate that certain plant viruses which are extremely fragile pass with great readiness in nature.

You are, no doubt, well acquainted with aster yellows. It is a disease that occurs on asters, carrots, buckwheat, lettuce and many

other plants (6). After working with this disease for about 10 years and finding that, although it could not be transmitted mechanically by means of juices from diseased plants, it was readily transmitted by the aster leaf hopper Macrosteles divisus, an attempt was made to study its epidemiology. At Princeton, New Jersey, it was found that the disease first appeared in aster plantings in from 2 to 3 weeks after the plants were set out. It spread very rapidly for 4 or 5 weeks, and then very slowly. The period of slow rate of spread usually began in June and continued until sometime in September. In September there was a second period in which the disease spread rapidly (9). This epidemiological behavior did not conform to what was expected. The leaf hopper vector was prevalent in small numbers during the first period of rapid spread; it became numerous during the summer when rate of spread was slow and was fairly numerous in September when rate of spread increased. We were surprised to find such slow spread in summer and decided to determine the percentage of leaf hoppers that were infective at different seasons of the year. In a midsummer experiment 25 aster leaf hoppers were caught in an aster plot where the incidence of yellows was high. Each individual insect was placed in a different cage on a healthy young aster plant. The insects were allowed to feed on the plants for several days. We expected a high percentage of infection for it was known that one infective insect feeding on a young aster plant for one day would transmit yellows with a high degree of certainty. We were astonished when all of the 25 aster plants remained healthy. How could such a result be explained? Then it was remembered that most of our successful greenhouse experiments with aster yellows had been carried out during the winter and early spring months. A few that had been undertaken in summer had, for some unknown reason, not turned out well. They had been discarded and had not been mentioned in any publication. Finally a study of the effect of temperature on transmission of aster vellows was undertaken. It was soon found that, although infective leaf hoppers held at about 25° C. transmitted aster vellows with great regularity, infective leaf hoppers placed at a temperature of 32° C. stopped transmitting in about 8 hours. There is not sufficient time to describe the different experiments that were made before we came to understand what was happening. It is enough to say that aster vellows virus is very easily inactivated by moderately high temperatures. It is a very fragile virus, much more fragile probably than Rous' chicken sarcoma virus. It is so fragile that potted vellows periwinkle and Nicotiana rustica plants can be cured by holding them at a temperature of from 38° to 42° C. for 2 or 3 weeks (10). Those temperatures cause no serious injury to the plants but they inactivate the virus. Infective aster leaf hoppers are freed of the virus of aster yellows when held at a temperature of about 32° C. for 12 days. That the virus is more readily inactivated in the insect than in the plant is understandable when it is remembered that the insect is very small and quickly reaches any temperature to which it is exposed, whereas plants are large and come to the temperature of their environments slowly. The fact that aster yellows virus has never been transmitted to plants mechanically by means of juices from diseased plants probably is due to its extreme fragility.

In other studies it was shown that peach yellows, little peach, peach rosette (8), potato witches'-broom (12), cranberry false blossom (13) and two other plant virus diseases that are currently under investigation can be cured by heat treatments. These diseases, none of which can be transmitted mechanically by means of juice, apparently are caused by fragile viruses. How can such fragile entities get from plant to plant? How can they maintain themselves in nature? Those that have been carefully studied are known to be transmitted by specific insect vectors. Some, such as the viruses of aster yellows, peach yellows, little peach and cranberry false blossom, as you know, get along very well in nature.

Peach yellows virus seems to be even more fragile than that of aster yellows. Potted yellows peach trees were cured by a temperature of about 35° C. in about 3 weeks. Dormant trees were cured by immersing them in water at 50° C. for 10 minutes. The trees were not seriously injured by these treatments. Yellows virus in buds was inactivated by immersing bud sticks in water held at a number of different temperatures. At 34.35° C. the virus was inactivated in from 4 to 5 days; at 38° C. in 11 hours; at 42° C. in 40 minutes; at 46° C. in 15 minutes; at 48° C. in 14 minutes; at 50° C. in 3 to 4 minutes; at 54° C. in $1\frac{1}{2}$ minutes; and at 56° C. in 15 seconds.

Figure 5 shows four trees budded with cured peach buds. We used bud sticks that were thoroughly invaded by the yellows virus; every living cell, we believe, contained the virus. After treating the sticks as described above we cut out the buds, inserted them in healthy peach seedlings, and wrapped them. After a suitable period during which the buds united with the seedlings in which they were implanted, the wrappers were cut and the seedlings pruned back. The

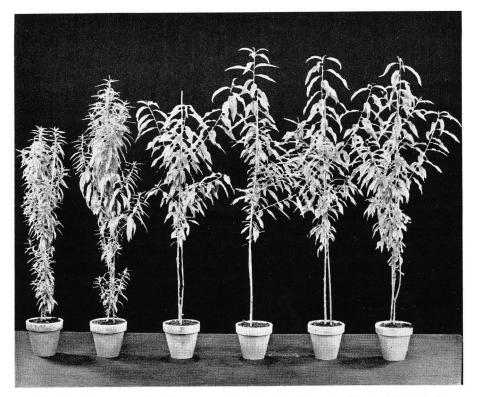


Fig. 5. Heat cure of yellows peach buds. Seedlings budded with buds from treated and untreated yellows bud sticks. Reading from left to right: tree with bud from untreated bud stick; trees with buds from bud sticks treated at 50° C. for 2, 4, 6, 8 and 10 minutes, respectively. The untreated bud and the bud treated for 2 minutes transmitted yellows, while the buds treated for 4, 6, 8 and 10 minutes did not. (Photograph by J. A. Carlile.)

implanted buds grew, produced fine healthy foliage and did not transmit virus to the trees. Buds from sticks that were not heat-treated also were implanted in healthy peach seedlings. When these buds grew they produced badly diseased foliage and transmitted yellows virus to the trees.

What a fragile entity this virus of peach yellows must be. It is destroyed by summer temperatures that frequently prevail for considerable periods in some parts of the country. The same can be said of the virus of little peach. These disease-producing agents lead a tenuous existence. Nevertheless they spread rapidly under favorable conditions and can cause great damage. In spite of their fragility they maintain themselves in nature. Peach yellows virus is one of those that was recognized earliest. Since we know that it is spread by a leaf hopper there is no occasion to postulate any theory involving the idea that it has arisen by grafting, or that it is continually arising de novo by mutations in normal peach proteins. In vivo, the viruses of peach yellows and little peach are much more fragile than normal peach proteins for they are completely destroyed by heat treatments that have no discernible effect on the latter. Heat-cured trees of ordinary peach varieties do not differ in the slightest degree from healthy trees that have never been treated. The same may be said of potato plants from tubers that were freed of potato witches'-broom virus by heat treatments, of cranberry plants cured of false blossom, of leaf hoppers freed of aster yellows virus, and of periwinkle plants cured of aster yellows. The thermal reactions of these viruses do not suggest that they have been derived from normal plant proteins, from genes, or from plasmagenes. The heat treatment experiments confirm the view that plant viruses are autonomous and unrelated genetically to their host plants.

It is not my intention to attempt an explanation of what viruses are, but in conclusion I will try to indicate to you what I think the plant viruses are. They are the most efficient parasites that we know anything about. While fungi clumsily bore from without in their attempts to anchor ill-fitting haustoria, the viruses get themselves injected into cells by some of the finest and most efficient hypodermic syringes known to man, the proboscides of insects. The fungi usually produce lesions that can be seen by quarantine officials armed with hand lenses. The viruses pass guarantine lines very simply in masked carrier hosts. If they get into trouble because some plant they try to invade is immune, they mutate to produce a virus strain that can attack this plant. To obtain distribution most fungi have to depend on having their spores blown around. The plant viruses have wings to take them where they wish to go, wings that are guided in their flights by the appetites of insects. In some instances at least, viruses multiply in their insect vectors while being carried from plant to plant. Heteroecism also is known in the fungi but it is confined to the rusts and a few other highly developed groups. When you add to all of these advantages the property of being invisible except under an electron microscope you have what are truly superb parasites. Can anyone believe that such well adjusted and well equipped parasites are of recent origin or belong in that level of organization characteristic of lifeless molecules?

BIBLIOGRAPHY

- Baur, E. Zur Aetiologie der infektiösen Panachierung. Ber. d. Deuts. Bot. Gesells. 22: 453-460. 1904.
- 2. Beadle, G. W. The gene. Proc. Amer. Philos. Soc. 90: 422-431. 1946.
- Darlington, C. D. Heredity, development and infection. Nature 154: 164-169. 1944.
- Katsura, S. The stunt disease of Japanese rice, the first plant virosis shown to be transmitted by an insect vector. Phytopath. 26: 887-895. 1936.
- 5. Kunkel, L. O. Insect transmission of yellow stripe disease. Hawaiian Planters' Record 26: 58-64. 1922.
- Kunkel, L. O. Studies on aster yellows. Amer. Jour. Bot. 13: 646-705. 1926.
- 7. Kunkel, L. O. Transmission of *Sida* mosaic by grafting. Phytopath. 20: 129-130. 1930.
- Kunkel, L. O. Heat treatments for the cure of yellows and other virus diseases of peach. Phytopath. 26: 809-830. 1936.
- Kunkel, L. O. Effect of heat on ability of *Cicadula sexnotata* (Fall.) to transmit aster yellows. Amer. Jour. Bot. 24: 316-327. 1937.
- Kunkel, L. O. Heat cure of aster yellows in periwinkles. Amer. Jour. Bot. 28: 761-769. 1941.
- Kunkel, L. O. New hosts as a key to progress in plant virus disease research. In: Virus Diseases, (Messenger Lectures), Cornell Univ. Press, Ithaca, N. Y. pp. 63-82. 1943.
- Kunkel, L. O. Potato witches'-broom transmission by dodder and cure by heat. Proc. Amer. Philos. Soc. 86: 470-475. 1943.
- Kunkel, L. O. Studies on cranberry false blossom. Phytopath. 35: 805-821. 1945.
- Orlando, A., and Silberschmidt, K. O vetor da "Clorose infecciosa" das Malváceas. O Biológico 11: 139-140. 1945.
- Oster, G., and Stanley, W. M. An electron microscope study of the contents of hair cells from leaves diseased with tobacco mosaic virus. Brit. Jour. Exp. Path. 27: 261-265. 1946.
- 16. Pirie, N. W. The viruses. Ann. Rev. Biochem. 15: 573-592. 1946.
- Price, W. C., Williams, R. C., and Wyckoff, R. W. G. Electron micrographs of crystalline plant viruses. Arch. Biochem. 9: 175-185. 1946.
- Rous, P. Concerning the cancer problem. Amer. Scientist 34: 329-358. 1946.
- Stanley, W. M. Progress in the conquest of virus diseases. Science 101: 185-188. 1945.