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Phloem necrosis of elm and other plant virus diseases

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PHLOEM NECROSIS OF ELM AND OTHER PLANT VIRUS DISEASES

by

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I. Introduction

The study of viruses and virus diseases has developed and broadened so rapidly in recent years that it is approaching such studies as bacteriology and mycology as a separate branch of biology. In spite of this development, there is still no generally accepted definition of a virus.

Originally the term virus was applied to all kinds of disease producing agents, about which little was known. After Pasteur's demonstration that bacteria were the causes of some diseases, the term virus became restricted to infective entities, as a synonym for microbe or germ. About 1870 bacteria were recognized as the causal agents of some plant diseases.

There were, however, some plant diseases for which no causal agent could be found, though it was known that fungi, insects, bacteria, mal-nutrition, and the environment could cause diseases, disease symptoms, or death. Many of these diseases were known to early workers by symptom names, such as mosaic, yellows, chlorosis, and mottling.

With Iwanowsky's demonstration that sap from a diseased plant, though passed through a bacteriological filter, could infect a healthy plant, the search for an invisible causal agent of these diseases began.

Even now, after more than 50 years of research, there
are at least two schools of thought on the nature of a virus. One school believes that the virus is a living ultramicroscopic particle descending in size from that of the rickettsia to slightly larger than some of the protein molecules. The other school believes that the virus is a protein molecule, related to, or actually a nucleoprotein, which has the ability to reproduce itself in the living cell, or causes the living cell to produce similar proteins.

This thesis will attempt to show the progress that has been made in the study of plant viruses and virus diseases, with emphasis on the work done in increasing the knowledge of a specific plant virus disease, phloem necrosis of the elm.
II. History

Virus diseases of plants, though not recognized as such, were known long before the discovery of bacteria. One description, possibly the first one of a virus disease, was that published in 1576 by Charles l'Ecluse, or Carolus Clusius, of a variegation in the color of tulips, now known as "breaking", and due to an aphid-transmitted virus of the mosaic type. The Theatrum Florae, published in 1662, has illustrations of broken tulips identified as the work of the painter, Daniel Rabel. In 1670 an article, published in Traite des Tulips, contained the first suggestion that this variegation might be caused by a disease. In 1715 an account of an infectious chlorosis of Jasminum was published in the Art of Gardening.

The so-called curl disease of potato became prominent about 50 years later, a great controversy arising as to its cause. One common explanation was that "degeneration", a kind of senile decay, was caused by long continued vegetative propagation. In reply to this explanation, it was pointed out that, in certain areas high up in the mountains and in wind-blown areas near the sea, the same variety of potatoes could be grown for many years, saving the seed each year from the current year's crop, without any signs of degeneration. The discovery that potato leaf-roll was an
infectious virus disease finally settled the controversy and showed that the degeneration was due solely to a gradual infiltration of the virus into the crops (Cook, 1946).

In 1886 Mayer described a disease of the tobacco plant which he named Mosaikkrankheit. This term, or its English translation, is now widely used to describe the mottling type of virus disease. He demonstrated that this mosaic disease of tobacco could be transmitted to a healthy plant by inoculation with the sap of an infected plant. Two years later Erwin F. Smith proved that peach yellows was transmissible by budding (Smith, 1946).

The first scientific proof of the existence of a virus came in 1892. Iwanowsky, working with the mosaic disease of tobacco described by Mayer, proved that sap from an infected plant could induce the disease in healthy tobacco plants after it had been passed through a bacteria proof filter and was bacteriologically sterile. Iwanowsky did not seem to grasp the true significance of his work. Beijerinck, having repeated Iwanowsky's work seven years later, propounded his theory of a "contagium vivum fluidum" (Cook, 1946).

Many years passed between the time when insects were first suspected as virus vectors and the actual demonstration of this method of transmission. The first to prove experimentally the relationship between an insect and a plant virus seems to have been a Japanese farmer, Hashimoto, who, in 1894, worked with the dwarf disease of
rice and the leaf hopper, *Nephotettix apicalis* Motsch var. *cincticeps*.

About 1907 Ball, Adams, and Shaw suggested that there was some connection between curly top of sugar beet and the leaf hopper, *Eutettix tenellus* Baker. Smith and Boncquet confirmed this in 1915, showing that a single insect from an infected plant, placed on a healthy plant for 5 minutes, would transmit the disease.

Cook (1946) in an historical review of plant viruses and virus diseases, divided the history of virus study into three arbitrary periods. The first period began with the first record, in 1576, of a virus disease and ended in 1868 with a description of the variegation of *Abutilon striatum* Dicks. During this period there was no research as that term is now understood, but there were some important discoveries. The first was that the breaking of tulips was passed on by bulbs from plants with that characteristic; the second, that peach yellows and the mottlings of *Abutilon striatum* Dicks. var. *thompsonii* were transmissible by budding; and the third that when a mottled branch of *Abutilon striatum* var. *thompsonii* was grafted onto a healthy plant, the mottling appeared in the new green leaves.

Cook's second period began in 1882 with the work of Mayer whose study of the mosaic disease of tobacco showed it to be transmissible. The previously mentioned work of Iwanowsky and Beijerinck was included in this period. His
third period began about 1906 when the study of plant viruses was really starting, though it did not become intensive until 20 years later.

Smith (1948) suggested adding a fourth period starting in 1935 with Stanley's isolation of the tobacco mosaic virus. That discovery enabled workers in the field to visualize a virus as a definite entity, not as a mysterious agent whose existence was deduced only from the disease it produced.

It was during the last 15 years that the really serious study of the virus as distinct from the virus disease has been carried out. The physicist, the biochemist, and the serologist have all joined in the research in an effort to find out just what a virus is. By means of the electron microscope, the new technique of shadow micrography, X-ray diffraction studies, and the aid of the ultra-centrifuge much information on the size and shape of virus particles has been obtained. In the biochemical field a number of plant viruses have been isolated, crystallized, and their chemical composition studied.

The reproductive method of viruses is still unknown. For information on this subject, Smith (1948) stated that it would be necessary to return to the living plant or the living cell.
III. Economic Importance

In 1939 Sir Patrick Laidlaw stated that virus diseases are of great importance and the sum total of the disharmony they produce rivals that caused the visible bacteria.

Smith (1948) stated that, in England, the most important crop losses from virus infections occurred in the potato, sugar beet, cruciferous crops (especially the genus *Brassica*), strawberries, raspberries, and tomatoes. The importance of virus diseases to the potato grower in England was shown by the extensive imports, due entirely to virus infection, of 400,000 tons of seed potatoes annually from Scotland and Ireland. The sugar beet crop also suffered heavily from virus diseases, particularly the so-called virus yellows, which may reduce the crop from 15 tons per acre to 3 tons, while a crop, heavily infected in July, may lose 50% of its sugar content.

Cook (1946) and Bawden (1950) stated that precise figures of losses from virus diseases are extremely difficult to obtain, even estimates being difficult due to varying conditions. Cook gave some interesting estimates; reduction in value of the tobacco crop by 60%, an estimate in 1937 of the Research Committee of the Tobacco Research Council, when infection occurred at the time the plants were set out in the fields. In 1847 Berrien County, Michigan had 654,000 peach trees, and in 1890 only 42,863. Between 1890
and 1908 Botecourt County, Virginia lost 100,000 out of 130,000 peach trees. These losses were due to peach yellows and other virus diseases.

Cook also stated that potato losses in the United States of America had been estimated at from 5 to 50% at various times and in various places; tomato losses from mosaic as high as 75%; bean losses from 15 to 85%; onion losses at 25%; beet and tomato losses, due to curly top of sugar beet, as high as 75%. To these estimates he would add the cost of methods for control and eradication of these virus diseases.

Bawden (1950) stated that more than 1,000,000 peach trees had been removed in the State of Georgia due to phony disease of peach. In West Africa, on the Gold Coast, over 1,000,000 cacao trees have been destroyed by the swollen shoot complex of viruses. There, production of cacao declined from 116,000 tons in 1936 to 64,000 tons in 1945. He estimated that mild mosaic, or potato virus X, must reduce the world's tuber crop by at least 10%. He quoted an estimated annual loss of 40,000,000 lbs. of tobacco in the United States of America caused by tobacco mosaic virus.
IV. Nature of Viruses

What a virus is, has been the cause of much controversy. Some workers considered viruses to be essentially similar to bacteria, while others considered them to differ from bacteria in properties more fundamental than size.

Mayer (1886), E. F. Smith (1888), Iwanowsky (1892), and others considered virus diseases to be caused by bacteria small enough to pass through a filter. Allard (1916) expressed the opinion that tobacco mosaic was due to an ultra-microscopic organism. He seems to have been the first to have visualized a plant virus as a separate entity.

Beijerinck (1899) was the first to give evidence that bacteria were not the cause of tobacco mosaic, having failed to find bacteria in the filtered juice of diseased plants which retained the power to transmit the disease. This was the foundation of his theory of a "contagium vivum fluidum". Hunger (1905), who worked on tobacco mosaic in Sumatra, believed that the disease was caused by a non-living toxin produced by the cells of the host plant as a result of metabolism. This was sometimes referred to as the non-living toxin theory.

Of the physiological theories advanced as to the nature of a virus, the most important was the oxidizing
theory of Woods (1899) and Heintzel (1900). Both were probably influenced by the biochemical studies on enzymes at that time.

Allard (1916) attempted to isolate the virus of tobacco mosaic by means of talc and ammonium sulfate. Vinson (1927) tried to precipitate and isolate this virus by chemical means, using acetone, ammonium sulfate, and safranin. This safranin virus precipitate was shown to be inactive by Vinson and Petrie (1932), but activity was restored by removing the safranin with amyl alcohol.

Stanley (1935) gave the first description of the crystallization of this virus. He was also the first to isolate the virus as a tangible entity and showed it to be a protein. Best (1936), working independently in Australia, precipitated the tobacco mosaic virus at its isoelectric point and showed that the precipitate gave positive tests for protein. Bawden and Pirie (1937) showed that the virus was a nucleoprotein.

Since 1937 many workers have confirmed this specific protein in plants infected with tobacco mosaic virus, showing it to be a liquid crystalline protein shaped like a rod. Other viruses which have been crystallized are tomato bushy stunt as true three-dimensional crystals or dodecahedra; tobacco necrosis as thin plate-like laminae; southern bean mosaic in alternative forms of rhombic prisms and bipyramids;
and turnip yellow mosaic as small octahedra. Potato viruses X and Y and cucumber viruses 3 and 4 also have been crystallized, like tobacco mosaic virus, as rod-shaped particles. Tomato bushy stunt, tobacco necrosis, southern bean mosaic, and turnip yellow mosaic all seemed to crystallize in a spherical form. Bawden (1950) stated that the one thing common to all these proteins is that they are all nucleoproteins containing a ribose nucleic acid.

Cook (1946) stated it was impossible to give a very clear definition of viruses until more was known about them, though there were certain characteristics more or less common to all of them. These are (1) they are not visible under the ordinary light microscope; (2) many of them will pass through bacterial filters; (3) they increase only in the living cell; (4) they have not been grown in artificial culture media; (5) they cause characteristic symptoms in healthy host plants of the same species as those from which they were obtained; (6) they respond to temperature, humidity, and other environmental factors; (7) they are inactivated by certain temperatures and chemicals; (8) that they mutate; and (9) they behave in many respects like living organisms. Bawden (1950) defined a virus as an obligately parasitic pathogen with dimensions of less than 200 mp.
V. Nomenclature

Much confusion has been caused by the absence of any systemic basis for the nomenclature of viruses. Often the same virus has been given different names by different workers, leading to many synonyms. Sometimes the same name has been given to different viruses, adding still more to the confusion. This was due largely to naming viruses on the basis of the symptoms they cause, without realizing that similar symptoms may be caused by different viruses or that the same virus may cause different symptoms under different conditions or in different host plants.

In 1930 the International Botanical Conference formed an International Committee on Virus Nomenclature under the chairmanship of Professor James Johnson to consider the whole question of naming viruses. In 1935 this committee reported a scheme which was adopted but never published. This was an elaboration of Professor Johnson's original scheme, suggested in 1927, designed to cover names for strains as well as for separate viruses. In addition to the common name of the host followed by the term virus and a number, strains were indicated by a capital letter and sub-strains by a small letter.

Since 1935 new systems of nomenclature have been put forward so frequently that near chaos has been reached. Well established names have been replaced by complicated
unknowns so that the name used by one virus worker might mean little or nothing to another.

Smith (1937) modified the proposed scheme of the International Committee by using the Latin generic name of the host instead of the English name. He added to the confusion by keeping some of the numbers for individual viruses, as proposed in Johnson's scheme, and altering others.

Holmes (1939) introduced a binomial system of nomenclature, similar to that in use for plants and animals, with a trinomial for indicating strains. In this scheme ordinary tobacco mosaic virus became Marmor tabaci var. vulgare and aucuba mosaic virus, Marmor tabaci var. aucuba. However, Smith (1948) pointed out that too many unrelated viruses were lumped together in this genus. Other workers have prepared other schemes that add to the confusion.
VI. Virus Diseases

Smith (1948) suggested a rough classification of the external symptoms of virus diseases, but emphasized that this was not a classification of viruses, for a single virus can cause half a dozen distinct diseases depending on what host plant is infected. He divided the external symptoms into five groups or types.

The first group contained the mosaic diseases. The main symptom is a mottling of the leaf which may be light or dark green, yellow, or even white. The breaking of the color of the flower, as in the tulip, is also a symptom of mosaic virus diseases. He also included the ring spot diseases in this group. Most of these virus diseases are sap-transmissible.

The second group included the distorting diseases. Among these there is no mottling, and generally not much necrosis of the cells. Most of the diseases in this group have self explanatory names such as potato leaf roll, tobacco vein distorting disease, tomato big bud, and cranberry false blossom. Unlike the mosaic type, most of these are not transmissible by mechanical means but have a specific insect vector.

Necrotic diseases formed the third group. In this type there is little or no mottling, but the cells are killed by necrosis which may be confined to the leaves, as
in tobacco necrosis, or may be systemic and often lethal, as in tomato black ring and tomato streak.

The fourth group was concerned with outgrowths and tumors. The commonest type is enation, consisting of a secondary leaf growing out from the underside of another leaf. These may vary in size from a few millimeters to larger than the original leaf. They are caused by several viruses, but only on certain hosts; for example, tobacco rosette virus complex on tobacco, and tomato black ring virus on frame cucumber plants.

The fifth group contained the yellows diseases. One or two viruses give rise to a uniform yellowing of the leaves, a different effect from mosaic mottling where there is a combination of color shades. Aster yellows and sugar beet yellows are good examples.

Bawden (1950) stated that there were two kinds of internal changes, the destruction or modification of normal tissues or cells, and the production of peculiar bodies not found in healthy cells. To these can be added the abnormal accumulation of starch characteristic of such virus diseases as potato leaf roll, curly top of sugar beet, and aster yellows. The peculiar bodies were later mentioned by Bawden as X-bodies. Smith (1948) stated that these X-bodies were intracellular, sometimes ameboid in shape, and usually associated closely with the nucleus.
A common phenomenon among plants is that they may act as symptomless carriers of virus diseases. These are of two kinds; in one there is a mild or severe reaction, then the symptoms disappear and the plant appears normal though still infected with the virus. This is the usual reaction of tobacco plants to ring spot viruses. In the second kind, there is no initial reaction to infection, the plant appearing normal in every way. This happens in potatoes, hops, strawberries, and raspberries.

Certain host plants react to infection by certain viruses so that the virus is localized in the inoculated leaf. This may be permanent or only temporary, followed by systemic infection. The usual reaction is the development of many necrotic spots or rings, termed local lesions. In cases where there is no systemic spread the use of local lesions permits the recognition of large numbers of successful transmissions on a single plant. This method, which has been compared to Koch's plate method with bacterial cultures, makes possible the quantitative study of plant viruses and allows for comparative estimates of virus concentrations.

Bennett (1940) divided virus movement in plant tissues into three main relationships restricted to the phloem and the parenchyma. The first was a relationship in which the virus was restricted, more or less, to the parenchyma including the epidermis. The second was a
relationship in which the virus was restricted closely to the phloem. The third relationship was one in which the virus occurred in both the phloem and the parenchyma.

Restriction to the parenchyma would handicap a virus for the movement is relatively slow and invasion of all the parts of a plant would require a long time. The amount of inoculum would be limited and the spread from plant to plant would occur less often than if the virus moved through the phloem. Such a virus would require special conditions to survive, especially if it were not seed transmitted and if its host plants were annuals.

Hutchins (1939) found that when whole root sections from peach trees infected with phony peach, were grafted onto the roots of healthy peach trees, the disease was transmitted in all cases where union took place. When bark from diseased roots was grafted onto healthy roots, no transmission took place. This indicated that the virus of phony peach is confined to the woody cylinder, probably moving and multiplying in the wood parenchyma or in the medullary rays, or in both.

Hutchins and Rue (1939) presented evidence that this virus is destroyed by subjecting dormant trees to a temperature of 48°C. for a period of 40 minutes. Therefore summer temperatures, high enough to inactivate the virus in the parts above ground, would be expected to cause restric-
tion of the virus to the roots and lower parts of the trunk.

There are probably several viruses that are more or less restricted to the phloem in their increase and movement in the plant. Studies by Bennett (1927, 1934) have shown that the viruses of raspberry leaf curl and sugar beet curly top may be confined to certain parts of a plant by destroying the phloem connections between the inoculated portion and other parts of the plant at the time of inoculation.

In beets affected by curly top, Esau (1933) found necrosis only in the primary and secondary phloem and the pericycle. She concluded, on the basis of extensive anatomical evidence, that the virus was active mainly in the phloem.

Characteristic symptoms of phloem limited viruses might include phloem necrosis, vein distortion, curling and crinkling of leaves due to growth disturbances in the veins, yellowing, and dwarfing of parts or entire plants. No mottling of the mosaic type would be expected to occur. This type of virus would not be seed borne, being unable to enter the gametes or pass through the meristematic or parenchymatous bridges separating the mother plant from the sporophyte.

Mechanical inoculation by rubbing or needle punctures would rarely cause infection due to the difficulty of placing the virus directly into the phloem. However, there
are certain viruses not limited to the phloem that also show this characteristic, so this cannot be accepted as positive evidence that a virus is limited to the phloem.

Phloem limited viruses should be almost exclusively insect transmitted. The vectors should be relatively few in number of species and should have a greater degree of specificity than is found in vectors not limited to feeding exclusively on the phloem. Only insects that habitually feed on the phloem, allowing the virus to pass through their bodies and to be injected with their saliva into a plant, should qualify as vectors.

Under the above conditions diseases such as peach yellows, peach rosette, potato leaf roll, potato yellow dwarf, aster yellows, cranberry false blossom, peanut rosette, and spike disease of sandal may be caused by phloem limited viruses. Recently published indirect evidence confirms this view as to peach yellows, little peach, peach rosette, and potato leaf roll.

Common tobacco mosaic is a well known example of a virus disease that occurs in both the parenchyma and the phloem, but numerous other viruses have a similar tissue relationship. Local lesions and mottling are the prominent symptoms in most cases. Phloem necrosis and other phloem disturbances occur less often and inconspicuously, indicating that the virus concentration in the phloem may be low.
Evidence tends to show that the vector relationships of viruses that occur in both the phloem and the parenchyma are much less specific than those of phloem limited viruses. Many mosaic type viruses are known to be transmitted by more than one species of insect. Drake et al. (1933) showed that the virus of yellow dwarf of onion was transmitted by more than 50 species of aphids. K. M. Smith (1937) listed 21 viruses transmitted by *Myzus persicae* Sulz., 10 by *Macrosiphum* *gei* Koch, and 8 by *Macrosiphum pisi* Koch. With a few exceptions these insect transmitted viruses produce mottling or local lesions.

K. M. Smith (1948) suggested adding a fourth group to Bennett's three, that of a virus apparently confined to the xylem. The example quoted was that of Pierce's disease of grape and alfalfa which is transmitted only by leaf hoppers that feed on the xylem. If the leaf hopper vector, *Draeculocephalo minerva* Ball., is prevented from feeding on the xylem, infection does not take place.
VII. Strains, Immunity, and Serological Relationships

There seems to be little doubt among investigators that plant viruses share with animal viruses a characteristic of living things, the power to mutate. It has been found that viruses vary, few occurring in single forms, but having variants usually called strains. Bawden (1950) stated that over 50 strains of tobacco mosaic virus have been recognized.

Many strains, found or induced in experimental work, would have little chance to survive naturally unless their infectivity was greater than that of the original virus. Jensen (1933) made 26 inoculations from yellow spots occurring naturally in tobacco mosaic. Many of these inoculations differed markedly in their symptomatology and infectivity from the type virus. Experiments with cucumber mosaic virus showed a similar state of affairs. Some other viruses with naturally occurring strains are sugar beet curly top, aster yellows, and potato yellow dwarf.

Plants may show a natural immunity to a particular virus, but the usual type of immunity is induced, occurring only between strains and closely related viruses. This is a non-sterile immunity where one virus immunizes the plant against certain other viruses. This is not similar to the sterile immunity acquired in some human virus diseases, such as smallpox.
Bawden (1950) stated that infection by one strain of a virus does not prevent another from entering the plant but prevents the second strain from multiplying sufficiently to produce its usual symptoms. This action depends upon how thoroughly the first strain is established. The protection acquired by an infected plant is usually specific against serologically related virus strains but not against unrelated viruses.

Serological work on plant viruses may be said to have begun with the work of Dvorak (1927) who prepared antisera separately against healthy and mottled Triumph potatoes. Purdy-Beale (1928, 1929, 1931) provided proof that a virus infected plant contained a specific antigen, using extracts of healthy tobacco plants and of plants infected with tobacco mosaic virus.

Gratia (1933) showed that plants infected with different viruses contained specific antigens for each virus. Birkeland (1934) further demonstrated that extracts from plants infected with viruses thought to be related contained common antigens.

Most workers considered that the viruses themselves were the antigens, though it was possible that the specific antigens were produced by the host plants after infection. The evidence of many experiments now strongly favors the view that the specific antigens are the viruses themselves.
There are four types of serological reactions which can be used: (1) the precipitin reaction, where precipitation occurs when the antigen and its antibody are brought into contact; (2) complement fixation; (3) neutralization of the infectivity of the antigen; and (4) anaphylaxis.

The precipitin reaction is the one most commonly used. Complement fixation, though more sensitive, has not been widely used in plant virus work, due, probably, to the difficult and laborious technique.

Serological methods have been used in differentiating between serologically unrelated viruses that cause the same symptoms on their host plants. Bawden (1941) showed that tobacco necrosis could be caused by a number of serologically unrelated viruses though they could not be distinguished by their respective symptoms on the host plants.

Bawden (1950) stated that serological techniques have been successfully applied to about 15 different plant viruses but have failed with many others. Experiments have shown that the virus content of sap used in serological testing is correlated with the serological activity that is demonstrably antigenic. Up to the present, serological methods have been successful mainly with sap-transmissible viruses.

The particle shape of viruses has an effect on serological reactions. Rod shaped viruses, such as tobacco
mosaic, when mixed with their antisera agglutinate rapidly, forming large clumps with a fluffy open structure. Spherical viruses, such as tomato bushy stunt, agglutinate more slowly, forming smaller clumps that are dense and granular. Experiments have shown that rod shaped particles, when smaller than normal, behave in a manner similar to spherical shaped viruses.
VIII. Natural Methods of Transmission

The most important natural method of transmission is by insect vectors. Other natural methods are by contact, by seed, through the soil, by natural grafting, and by all methods of vegetative propagation.

Except under unusual circumstances only the more infectious viruses, i.e. those occurring in high concentration in their host plants, are spread by contact. Examples are given by tobacco mosaic virus and potato virus X. Since tobacco mosaic is so infectious it is spread not only by contact, but is also carried on the hands of workers and on contaminated implements.

Transmission of viruses by seed is rare but not negligible. This method of virus transmission seems to be characteristic more of leguminous plants than of others. The best known case is that of mosaic of bean, first demonstrated in 1919 by Reddish and Stewart. Lettuce mosaic is another virus that is regularly seed transmitted, about 5% of the seed set by diseased plants being infected. The exact reason for the rarity of this type of transmission is still an unsolved problem. Several theories have been advanced, such as the anatomical isolation of the sporophyte, the inactivation of the virus by adsorption onto the seed protein, or by maturation processes, but none has been supported by convincing experimental evidence.
Soil transmission of viruses is also rare, though there are several cases. The best known case is that of tobacco necrosis which occurs in the roots of apparently normal tobacco plants and others. This highly resistant virus is washed down into the soil where it comes in contact with the roots of other plants. A wound, necessary to permit the virus to enter the plant, is provided by the breaking of the root hairs during the growth process. Other viruses spread by contaminated soil are tobacco mosaic, winter wheat mosaic, and lettuce big vein.

Another method of transmission is by natural root grafting and by parasitic plants, especially the dodder, Cuscuta sp.. Tilford (1942) stated that natural root grafting may be responsible for the spread of elm phloem necrosis among closely planted elms. Bennett (1940) and Johnson (1941) found that dodder stems could be used successfully to transmit virus diseases. Bennett found that the dodder, Cuscuta californica Choisy, transmitted cucumber mosaic virus and Cuscuta subinclusa Durand and Hilgard, the virus of sugar beet curly top. Johnson, using Cuscuta campestris Yuncker, transmitted aster yellows, bushy stunt, cucumber mosaic, sugar beet curly top, and tobacco mosaic viruses, but not tobacco ring spot or pea wilt viruses.
Kunkel (1943) favored the use of the dodder in transmission to provide new hosts for experimental work on viruses that are difficult to study in their original host plants. By using *Cuscuta campestris*, he transmitted cranberry false blossom from the cranberry, its only previously known host, to 28 different species belonging to 10 different families, including tobacco and tomato plants. In the tomato plant he demonstrated that there were at least two distinct strains of the virus involved. False blossom was also transmitted to the periwinkle, *Vinca rosea* L., a heat enduring plant, where, by the use of moderate heat treatments, the disease was cured. He also used dodder transmission in determining whether two viruses, such as witches' broom and aster yellows, were identical. The experiments showed that they were not identical.

Transmission by vegetative propagation was known long before our present knowledge of virus diseases. Since the majority of plant viruses are systemic all organs of the plant, except the seed, being invaded, the virus persists in the organs of vegetative reproduction such as rhizomes, tubers, and bulbs. The classic example is that of the potato whose tubers pass on the viruses with which the plant gets infected each year until complete "degeneration" has set in. Dahlias, infected with spotted wilt or mosaic, irises and daffodils, infected with mosaic
or stripe, reproduce the diseases indefinitely. The oldest recorded propagation of this type is the breaking of tulips in 1576. Propagation by cuttings or suckers from infected plants also results in the production of diseased plants, though apparently healthy plants also occur.

The most important method of virus transmission is by means of insects. The great majority of insects that act as virus vectors feed by sucking plant juices, with only a few authenticated cases of insects with biting mouthparts acting as vectors. The first instance, in England, of a virus disease being transmitted by an insect with biting mouthparts was reported by K. M. Smith and R. Markham in 1949. They found that turnip yellow mosaic was transmitted by two species of flea beetles, the mustard beetle, an earwig, and a grasshopper. However, the great majority of insect vectors are found among the leaf hopper and aphid families.

Smith (1948) and Bawden (1950) stated that one of the characteristics of insect-virus relationships was the specificity of insect vectors, bringing some interesting facts to light. Due to the selectivity in transmission shown by an aphid it was discovered that a certain potato virus disease was caused by two viruses instead of one. When parallel transmissions were made from the same diseased plant to separate indicator plants, such as tobacco, by sap
inoculation and by an aphid, two separate and different diseases were produced. Further investigation revealed that there were two viruses in the potato plant, both sap-transmissible but only one aphid-borne.

K. M. Smith (1945) found tobacco rosette to be caused by the joint action of two viruses, vein distorting and mottle. In this disease only the mottle virus is mechanically transmissible and is not transmitted by the aphid when it is by itself. If both viruses are together in a plant the aphid, *Myzus persicae* Sulz., can pick up and transmit them.

In certain cases it appears that a related strain of a virus may have its own specific insect vector differing from that which transmits the type strain of the virus. The specificity seems to be absolute, neither insect being able to transmit the other virus. This occurs in potato yellow dwarf which has two variants, the New York and the New Jersey strains. *Aceratagallia sanguinolenta* Prov., the leaf hopper which transmits the New York strain, cannot spread the New Jersey strain, whose leaf hopper vector is *Agallia constricta* Van D..

This also seems to be true of sugar beet curly top, in which the North American virus is spread by a specific leaf hopper, *Eutettix tenellus* Baker, while the Argentine strain of the virus is transmitted only by another leaf hopper, *Agalliana ensigera* Oman.
IX. Relations between Viruses and their Vectors

Smith (1948) divided transmission between insect vectors and particular groups of viruses into three types, non-persistent, persistent, and mechanical.

Non-persistent viruses are those which are lost rapidly by the insect unless it has access to a fresh source of the virus. In a series of successive 24-hour transfers from plant to plant only the first plant or two are affected by this type of virus.

Persistent viruses are those which are retained by the insect for long periods, often for life, without access to a fresh source of the virus. In a series of successive transfers the first plant is not usually infected while the others become infected. This occurs because of the delay in the development of infective power or the incubation period of the virus in the insect.

Mechanical transmission refers to the purely passive transfer of a virus by contamination of the mouthparts of a biting insect.

The difference between the first two types may be quantitative rather than qualitative. This may be one reason why non-persistent viruses, in contrast to persistent viruses, are usually sap-transmissible. If it is supposed that non-persistent viruses are all present in high concentrations in their source plants, they would be easily
transmissible by sap inoculation, whereas a virus present in a concentration too low for sap transmission could be insect transmitted, since this is a more efficient method of transmission.

Bawden (1950) stated that many differences in behavior between viruses could be explained quantitatively, as suggested above, but some facts seemed incompatible with this idea. He stated that this explanation did not apply to viruses like sugar beet yellows and maize streak, which seem to occur in high concentrations in the plant but have not been transmitted mechanically. As a rule the so-called persistent viruses are not sap transmissible, one notable exception being potato yellow dwarf.

The vectors of all persistent viruses seem to need a latent period, after feeding on an infected plant, before they can infect a healthy plant. This latent, or incubation, period varies from less than an hour for sugar beet yellows to 16 days for corn stunt. One interpretation of this variation is that the latent period is the time needed, in the vector, for the virus to undergo some developmental change necessary for inoculation. Another explanation is that it represents the time needed by the virus, ingested by the vector, to be ejaculated or pass through the body to reach the salivary glands. A third theory, widely accepted, is that viruses have to multiply in the vector's
body to an amount necessary for infection.

If plant viruses do multiply in an insect's body, it becomes, in part, an animal virus, suggesting affinities between plant and animal viruses. Evidence supporting this hypothesis was presented by Black (1941). He colonized a number of leaf hoppers, of uniform size and age, upon a source of aster yellows virus for a given period of time. The insects were then removed and colonized on a rye plant which is immune to the virus. In this way all the insects received the same dose of the virus. At 48 hour intervals about 50 leaf hoppers were removed, ground up, diluted to varying strengths, and inoculated into virus free leaf hoppers which were then caged on healthy aster seedlings. Black found that the insects which had been longest on the rye plant would withstand the highest dilution, while still producing infection upon inoculation into virus free leaf hoppers. He interpreted this as indicating virus multiplication since, presumably, all the insects had received the same initial dose of the virus.

Kunkel (1937) had also carried out experiments with leaf hoppers and aster yellows virus which, he considered, supported the theory of virus multiplication in the insect. He found that if insects, infected with aster yellows virus, were heated for a long period they lost the power to infect unless recolonized on a fresh source of the virus. If
infected insects were heated for a shorter period, they also lost the power to infect but gradually regained it after an interval of time. He interpreted this to mean that in the first case the virus was completely destroyed by the prolonged heating and, in the second case, it was reduced below the threshold of infection but regained infective power after a lapse of time by multiplication in the insect's body.

Smith (1948) claimed that the storage of the virus, rather than multiplication, was an alternative explanation for these results and the findings of other experiments. He stated that it has been shown that the length of time a leaf hopper retains the virus is correlated to the length of time of feeding on the source of the virus.

Bawden (1950), in an addendum to his chapter on viruses and their vectors, stated that Black had obtained evidence that clover club leaf virus multiplies in its insect vector, *Agalliopsis novella* Say. From one infective insect the virus had been transmitted through the eggs for twenty successive generations without recourse to a fresh source of the virus. This result was totally incompatible with the hypothesis that the original insect contained all the virus detectable in the progeny of later generations. It seems that this virus can maintain itself indefinitely in its insect vector.
Another case of the inheritance of a virus by the progeny of an infected insect is that of the leaf hopper, *Nephotettix apicalis* Motsch, the vector of the dwarf disease of rice. Fukushi (1934, 1935, 1939) found that the transmission of rice dwarf virus is determined solely by the female. If the female is infected, some of the progeny will inherit the virus through several generations. If the female is not infected, the offspring will be virus free. The record seems to be inheritance to the seventh generation. In another experiment by Fukushi more than 1,000 plants were infected by 26 different leaf hoppers derived, in five generations, as the progeny from one egg.

Non-persistent virus vectors can infect a healthy plant immediately after leaving a diseased plant. Except cacao swollen shoot viruses which are transmitted by mealy bugs, all non-persistent viruses, so far identified, have aphids as vectors. The fact that the process of becoming infected and infecting a healthy plant occupied a vector only a few minutes led many workers to conclude that the insects acted only in a mechanical way, as is done by needle inoculation. The rapid loss of infectivity by these vectors obviated the need of investigating the multiplication of these viruses in the insects.

If the transmission were merely mechanical it might be expected that the viruses most readily transmitted by
artificial inoculation also would be most readily transmitted by insects. However, tobacco mosaic virus and potato virus X are two of the most easily transmitted viruses by mechanical inoculation but neither of these normally appears to be aphid transmitted.

Some light has been thrown upon the course of the virus in the insect's body by Storey (1932), working on the streak disease of maize. He has shown that there are two distinct races of the insect vector, the leaf hopper, Cicadulina mbila Naude. Both races, of the same species, show no visible difference. The sole difference is in the fact that one race, called active, can transmit the streak virus, while the other, called inactive, cannot do so. If the wall or the alimentary canal of an inactive insect should be punctured, just before or after it has fed on a diseased plant, it becomes active and able to transmit the virus. This suggests that, for some reason, the virus cannot diffuse through the walls of the gut, in the inactive insect, in order to reach the salivary glands. That the inactive insect does imbibe the virus is shown by the recovery of the virus from the feces. Although there are other factors, it appears that the permeability of an insect's gut wall may play a part in determining an insect's ability to transmit a plant virus.

Smith (1948) and Bawden (1950) noted a curious
feature in the relationships of tomato spotted wilt and its vectors, *Frankliniella insularis* Franklin and *Thrips tabaci* Lind. The virus can be transmitted by the larvae but not by the adult thrips unless it has fed as a larva on a source of the virus. The adult seems to be unable to pick up the virus from a fresh source and become infected. This anomaly is still unexplained.

Bawden (1950) suggested that if the results of Storey's work with maize streak could be applied to the spotted wilt virus, it might be possible to show that the permeability of the gut wall differs in the larval and adult thrips.

There seems to be no doubt that the insect's saliva is the vehicle of virus transfer. Smith (1941) demonstrated this by feeding leaf hoppers, infected with curly top virus, on drops of sugar water. He then fed known virus free leaf hoppers on the same drops. After this feeding the leaf hoppers were colonized on sugar beet seedlings. They infected a portion of the seedlings with curly top which they had picked up from the salivary secretions left by the first lot of insects in the drops of sugar water.
X. Control Measures

The control or eradication of plant diseases is the final aim of all plant disease studies. Most plant virus diseases have been discovered or identified since 1914. Cook (1946) listed only 27 virus diseases that were discovered and identified before that year. It is possible that many virus diseases existed in wild plants before invading cultivated crops. Cook suggested that curly top of sugar beet may have existed in wild plants in southern California before the introduction of the sugar beet into that area. Spotted wilt of tomato was discovered in a short period of time in four widely separated countries, Argentina, Australia, England, and the western United States. It does not appear probable that this disease spread that rapidly in so short a time.

Heat has been used to cure some virus diseases in plants. It is necessary that the host plant should be able to withstand higher temperatures than the virus. The first established virus disease reported cured by heat was peach yellows. Kunkel (1935) reported that trees recovered from peach yellows if they were kept at a temperature of more than 35°C. for two weeks. He also reported that this method cured peach trees of little peach, red suture, and rosette, but not peach mosaic. He showed that the recovery was not the masking of symptoms by high temperatures or did occur
because avirulent strains were segregated. However, economically heat therapy does not seem to be applicable generally and it does not make a plant immune to reinfection by the eliminated virus.

The practice of good farming can help to reduce the incidence of some virus diseases. The destruction of weeds may eliminate alternative hosts of both viruses and their insect vectors. Bawden (1950) stated that the probability that a long rotation of crops will reduce soil borne diseases is too obvious to be stressed.

Variations in normal cultural practices seems to offer some hope for the control of virus diseases. Varying the sowing dates may affect the incidence of some diseases or may reduce the losses even if the incidence is unaffected. Early sown sugar beets suffer less from both curly top and yellows than late sown crops because the latter are at a more susceptible stage when the vectors are active.

Too few infective insects or other virus sources usually come into crops to infect all the plants, while the number entering will probably be independent of the density. Therefore, doubling the number of plants per unit area could be expected to halve the proportion of the crop that becomes infected. Van der Plank and Anderson (1945) have shown that this was realized with tomato
spotted wilt infecting tobacco, in South Africa, where the disease enters the crop early in the spring, spreading little thereafter. By setting double the number of plants and thinning to normal after the thrips invasion occurred, the diseased plants could be removed, leaving a full stand of healthy plants.

The simplest method of avoiding virus diseases is to grow disease resistant varieties of plants. Several diseases are now being combatted by raising such varieties. In parts of the United States of America where curly top made sugar beet growing impossible, new varieties are giving profitable crops. The term resistance is usually applied to varieties that withstand losses in the field, though it is often difficult from published reports to discover to what type of behavior they owe this property. Tolerance, rather than resistance, was probably the characteristic of beet varieties first used to combat curly top, but later varieties seem to combine resistance to infection with tolerance.

Immunity is an ideal goal, but only a few crops show any evidence of the requisite genes. The only success achieved so far is with a potato (U.S.D.A. seedling 41956) that is immune to potato virus X, which is widely distributed in most commercial varieties.

Plants that are tolerant to viruses, or are symptomless carriers, have proven to be valuable economically,
producing good yields where intolerant varieties fail. However, the use of tolerant varieties is not a good control measure for they often provide the main sources of virus perpetuation endangering other varieties and crops. Since tolerant sugar beet varieties were introduced into the United States, curly top has been recorded as a serious disease of many different crops.

Resistance to infection and extreme sensitivity are better features to breed for, in new varieties, than tolerance. These features tend to reduce the rate of spread and the number of infected plants, instead of providing a large number of host plants that can carry the virus, as happens with tolerant plants. Genes determining these two characteristics may exist in many species though only the potato, tomato, and Nicotiana sp. have been studied in any detail, and only with a few viruses. The relative effects of tolerant and intolerant varieties in encouraging the spread of viruses is shown by the distribution of some viruses in different potato varieties. Potato viruses A and X occur almost universally in commercial stocks of tolerant varieties but are rare in intolerant varieties.

However, due to the extreme mutability of viruses, it is vain to hope that the plant breeder can successfully control virus diseases by breeding new varieties of plants.
Viruses, in mutating, can be expected to give rise, continuously, to new strains that will attack new hosts. There is evidence of such mutations for sugar beet varieties, introduced as resistant to curly top, are now reported as suffering losses. The Pearl Harbor variety of tomato, which resists spotted wilt in Hawaii, is not resistant in the United States of America, and potato seedlings, hypersensitive to potato virus Y in Australia, are not hypersensitive to all the strains that occur in Great Britain.

The control of insect borne virus diseases is a complex affair. The methods vary with the crops, the conditions under which they are grown, and the habits and size of the insect vector. The obvious method of control would be to eliminate the vectors. Plants raised in greenhouses can be kept free of insect vectors by regular spraying or fumigation, but controlling insect vectors in field crops is another matter. Dusting and spraying crops with the proper insecticides, and fumigating under portable tents or towed covers for low lying crops are established methods. These methods are used mainly for controlling insect pests, but there have been few claims of success in the use of insecticides for controlling virus diseases.

The control of mechanically transmitted viruses depends on reducing the chances of infection through machines, tools, and the workers. Tobacco mosaic virus
is an excellent example of a mechanically transmitted virus. It has been shown that this virus can spread by plant to plant contact. Thus it needs only a few infected plants to infect a large proportion of the crop in a short time. Several investigations have shown that plants can become infected, especially during weeding or transplanting, by workers whose hands have become contaminated from handling diseased plants. Barn cured tobacco is quite likely to be contaminated and the virus is often found in commercially prepared tobacco. Workers should not use tobacco and should wash their hands thoroughly with soap and water when handling tobacco or tomato plants. These precautions, together with clean tools, should reduce the incidence of tobacco mosaic virus.

Another important source of tobacco mosaic virus is soil contaminated by plant debris and roots from previous crops remaining in the ground, for the virus can remain active in such debris for years. Few plants become infected if the contaminated soil is undisturbed but such work as weeding leads to a high rate of infection.

Cultural operations should be carried out when the plants are dry for it has been found that more than four times as many plants become infected during cultivation when crops are wet with rain or dew as when they are dry.
XI. Phloem Necrosis of American Elm

A. Discovery and Symptoms

Swingle (1942) stated that the American elm, first in value as a shade tree and tenth among hardwoods in stumpage and log sales, is attacked by many diseases. Among these diseases phloem necrosis is considered as great a destructive agent, in the Mid-west, as the Dutch elm disease.

In 1893 and 1899 H. Garman reported an epidemic dying of elms in Kentucky. S. A. Forbes (1912) reported the dying of elms described by Garman as having occurred in Illinois since 1882. Though their descriptions of the symptoms resemble phloem necrosis, in many respects they are inadequate for positive identification of the deaths as having been caused by phloem necrosis. This was due to the lack of knowledge of tree pathology at that period.

Phloem necrosis attracted attention when the disease was reported from Ironton, Ohio in 1918 and from Dayton, Ohio in 1927. In Chillicothe, Ohio an epidemic began in 1935 that killed off, in the next two years 1,000 trees, 50% of the city's elms. These elm trees died in three to thirty-six months after the first apparent symptoms were observed. No diseased tree was observed to recover.

The foliar symptoms are first noticeable in the extreme top of the tree at the outer tips of the branches
where the leaves droop. The leaf blade curls up at the margin, giving a trough-like or narrowing effect. This narrowing of the leaves causes the foliage on the crown to appear thin. The leaves often become brittle, turn yellow-green, later yellow and defoliation follows, usually through the entire crown. On small trees there is usually no curling of the leaves but the crown becomes yellow, followed by defoliation and death.

In the roots of an infected tree the first symptom is the dying of the small fibrous roots. A characteristic discoloration develops in the inner bark, or phloem tissue, of the larger roots before death. The discoloration is at first yellow but soon becomes a typical butterscotch color, often turning a golden-brown or raw sienna which may contain scattered brown or black flecks. This discoloration must be present when a sample is taken for the phloem tissue will darken in a few minutes when exposed to the air. When a piece of moderately discolored tissue is put into a stoppered vial or held in the closed hand for a while, a faint but distinct odor of wintergreen can be detected. This odor is not detectable in tissue from healthy trees.

The typical inner bark discoloration and the wintergreen odor may be considered as specific symptoms of phloem necrosis. The defoliation, although usually the first noticeable sign of the disease, cannot be considered
specific for it is similar to those of Dutch elm disease, drought, girdling, some nutrient deficiencies, and other diseases.

Swingle (1938) published some experimental work on finding the causal agent of the disease. Over 4,000 attempted plate isolations, averaging 10 plantings each, were made from the roots, trunks, and branches of over 400 diseased trees. No organism was secured consistently or seemed to be associated with the disease. Over a two year period, inoculations with the organisms obtained from these isolations failed to indicate their pathogenicity. Histological study also showed no organism to be consistently present.

The direct insertion of diseased tissues into 72 healthy elms did not result in transmission of the disease. Healthy elms, with injured roots, planted in soil taken from around diseased trees, remained healthy.

In July 1937, 21 healthy American elms were grafted with bark patches from diseased trees, the patches showing typical phloem necrosis discoloration. In August 1938, 14 of these trees showed complete symptoms of phloem necrosis, while other healthy elms nearby remained healthy.

In January 1938, 20 healthy trees were grafted with branch scions from diseased trees while the roots of 26 healthy elms were grafted with diseased root scions. Eight
control trees had healthy branch and root scions grafted to them. The branch grafts were successful on the control trees and 15 of the test trees. Up to September 12, 1938 all control trees and unsuccessfully grafted trees remained healthy. Of the 15 successfully grafted trees, 13 showed transmission and complete symptom expression. Up to that date 5 of the 26 trees grafted with diseased root scions showed transmission of the disease and complete symptom expression. Whether the root grafting on the other trees was successful could not be determined until they have been dug up.

From these experiments it appeared that this disease was of a virus nature and was systemic. The mode of transmission under natural conditions was unknown at that time.

Tilford (1942) reported that the American elm, Ulmus americana L., and its varieties, the vase and moline elms, are very susceptible to phloem necrosis. He reported that numerous English elms growing in an infected area, Columbus, Ohio, have not been affected.

Swingle (1942) reported that artificial inoculation experiments have shown that trees may be infected for six months to a year or more before symptoms become apparent.

McLean (1944) reported that the winged elm, Ulmus alata Michx., may also be susceptible. His experiments and
nistological studies of the disease have shown that the virus moves out of the diseased graft into the tree within eight days. In diseased roots he found that there was no discernable deviation from that of healthy roots. In phloem tissue developing after infection of the roots, degeneration seemed to follow the maturation of the primary sieve tubes, usually consisting of hypertrophy of the nuclei and cells surrounding the mature sieve tubes. He observed no distortion of the xylem tissue. Under microscopic observation, the most striking characteristic of older diseased phloem tissue, of both root and stem, was the almost complete destruction of sieve tube cells with a marked increase in the number and size of parenchyma cells.

McLean found that diseased trees which were pruned back or defoliated after inoculation had symptoms sooner than untouched inoculated trees.

He found that the virus did not move out of a diseased bark patch into an inoculated tree, after isolation of the patch by removing a section of bark tissue completely around it. Likewise the virus did not move through the xylem cylinder to the roots when a band of bark was removed from around the stem below the graft inoculation. He inferred from these results that the virus did not move through the xylem.
B. Discovery of the Insect Vector

In 1940 the Bureau of Entomology and Plant Quarantine, in co-operation with the Bureau of Plant Industry, Soils, and Agricultural Engineering, established a laboratory at Columbus, Ohio, to study phloem necrosis and to determine its possible insect vectors.

Baker (1948, 1949) and Whitten and Baker (1948) have published the results of these experiments. In late August and early September, 1940, some adults of the leaf hopper genus, Erythroneura, were collected from elm trees in the Columbus area and confined for 4 days in cloth sleeves placed over the foliage of diseased trees. After this period of feeding, the insects were divided into two lots and placed in two cloth-covered cages, each containing four healthy elm seedlings. The insects were not disturbed again, being left to feed until they died. The cages were kept covered during the active insect seasons through 1942. No symptoms having developed up to this time, the cages were removed and the trees left to grow unprotected. Each succeeding year the trees were examined for signs of the disease. In August, 1945, 3 of the 8 trees showed typical symptoms of phloem necrosis. Two died that summer, the third in the following spring. The other 5 trees remained healthy. After these results further extensive tests were started.
The *Erythronoeura* specimens used in the 1940 tests could not be recovered, so that the species involved remain unknown.

In early surveys of elm inhabiting insects, in the disease region, several species of the leaf hopper genus, *Scaphoideus*, were collected. One of these species, *Scaphoideus luteolus* Van D., is a consistent inhabitant of the elm tree. Although this species is difficult to separate from closely related ones in the adult stage, it was found to differ markedly from the others in the nymphal stages. This difference facilitated their collection and permitted the establishment of an extensive series of transmission tests from 1941 through 1943. No tests were made in 1944 and only a few nymphs were used in 1945.

In July 1946, a small number of nymphs were collected, and after having fed on diseased foliage for various periods, were placed under test on healthy one-year-old American elm seedlings.

On July 12, two seedlings were exposed to nymphs and adults that had fed for the previous 9 days on diseased elm foliage; on July 15, five seedlings were exposed to nymphs that had fed the previous 12 days; and on July 26, two seedlings were exposed to nymphs and adults that had fed the previous 3 days. In all cases the infective insects were left on the test seedlings until they died. When all
the insects were dead, the seedlings were placed in a
cloth-covered cage and sprayed with DDT as an additional
precaution against possible contamination by unwanted insects.

Symptoms of phloem necrosis are seldom visible
before mid-June. In late June 1947, all test trees were
checked routinely for the possible appearance of the
disease. At this time one of the two seedlings placed under
test on July 12, 1946, showed typical symptoms of phloem
necrosis and by the first of August the foliage was dead.
Upon removal and examination, pronounced symptoms were
found in certain portions of the still living phloem near
the ground line.

Before discarding this seedling three bark patches
were removed and grafted onto three healthy seedlings to
determine whether the virus could be transmitted from the
test seedling. In October 1947, one of these seedlings,
showing early symptoms of phloem necrosis, was removed to
a propagation room where it continued to grow until January,
when its foliage suddenly died. At this time the seedling
showed typical late stage symptoms of phloem necrosis.

On July 2, 1947, one of the seedlings placed under
test on July 26, 1946, was found to have died so suddenly
that its leaves had failed to abscise and still hung on.
This phenomenon is by no means uncommon among naturally
infected trees growing in the open. Examination of this
seedling revealed the typical phloem necrosis discoloration.

These test trees developed symptoms in less than a year after being exposed to infected insects. This contrasted strikingly with a possible incubation period of five years where species of *Erythroneura* were used. The evidence favored transmission of the virus by this species of *Scaphoideus*, whereas transmission by species of *Erythroneura* is more doubtful due to the non-protection of the test trees in the last two years before the onset of the disease. Among several hundred other test trees in the same and adjoining cages and among several thousand trees in a nearby nursery there was no other evidence of insect transmission of the virus.

The *Scaphoideus* insects used in the 1946 tests were recovered and, after the development of disease symptoms in the test trees in 1947, were forwarded to the Division of Insect Identification. According to P. W. Oman, all were found to belong to a single species, *Scaphoideus luteolus* Van Duzee. Oman's determination of these specimens was based on comparison with type specimens of *S. luteolus*. Therefore they are not the same, according to him, as the species DeLong described as *S. luteolus*, but are the same as the species DeLong and Mohr described as *S. baculus*.

*Scaphoideus luteolus* Van D. is widespread throughout the region where phloem necrosis occurs, having been taken
in surveys from Ohio on the east to Kansas on the west and to Jackson, Mississippi, on the south. That this species occurs in regions not yet known to harbor the virus is demonstrated by Oman's statement accompanying the report. In this report he states that he has seen specimens from the following states outside the disease area; New Jersey, New York, Pennsylvania, Maryland, Virginia, Georgia, and Alabama.

In July 1947, an extensive series of duplicate tests were begun to check the results of the 1946 tests. A total of 119 two-year-old elm seedlings, grown from seed in insect proof cages, were used. To serve as checks on the results, hundreds of seedlings, grown under the same conditions, were not exposed to insects. Other checks were furnished by several hundred seedlings in other cages that had been used in tests with other insect species.

The leaf hoppers used in these tests were collected almost entirely as nymphs, the only stage in which the species can be collected easily and in sufficient numbers for testing. The nymphs were exposed to the virus by confining them on diseased elm foliage, in cheese-cloth sleeves, for periods ranging from 5 to 13 days. After this feeding period, they were divided into groups of 25 insects, each group being placed on a potted, healthy, two-year-old elm seedling. After feeding on these seedlings for a few
days, the survivors were removed and placed on a second series of healthy seedlings and permitted to feed. Further transfers were made to additional seedlings at various intervals, depending upon the survival of the insects. Therefore each lot of insects fed on at least two healthy seedlings in succession, many on three seedlings, a few on four, and at least two on five.

After the cloth sleeves and the insects were taken off the seedlings, they were placed in a cloth-covered cage, where further precautions were taken against contamination by other insects by spraying the cage and the seedlings with DDT. Before the beginning of the 1948 growing season, the seedlings were removed from the pots and planted in the ground inside the shelter cage.

Since it was possible for some of the seedlings to have become diseased in the fall of 1947 and to have died before the date when foliar symptoms normally appear, they were examined in May, 1948. At that time several seedlings had stunted foliage, indicating a diseased condition. Some of these seedlings were dug up to allow a thorough examination of the fibrous roots and the phloem in the basal portion of the stems for other symptoms. Many of the smaller were found to be dead, the inner phloem surface was butterscotch in color, and emitted a distinct odor of wintergreen-positive evidence of phloem necrosis.
The other seedlings with stunted foliage were left in the ground to await further development of the symptoms. Just before these seedlings were completely dead, they were dug up and the living portions of the phloem examined. All had unmistakeable symptoms of phloem necrosis. By late July still others had developed symptoms, confirming the presence of the disease.

Altogether a total of 22, or 18.6%, of the 1947 test trees developed symptoms and died in 1948. Examination of the hundreds of check seedlings, and the hundreds of seedlings under test with other insect species, failed to disclose the presence of a single additional diseased tree.

These tests were not designed to show the length of the incubation, or latent, period of the virus in the insect that seemed necessary before transmission. Evidence was obtained, however, which indicated that a period of several days must elapse after insects have fed on diseased foliage before they can transmit the virus. For instance, when insects were allowed to feed on a series of healthy trees for 20 days, after feeding on diseased foliage, only 5.5% of the healthy trees developed disease symptoms. In a second series of trees to which these insects were transferred after an incubation period of 20 days, and allowed to feed for another 30 days, a total of 34% of the healthy trees became diseased.
Additional research is necessary to show whether the lower percentage of transmission in the first series was due to a latent period of the virus in the insect or to a shorter period of feeding.
C. Life History, Habits, and Distribution

of the Insect Vector

Beyond its description, a few distribution records, and occasional references to host plants, the literature contains practically nothing about *Scaphoideus luteolus* Van Duzee. It had not been known previously to be economically important and does not show up abundantly in insect collections made at random. Where elm trees are swept, adults are seldom found in large numbers, even when search is made for them in stands harboring heavy nymphal populations. This may be accounted for, largely, by the habit of the adults of frequenting the inner part of the crown until well along in the season. Late in the summer, although the adults may disperse to other parts of the crown, it is still difficult to collect many of them because so much of the foliage is beyond the collector's reach.

*Scaphoideus luteolus* Van D. belongs to the *Athyssaninae*, a subfamily of the leaf hoppers. In the adult stage it so closely resembles certain other species in the genus that it can be separated from them only by a study of the structural differences in the internal genitalia of the males. DeLong and Mohr (1936) described this species as *S. baculus*, but Oman, of the Division of Insect Identification, reported this to be *S. luteolus*. 
Although adults are difficult to separate from related species, the opposite is true of nymphs. After the second instar practically all nymphs are dark brown with a transverse white dorsal band just behind the thorax, covering the first two and part of the third abdominal segments. Specimens are rarely encountered where this band is obscure or absent. The value of this nymphal characteristic in identifying the species is indicated by the fact that in all the studies of insects associated with the elm tree in Ohio and other widely scattered sections of the Mid-West undertaken in recent years, no nymphs of another leaf hopper species closely resembling Scaphoideus luteolus have been encountered on elm trees.

Prior to 1948, apparently very little was known about the host plant preferences of this species. Osborn (1923) reported collecting it from elm trees, but DeLong (1948), in discussing it as S. baculus, stated that its food plant was unknown.

In surveys of elm-inhabiting insects, conducted in the course of the experiments to find the vector of phloem necrosis, it was soon discovered that the species could be collected consistently from elm trees but seldom from associated vegetation. As early as 1941 it was discovered that the nymphal stage was to be found only on the elm. Subsequent observations have failed to disclose the presence
of nymphs on any other plant.

Although surveys of elm-inhabiting insects have disclosed the presence of at least 150 species of sucking insects on elm trees at one time or another, they have also shown that very few of these are as abundant on the elm as on associated plant species. The records suggest that, with the possible exception of certain insects in the genus Erythromera, Scaphoideus luteolus comes closer than any other leaf hopper, in the Mid-West, to being confined entirely to the elm.

Since Scaphoideus luteolus was found to transmit the virus of elm phloem necrosis, its life cycle has been tentatively worked out. Insect eggs, found in the cork parenchyma of elm bark, were allowed to hatch out on moist blotting paper in culture dishes in the laboratory. Many of the nymphs hatching from these eggs were reared into the later instars and were found to be of this species.

The species overwinters in the egg stage, hatching, in Ohio, early in the spring soon after the elm foliage first appears. In 1948, the first nymphs were seen on April 26 at Kansas City, Missouri. At Columbus, Ohio, nymphs were first observed on May 11, two weeks later. Field observations indicate that hatching may occur over a period of several weeks, for it was not uncommon to find first-, second-, and third-instar nymphs feeding together on a single
elm leaf. Even under controlled laboratory conditions it took several weeks for all the eggs in infested bark to hatch.

In the laboratory five nymphs, hatched from eggs, were reared to the adult stage, four males and one female. Two males took 36 days to reach the adult stage, while the other two needed 37 days. The female required 42 days to complete nymphal development. Both sexes had five nymphal instars.

Under field conditions it may take longer for the first nymphs to reach the adult stage. Although hatching began as early as May 11, 1948, in the Columbus area, the first adult was not seen until June 29, 49 days later. From that date to August 25 both nymphs and adults were present, though nymphs became scarce by the end of July. Just how long adults live was not accurately determined. Adults, in a sleeve over elm foliage, lived for 68 days until they and the foliage were killed by the first severe frost on October 15. Data are not available on the length of either the preoviposition period or the egg laying season.

Since the eggs are laid in roughened bark, the newly hatched nymphs are forced to wander in search of foliage on which to feed. Therefore, the foliage nearest the trunk or limbs becomes infested first, the nymphs being most abundant on the leaves of branchlets growing from the
trunk. It is common to find a dozen or more newly hatched nymphs on the undersurface of one of these leaves. Observations of feeding habits indicate that the species feeds mainly on the midribs and larger leaf veins on the undersurfaces of leaves.

As the nymphs continue to grow they tend to disperse, though, until the end of this stage, they are most abundant on the foliage of these trunk branchlets. Not only may nymphs be found most easily on this foliage but, in the early days of the adult stage, adults may be collected easily from the stems and twigs or the branchlets by using an aspirator. In fact, much larger numbers were collected from these branchlets by this method in late July and early August than by sweeping the outer foliage of the same trees with a net. After the middle of August, the reverse was true, indicating that most of the adults had dispersed.

Scaphoideus luteolus Van D. is widely distributed in the eastern states. In surveys, it has been collected in New Jersey, Pennsylvania, Ohio, West Virginia, Kentucky, Tennessee, Mississippi, Kansas, Nebraska, Iowa, Missouri, Illinois, Wisconsin, and Indiana. Medler (1942), though referring to it as S. baculus, reported it from Minnesota. Oman reported having seen specimens from New York, Maryland, Virginia, Alabama, and Georgia. Thus it is known to be present not only as far west and south as phloem necrosis
is known to occur, but in several states east and north of the known limits of the disease area.
D. Distribution of Phloem Necrosis

Tehon (1945) was inclined to believe that the epidemic dying of elms reported by H. Garman in 1893 and 1899 and by S. A. Forbes in 1912 were caused by phloem necrosis. He suggested that the evidence indicated that phloem necrosis might be a native disease.

Phloem necrosis of elm was reported at Ironton, Ohio in 1918, at Dayton, Ohio in 1927, and at Chillicothe, Ohio in 1935. Leach and Valleau (1939) reported epidemics taking place in southwestern West Virginia and near Lexington, Kentucky. Bretz (1944) reported that the disease was spreading in Missouri, Kentucky, Ohio, Indiana, and Illinois. Tidd (1944) listed the areas in Indiana where phloem necrosis had been found. Slagg (1944) reported that phloem necrosis was first seen in Wyandotte County, Kansas, on September 19 of that year. Caldwell (1945) called phloem necrosis an old disease in Indiana. He stated that the recent rapid spread of the disease in the southern half of the state was alarming. In Indianapolis phloem necrosis killed 4,000 elms in 1944.

Carter (1945) stated that, in Illinois, the disease was confined to the southern two-thirds of the state. He believed that the disease was spreading rapidly for only 188 affected trees were observed from 1939 to 1943, while 1653 affected trees were observed in 1944. He conceded that this
may have resulted partially from limited observations. Beilman (1945) stated that phloem necrosis could be found in many small towns and cities in Missouri. He estimated that at least 250,000 elms are being killed by phloem necrosis each year in the area from Columbus, Ohio to Kansas City, Missouri and from Chicago to central Kentucky.

In all, 13 states are in the affected area. They are Indiana, Illinois, Kentucky, Ohio, Missouri, Tennessee, West Virginia, Mississippi, Iowa, Nebraska, Kansas, Oklahoma, and Arkansas.
E. Control

Bretz (1944) and Harris (1945) estimated that 20,000 elm trees were killed in Dayton, Ohio. Harris stated that the estimated cost of removal of these dead trees was $1,500,000. She also stated that the Columbus authorities expected to lose 10,000 trees, whose removal cost was estimated at $750,000.

Attempts to control the disease before the discovery of the insect vector seemed futile. Leach (1939) stated that there was no known effective means of combatting the disease. Tilford (1942), Swingle (1942), and Beilman (1945) concurred in this. However, Valleau (1939), Swingle (1942), Bretz (1944), and Beilman (1945) suggested that seedlings from old trees that have passed through epidemics and are resistant to, or apparently immune to, phloem necrosis may offer a means of replacement.

Harris (1945) stated that a few seedlings, from old trees which had survived epidemics, had been taken to the laboratories at Columbus, Ohio, where phloem necrosis infected parts were grafted onto them. These seedlings resisted the disease and remained healthy. Now 13,000 of these seedlings are undergoing tests at the Forest Pathology Field Office of the U. S. Department of Agriculture at Columbus, Ohio.
Whitten and Baker (1948) reported that DDT was used in two experiments on control in 1944. In the first experiment, groups of living elms were sprayed with a DDT formula, at Columbus, Ohio and Kansas City, Missouri, to reduce the loss. There has been no further report on this experiment. The second experiment was to test the residual toxicity of DDT formulas. Insects were found to be very susceptible, especially to emulsions.

Swingle, Whitten, and Young (1949) stated that the prevention of the spread of phloem necrosis depended on preventing the insect vectors from feeding on the elm trees. This might be accomplished with a spray containing DDT, provided that the sprays are correctly formulated, properly applied, and used in sufficient quantities at the right time. They gave three formulas; A and B to be used with hydraulic sprayers, and C to be used with mist blowers.

Formula A

16 lbs. of technical DDT dissolved in a mixture of 2.25 gallons of benzene and 1 gallon of Velsicol AR-50. To this solution add 1 pint of Triton X-100. Dilute with water to make 100 gallons.

Formula B

16 lbs. of technical DDT dissolved in 4 gallons of xylene. To this add 1 pint of Triton X-100. Dilute with water to make 100 gallons.
Formula C

20 lbs. of technical DDT dissolved in a mixture of
5 gallons of xylene and 2.5 gallons of Acme white oil.
To this solution add 1.5 pints of Triton X-100. Dilute
with water to make 20 gallons.

Directions for applying
1. Apply the spray before the elm leaves or flowers appear.
2. Dilute solution to half strength. Apply 2 1/2 to 3
   months after the first treatment.
3. Repeat #2 after 2 1/2 to 3 months.
Bibliography

Baker, W. L.
1948. Transmission by leaf hoppers of the virus causing phloem necrosis of American elm.
Science 108:307-308

Baker, W. L.
1949. Studies on the transmission of the virus causing phloem necrosis of American elm, with notes on the biology of its insect vector.
Jour. Econ. Ent., 42:729-732

Bawden, F. C.

Beilman, A. P.
1945. Phloem necrosis--a serious threat to the elm.

Bennett, C. W.
1940. Acquisition and transmission of viruses by dodder (Cuscuta subinclusa)
Phytopath., 30:3

Bennett, C. W.
1940. The relation of viruses to plant tissues.
Bretz, T. W.
1944. Phloem necrosis of elms in Missouri.

Bretz, T. W. and Swingle, R. U.
1946. Known distribution of phloem necrosis of the American elm.
U.S. Dept. of Agr., Plant Disease Reporter 30:156-159

Caldwell, R. M.
American Nurseryman 81:27

Carter, J. C.

Cook, M. T.
1946. Plant viruses and plant diseases.
Mimeo. by Dept. of Bot., Louisiana State Univ. 190 p.

Harris, K.
1945. A lost generation of elms.
Nature Magazine 28:15-16, 50

Herrick, J. A.
Men's Gard. Clubs Am. Yearbook, 1949:11-12, 90
Kunkel, L. O.
1943. New hosts as a key to progress in plant virus
disease research.
Viruses diseases. Cornell Univ. Press 63-82

Kunkel, L. O.
19 p.

Leach, J. G. and Valleau, W. D.
1939. Two reports on phloem necrosis of elm.
U.S. Dept. of Agr., Plant Disease Reporter 23:300-301

McLean, D. M.
1944. An experimental and histological study of phloem
necrosis, a virus disease of American elm.
Abstracts of Doctoral Dissertations,
Ohio State Univ. 43:93-98

McLean, D. M.
1944. Histo-pathologic changes in the phloem of American
elm affected with the virus causing phloem necrosis.
Phytopath. 34:818-826

Slagg, C. M.
1944. Phloem necrosis found on elm in Kansas.
U.S. Dept. of Agr., Plant Disease Reporter 28:1053

Smith, K. M.
1946. Plant viruses.
Smith, K. M.
1951. Recent advances in the study of plant viruses.

Swingle, R. U.
1938. A phloem necrosis of elm.
Phytopath. 28:757-759

Swingle, R. U.
1940. Phloem necrosis in the Ohio River Valley.
Phytopath. 30:13

Swingle, R. U.
1942. Phloem necrosis, a virus disease of the American elm.
U.S. Dept. of Agr., Cir. 640:8 p.

Swingle, R. U., Whitten, R. R., and Young, H. C.
1949. The identification and control of elm phloem necrosis and Dutch elm disease.

Tehon, L. R.
1945. American elms die by tens of thousands from phloem necrosis disease epidemic.
The Greenkeeper's Reporter 13:17-19

Tilford, P. E.
1942. Phloem necrosis and elm mosaic.
National Shade Tree Conf. Proc. 16:163-166
Tidd, J. S.
1944. Dying of elms in Indiana.

Whitten, R. R. and Baker, W. L.
1948. Recent experimental results on control of vectors of two elm diseases.
Arborist's News 12:41-42
Abstract

In spite of the rapid development of the study of plant viruses and virus diseases there is still no generally accepted definition of a virus.

Cook (1946) divided the history of plant virus study into three periods. The first dated from 1576, with the first published description of a virus disease, the breaking of tulips by Carolus Clusius and ended in 1868 with a description of the variegation of *Abutilon striatum* Dicks. The second began in 1882 with Mayer's work on tobacco mosaic. The third started in 1906 when the study of plant viruses was really beginning.

Smith (1948) suggested adding a fourth period commencing in 1935 with Stanley's crystallization of the tobacco mosaic virus as a definite entity. Since then the physicist, the biochemist, and the serologist have joined in the research.

Estimates of losses due to plant virus diseases are difficult to make because of varying conditions.

Bawden (1950) stated that the one thing common to all plant viruses is that they are nucleoproteins containing a ribose nucleic acid.

Much confusion has been caused by the absence of any systematic basis for the nomenclature of viruses. Often the same virus has been given different names, or the same name has been given to different viruses by different workers, due
largely to naming viruses on the basis of symptoms.

In certain host plants some viruses are localized in the inoculated leaf, causing necrotic rings or spots usually termed local lesions. This reaction has been used, like Koch's plate method with bacterial cultures, in the quantitative study of viruses.

Bennett (1940) divided virus movement in plant tissues into three main relationships restricted to the phloem and the parenchyma. Restriction to parenchyma, to the phloem, and to both the parenchyma and phloem are the three divisions, with Smith (1948) suggesting the addition of a fourth division, that of restriction to the xylem.

There seems to be little doubt among investigators that plant viruses share with animal viruses the power to mutate. Bawden (1950) stated that over 50 strains of tobacco mosaic virus have already been recognized.

Plants may show a natural immunity to a particular virus, but the usual type of immunity is induced and occurs only between strains and closely related viruses.

Serological reactions have been used to differentiate between unrelated viruses that cause the same symptoms on their host plants. Though successful with only about 15 viruses, serological methods have been used to separate the different strains of these viruses.

Natural methods of virus transmission are by contact, by seed, through the soil, by all methods of vegetative pro-
pagation, and by insect vectors, the last being most important.

Smith (1948) divided transmission between insect vectors and particular groups of viruses into three types, non-persistent, persistent, and mechanical.

In attempting to explain the latent period needed by persistent virus vectors, Black (1940) and Kunkel (1937) favored the theory of virus multiplication in the insect. Smith (1948) and Bawden (1950) favored virus storage as an equally good interpretation of experimental results. Recently Black has shown that one infected insect had transmitted clover club leaf virus through 20 successive generations without access to a fresh source of the virus. Bawden considered this as good evidence in favor of multiplication.

The control or eradication of plant diseases is the final aim of all plant disease studies. Heat has been used to cure some virus diseases but does not seem economically applicable. Variations in normal cultural practices offer some hope. Doubling the number of plants per unit area, other factors being equal, could be expected to halve the proportion of the crop that becomes infected.

Plants tolerant to viruses have proven economically profitable, but provide the main sources of virus perpetuation, becoming a potential danger to other crops. Resistance to infection and extreme sensitivity are better features to breed for in new varieties, since they tend to reduce the rate of spread and the number of infected plants.
Elimination of insect vectors seems to be the best and
most obvious method of controlling insect borne virus diseases.

Phloem necrosis of elm first attracted attention at
Ironton, Ohio in 1918, at Dayton, Ohio in 1927, and at
Chillicothe, Ohio in 1935. H. Garman (1896, 1899) and S. A.
Forbes reported similar epidemics of dying elms in Kentucky
and Illinois.

Foliar symptoms, though a warning sign, cannot be con-
sidered as specific for they are similar to those of the
Dutch elm disease, drought, girdling, and some nutrient
deficiencies.

The first root symptom is the dying of the small
fibrous roots. A characteristic discoloration, starting as
yellow but soon turning to a typical butterscotch color, de-
velops in the phloem tissue of the larger roots and the lower
part of the trunk before death. A second specific symptom is
a faint, but distinct, wintergreen odor that can be detected
from a freshly cut piece of diseased tissue.

Swingle (1938) reported that the American elm, Ulmus
americana L., and its varieties, the vase and moline elms, are
very susceptible. McLean (1944) reported that the winged elm,
Ulmus alata Michx., may also be susceptible.

Experiments have shown that the leaf hopper, Scaphoideus
luteolus Van D., is the insect vector of phloem necrosis. The
adults closely resemble the adults of other species, but the
nymphs are easy to distinguish due to a transverse, white,
dorsal band on the first two and part of the third abdominal segments. The nymphs have been found only on elm trees.

The species overwinters in the egg stage, hatching early in the spring soon after the elm foliage appears. Complete nymphal development requires 36 to 42 days in the laboratory and may take longer in the field. The species feeds on the underside of the leaves, being most abundant on foliage close to the trunk.

*Scaphoideus luteolus* Van D. has been collected in surveys in New Jersey, Pennsylvania, Ohio, West Virginia, Kentucky, Tennessee, Mississippi, Kansas, Nebraska, Iowa, Missouri, Illinois, Wisconsin, Indiana, Minnesota, New York, Maryland, Virginia, Alabama, and Georgia.

Phloem necrosis of elm has been reported from Kentucky, Illinois, Ohio, West Virginia, Indiana, Missouri, Kansas, Tennessee, Mississippi, Arkansas, Oklahoma, Iowa, and Nebraska.

Attempts to control the disease seemed futile before the discovery of the vector. Harris (1945) stated that seedlings from old trees which had survived epidemics were taken to laboratories at Columbus, Ohio where, under tests, they proved resistant to phloem necrosis. Now 13,000 of these seedlings are being tested as possible replacements for trees killed by phloem necrosis.

Spraying was considered useless at first but Swingle, Whitten, and Young (1949) stated that prevention of the spread of phloem necrosis depended on preventing the insect vectors
from feeding on elm trees. This can be accomplished by sprays containing DDT, provided that they are correctly formulated, properly applied, and used in sufficient quantity at the right times.