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**VIRUS DISEASES
AND NONINFECTIOUS
DISORDERS
OF STONE FRUITS
IN NORTH AMERICA**

Agriculture Handbook No. 437

Agricultural Research Service
UNITED STATES DEPARTMENT OF AGRICULTURE

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AND NONINFECTIOUS
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This handbook supersedes Agriculture Handbook 10, Virus Diseases and Other Disorders with Viruslike Symptoms of Stone Fruits in North America.

Agricultural Research Service
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FOREWORD

The study of fruit tree virus diseases is a tedious process because of the time needed to produce experimental woody plants and, often, the long interval from inoculation until the development of diagnostic symptoms. The need for cooperation and interchange of information among investigators of these diseases has been apparent for a long time. As early as 1941, a conference was called by Director V. R. Gardner at Michigan State University to discuss the problem. One result of this early conference was the selection of a committee (E. M. Hildebrand, G. H. Berkeley, and D. Cation) to collect and classify both published and unpublished data on the nomenclature, symptoms, host range, geographical distribution, and other pertinent information on stone fruit virus diseases. This information was used to prepare a "Handbook of Stone Fruit Virus Diseases in North America," which was published in 1942 as a miscellaneous publication of the Michigan Agricultural Experiment Station.

At a second conference of stone fruit virus disease workers held in Cleveland, Ohio, in 1944 under the chairmanship of Director Gardner, a Publication Committee (D. Cation, G. H. Berkeley, L. C. Cochran, F. P. Cullinan, and R. J. Haskell) was appointed to revise the text of the first handbook. This revision resulted in the publication in 1951 of Agriculture Handbook 10, "Virus Diseases and Other Disorders with Viruslike Symptoms of Stone Fruits in North America." The work of this committee was helped greatly by the passage of the Research and Marketing Act of 1946 and the subsequent establishment and funding of regional research projects to investigate stone fruit virus diseases. Research and Marketing Act funds were used to publish Handbook 10.

State workers in the several regions cooperated through an informal Inter-regional Research Committee that functioned actively, and helped to establish the formal IR-2 Committee and project. The regional projects, with the active cooperation of the United States Department of Agriculture and the Canadian Department of Agriculture, have reduced duplication of research efforts and improved dissemination of information to the public. The formation of the European Committee for Cooperation in Fruit Tree Virus Research has aided the cooperative effort in North America through exchanges of information and personnel and has helped to establish international standards for the detection and control of fruit tree virus diseases.

Since the publication of Handbook 10, several pathologists, who were prominent authors in that book, have retired from active teaching or research. Because so much of our present knowledge about stone fruit virus diseases was obtained from their pioneering efforts, we wish to acknowledge their influence on this discipline and acknowledge our debt to the guidance they provided in the original edition from which we have drawn freely in this new handbook. We particularly want to thank G. H. Berkeley (deceased), E. M. Hildebrand, Lee

M. Hutchins, G. W. Keitt (deceased), T. B. Lott, John A. Milbrath, D. H. Palmiter (deceased), T. E. Rawlins (deceased), E. L. Reeves, B. L. Richards, and R. S. Willison.

Illustrations in this book were taken from Agriculture Handbook 10, "Virus Diseases and Other Disorders With Viruslike Symptoms of Stone Fruits in North America," or were submitted by the authors of the various sections. Credit is gratefully extended to the following contributors of illustrations for sections of which they are not authors: American Potash Institute, figure 152; Nels R. Benson, figures 145, 146, and 147; and A. E. Hitchcock, figures 142, 143, and 144.

This handbook is the result of the cooperative effort of the authors, who represent all the fruit-growing regions of North America. The Editorial and Publication Committee is deeply indebted to them for their willingness to participate in the preparation of this revision. Special credit is due L. C. Cochran, retired, former chief, Fruit and Nut Crops Research Branch, Plant Science Research Division, ARS, for his help in initiating the production of this book. We want to recognize J. R. McGrew, plant pathologist, USDA, for his technical advice and review of the manuscript and his efforts in collecting of the illustrations.

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[Thomas Sheffield Pine, who served as chairman of the Editorial and Publication Committee for this book, died April 2, 1970, after an illness of several months. The other members of the Editorial and Publication Committee wish to give special recognition to his dedication, organizational ability, and hard work in assembling the manuscripts, unifying and verifying literature citations, and mounting the illustrations. He completed the work on schedule under extremely difficult conditions and failing health.]

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INTRODUCTION

Much new information has been obtained about stone fruit viruses and the diseases they incite since the publication of Agriculture Handbook 10 in 1951. The purpose of this new publication is to provide professional investigators and students with an updated reference work and agricultural technicians and orchardists with an illustrated handbook to help in diagnosing virus diseases in the field. Therefore, this book contains sections on the basic and applied approaches to research on stone fruit virus diseases as well as extensive descriptive material on the symptoms, distribution, control, and economic factors pertaining to these diseases.

Progress in our knowledge of fruit tree virus diseases has not simplified the role of the scientist in explaining them or the field adviser in diagnosing them. Only five virus diseases of stone fruit trees were recognized in 1930; 48 were known in 1951. This publication describes 95 virus diseases. Although this may appear to indicate a phenomenal rise in the number of virus diseases in North American orchards, the figures are somewhat misleading. Many diseases occur in very limited areas and are not economically important. They are described briefly in this book.

It is now known that several diseases, previously described under different names or from different areas of the continent, are caused by viruses so similar to each other that they justify being grouped as strains. Thus, some diseases mentioned individually in Agriculture Handbook 10 will be found under different headings in this book. Diseases thought to be virus-induced, but for which no virus has yet been isolated and purified, have been retained as virus diseases; however, this does not preclude the possibility that future research will show they are caused by mycoplasmas or still unidentified pathogenic agents. Plant virology is a dynamic discipline, and reevaluation of present knowledge should be expected.

The diagnosis of virus diseases in orchard trees often is confused by the presence of many nontransmissible disorders with symptoms resembling those caused by viruses. These disorders include genetic abnormalities, nutritional deficiencies or excesses, injuries caused by the improper use of pesticides and herbicides, and injuries caused by air pollutants. Because these disorders must be considered when attempting to diagnose virus disease problems in the field, chapters on these subjects are included in this handbook.

The ultimate objective of all research on stone fruit virus diseases is the control of these diseases in the orchard. Three sections of this handbook deal directly with problems of control, ranging from fundamental orchard house-keeping to Federal quarantines against the chance importation of foreign viruses. Today it is possible for orchardists to plant fruit trees that are free from all known viruses, but preventing virus infection in the orchard is still a major problem. The insect or mite vectors of only a few fruit tree viruses have been

identified. At least two viruses are transmitted from diseased to healthy trees in pollen. Nematodes are known vectors of viruses but have been implicated in the spread of very few stone fruit diseases. Native herbaceous and woody plants that may act as reservoirs of virus for spread to fruit trees have been identified for relatively few diseases. For many tree fruit virus diseases the only known means of spread is in infected propagating materials. Therefore, despite progress in our ability to identify viruses and to produce virus-free trees, much work remains to be done on disease control in the orchard.

Virus diseases of stone fruit trees are generally referred to by their common names. Terminology used in the study of these diseases varies widely, but the text adheres as closely as possible to the terms suggested by Welsh (714).¹

¹Italic numbers in parentheses refer to Literature Cited, p. 365.

TECHNIQUES OF INVESTIGATING STONE FRUIT VIRUSES

ROBERT W. FULTON

The association of a causal agent with a disease is fundamental in pathology. The mere study of symptoms in a diseased plant is inadequate to demonstrate the cause of the disease or its relationship to other diseases. The requirements outlined in the classical Koch's postulates are just as necessary for virus diseases as for those caused by fungi and bacteria.

Progress in research on viruses infecting woody plants has lagged behind the general development of plant virology. Stone fruits, like other rosaceous plants, are difficult to inoculate and are poor inoculum sources. Most stone fruit viruses are unstable. Usually they are present in low concentration.

The difficulties of inoculating and transmitting some stone fruit viruses have been overcome, and their properties, morphology, and serology have been investigated. This has provided the opportunity to associate causal agents with diseases by a sequence similar to that outlined in Koch's postulates.

In this section we will concern ourselves only with mechanical transmission of stone fruit viruses, not their transmission by insect or nematode vectors, dodder, or pollen. Information pertaining to viruses not occurring in North America will usually not be included. Many potentially useful techniques have been applied to viruses similar to those in stone fruits, but a discussion of these is beyond the scope of this section.

Mechanical Transmission

Source of inoculum. — Transmission of necrotic ring spot virus from cherry to cucumber (473) was first accomplished by grinding in buffer young expanding leaves showing shock symptoms and applying the homogenate to carborundum-dusted cucumber cotyledons. Immature leaves are usually a good source of inoculum even when the virus is latent. Older leaves, or any leaves after terminal growth has ceased, usually make weakly infective inoculum, and often virus cannot be transmitted from them.

Although young leaves with recently developed symptoms usually provide choice inoculum, other tissues are sometimes effective. Petals may provide infective inoculum of some viruses difficult to transmit from leaves (251, 432, 446, 668). Viruses also have been transmitted by mechanical inoculation from pollen, inner bark, buds, fruit, seeds, and roots (16, 172, 196, 725, 758).

The species of *Prunus* host may have a marked effect on the infectivity of extracts prepared from it. *P. tomentosa* is difficult to transmit virus from by mechanical inoculation (252); *P. pensylvanica*, *P. mahaleb*, and *P. persica* are

much better sources of most viruses. Plum line pattern virus, for example, was much more readily transmitted from *P. mahaleb* than from *P. domestica* (512). Transmission of virus by grafting from an unsatisfactory host to a host amenable to mechanical transmission is a relatively unexplored field. Viruses may sometimes be transmitted by approach grafting between woody and herbaceous hosts (172, 234, 494).

Preparation of inoculum. — Inoculum is usually prepared by grinding infected tissue in phosphate buffer. An optimum of about pH 8 was found for infectivity of a number of stone fruit viruses (226). The optimum concentration of buffer for infectivity, however, is around 0.03 M, which is not likely to maintain the pH unless the proportion of tissue to buffer is small. Many viruses infecting stone fruit are inactivated rapidly by oxidized materials in plant sap. Thus, the inclusion in extracts of an antioxidant helps prevent rapid loss of infectivity. The most useful antioxidant is probably 2-mercaptoethanol (2-ME) at about 0.02M (232, 512). Sodium diethyldithiocarbamate (DIECA) inhibits polyphenol oxidase and thereby stabilizes some viruses (271). Unfortunately, it is itself an inactivator of certain viruses (22) and should not be used in inoculum indiscriminately.

Various compounds adsorbing or reacting with tannins or other virus-inactivating substances have been used to increase infection. Alkaloids form insoluble complexes with tannins; nicotine base (2.5 percent) was included in inoculum of plum line pattern virus (372) in transmitting it from plum to herbaceous plants. Similarly, caffeine has been beneficial in the transmission of necrotic ring spot virus (190). Alumina also adsorbs tannins and has been used as an aid in mechanical transmission (102). Although these additives may increase infectivity, none has been essential; some transmission can usually be obtained without them.

Application of inoculum. — Inoculum may be efficiently applied to abrasive-dusted leaves with a gauze pad, forefinger, glass spatula, or brush. Washing inoculated leaves with water is usually detrimental to infection (226), although plant extracts left on the abraded leaf surface may be toxic. Wiping inoculum from the leaves immediately with the pad used to inoculate, or fresh gauze, so that the inoculated surface dries quickly, results in good infection with a minimum of injury (753).

Application of inoculum with an artist's spray gun has been described as more efficient than wiping (402). An air pressure of 60 pounds, with the nozzle held 1 cm. from the surface being inoculated, was recommended. An inoculum delivery rate of 10 ml. per minute was optimal. Carborundum (1 percent, w/v) was incorporated in the inoculum.

Inoculations also may be made rapidly by rubbing the freshly cut surface of a stack of disks, or a roll, of infected tissue over the surface of carborundum-dusted, buffer-sprayed leaves (754). This method is used to overcome difficulties arising from oxidation in extracts.

Characterizing and Identifying Stone Fruit Viruses

Once a stone fruit virus has been transmitted to an herbaceous host, it can be characterized by methods conventional for mechanically transmitted viruses. Determining the host range is a useful first step. The host range can be compared with host ranges, including diagnostic and differential hosts, that have been described for other viruses. Another purpose in investigating host ranges is to find hosts suitable for producing virus for purification and to find a host suitable for infectivity assays.

When the host range and symptoms of an unknown isolate match those of a known isolate, the identity of the two usually can be presumed. Unfortunately, strains of a virus often vary in host range as well as in symptoms caused, so that host range differences need not indicate that the viruses are unrelated. For prunus ring spot and prune dwarf viruses, some hosts consistently differentiate a wide range of strains of each virus, whereas other hosts do not (706).

The use of differential hosts to identify stone fruit viruses is subject to the same difficulties as the use of host ranges; strains may vary in the symptoms they produce. On some hosts, however, symptoms are relatively consistent for many isolates. *Prunus* ring spot and prune dwarf virus strains, for example, produce typical reactions on *Sesbania exaltata*, *Momordica balsamina*, *Cyamopsis tetragonaloba*, and *Cucurbita maxima* cv. Buttercup (706).

Host ranges and symptom type are also useful in separating mixtures of viruses. Inoculation of a mixture to a host which is insusceptible to or which localizes one of the viruses makes possible the isolation of the other virus. This can be done with *Prunus* inoculum provided it is sufficiently infective. Some differential hosts are not particularly easy to infect, however, and may be infected by inoculum from cucumber more readily than by inoculum from *Prunus*. Table 1 lists some hosts reported as being diagnostic or differential for stone fruit viruses.

Properties. — As with other mechanically transmitted viruses, the thermal inactivation point, resistance to aging in vitro, and dilution end point vary among the stone fruit viruses (table 2). The dilution end point of most of the stone fruit viruses is highly variable and depends to a great extent on the choice of host supplying the extract and the length of the incubation period. Cucumber cotyledons, for example, provide more infective extracts 4 or 5 days after inoculation with necrotic ring spot virus or prune dwarf virus than after 7 or 8 days (228). The dilution end point, therefore, is not particularly useful in the differentiation of stone fruit viruses.

Several stone fruit viruses lose infectivity very rapidly in crude sap; a loss of 95 percent of the infectivity within 2 minutes is common (226). This lability will differentiate prune dwarf, *Prunus* ring spot, plum line pattern, and apple chlorotic leaf spot viruses from the somewhat more stable peach yellow bud mosaic, tomato bushy stunt, and cucumber mosaic viruses. Aging in vitro,

TABLE 1.—*Reactions of some diagnostic herbaceous hosts to certain viruses found in Prunus*¹

Host	Virus					
	PRSV ²	PDV	PLV	YBMV	CMV	CLSV
<i>Chenopodium amaranticolor</i>	S-Cl ³	—	+ or —	Ll	NLl	LSC
<i>Cucurbita maxima</i>	N or Cl	S-C	—	S-Sp	S-M	
<i>Cyamopsis tetragonaloba</i>	S-N	— or Ll	—			
<i>Gomphrena globosa</i>	S-NSp	— or LNSp	—			
<i>Momordica balsamina</i>	NLl	SM	CLL		S-M	
<i>Phaseolus vulgaris</i>	— or SM	—	LNl	Ll-SM	Ll	Ll
<i>Ricinus communis</i>	± or —	SM	—	+	—	
<i>Sesbania exaltata</i>	+H	NLl	NLl		—	
<i>Tithonia speciosa</i>	—	— or SC	—			
<i>Physalis floridana</i>	—	—	S-CM		SM	

¹ Adapted from 225, 374, 512, 706.

² PRSV, Prunus ring spot virus; PDV, prune dwarf virus; PLV, plum line pattern virus; YBMV, yellow bud mosaic virus; CMV, cucumber mosaic virus; CLSV, apple chlorotic leaf spot virus.

³ S, systemic; C, chlorotic; L, local; l, lesion; H, no symptoms; N, necrotic; M, mottling; Sp, spotting; +, infection; —, no infection.

however, does not provide a criterion for differentiation among the very labile viruses.

As conventionally determined, aging tests determine the last bit of infectivity remaining in an extract. The rate of infectivity loss provides more useful information and can be expressed conveniently by determining the half-life of the virus (759). This determination requires reasonably accurate quantitative assays of infectivity. In determining the infectivity loss of very unstable viruses, the extrinsic factors are probably more important than the intrinsic factors. Therefore, unless tests are carefully controlled, differences between viruses may reflect differences in the type of extract.

Thermal inactivation trials involve exposure of infectious extracts to controlled temperatures for 10 minutes. With undiluted or concentrated plant sap, this period is sufficient for most of the infectivity to disappear even at room temperature. Determination of thermal stability, therefore, requires diluted extracts in which infectivity has been stabilized by chemical additives. For practical purposes, the diluent is usually phosphate buffer containing DIECA, 2-ME, or other antioxidants known to stabilize infectivity. Such preparations contain more infective virus than nonstabilized crude extracts, and higher thermal inactivation points result. Thermal inactivation points for *Prunus* ring spot virus of 52° to 54° C. and for prune dwarf virus of 44° were reported for dilute

extracts without stabilizing chemicals (226). Tests with a wide range of isolates similarly diluted, but stabilized with antioxidants, gave end points of 55° to 62° for necrotic ring spot virus and 45° to 54° for prune dwarf virus (706). Although dilution lowers the virus concentration, data obtained from such extracts do show differences among stone fruit viruses and are probably comparable with conventional data on more stable viruses in undiluted sap.

Preservation of Virus Isolates

The maintenance of stock cultures of stone fruit viruses requires attention because an assured supply of inoculum is necessary during an investigation, and later, when comparisons with new isolates become important. Maintaining a virus in living plants is the method of choice when inoculum is needed frequently. However, this has disadvantages; plants may grow sufficiently rapidly to require frequent transfer of the virus. If they grow slowly, transmission of

TABLE 2.—*Properties of viruses isolated from Prunus*
[Italic numbers in parentheses refer to Literature Cited, p. 365]

Virus	Thermal inactivation point (° C.)	Stability	Size and shape
<i>Prunus</i> ring spot	55° to 62° (706)	Very unstable	23 m μ sphere (228)
Prune dwarf	45° to 54° (706)	. . . do	22 m μ sphere (228)
Plum line pattern	66° (512)	. . . do	30 m μ sphere
Apple chlorotic leaf spot	49° to 52° (405)	. . . do	12 by 500 to 700 m μ flexuous rod (405)
Peach yellow bud mosaic	60° (107)	Several days	25 to 28 m μ sphere (107)
Cucumber mosaic (736)	65° to 70°	. . . do	28 to 30 m μ sphere
Tomato bushy stunt (16)	80°	. . . do	30 m μ sphere
Tobacco mosaic (251)	94°	Very stable	18 by 300 m μ rigid rod
Cherry rasp leaf (171) (Arabis mosaic virus).	55° to 60°	15 to 21 days	30 m μ sphere (281)
Cherry rasp leaf (171) (Raspberry ring spot).	65° to 70°	. . . do	Do.
Cherry leaf roll (170)	55° to 60°	5 to 10 days	Do.
Sarka (350)	45° to 47°	Very unstable	20 by 764 m μ rods (350)
Peach shoot stunting (471) (tomato black ring).	58°	7 days	30 m μ sphere (281)

the virus may be difficult from old or dormant tissue. Infectious inoculum is not always available from woody hosts that undergo a cycle of growth and dormancy. Furthermore, cultures in living plants are subject to contamination with unwanted viruses. Some of these difficulties may be avoided by keeping several cultures of each virus. We keep cultures in *Prunus* hosts (*P. mabaleb* or *P. persica* whenever possible), and also, when possible, in *Vinca rosea*, which can be cut back severely from time to time. The young leaves of *V. rosea* usually provide infectious inoculum.

A better method of maintaining cultures is in the form of vacuum-dried tissue (371). In this form, the virus is not subject to contamination or change and is readily available. The tissue selected for preservation should contain a high concentration of infectious virus. Some isolates are more readily maintained in this way than others. Without previous experience with an isolate, preserved material should be tested periodically to detect decreases in infectivity that might foreshadow loss of the culture.

Transmission to the Original Host

Transmission of virus to plants of the original host species is of primary importance in identifying stone fruit viruses. No amount of correlation between symptoms and type of virus isolated will equal the direct demonstration that a characterized virus isolate will cause a specific disease when introduced into known hosts.

The susceptibility of *Prunus* to mechanical inoculation is low, unfortunately, and trees must usually be infected by budding or grafting techniques. However, young six- to eight-leaf seedlings of a number of species, particularly *P. pennsylvanica*, *P. mabaleb*, and *P. persica*, are susceptible to mechanical inoculation (227, 757). After infected seedlings reach sufficient size, virus may be transmitted by budding from them to older *Prunus* trees.

Young *Prunus* trees also may be infected by implanting infected herbaceous stem tissue beneath a bark flap. The tissue is trimmed so that cut surfaces contact those of the woody host (175, 473). This is not, however, an efficient method of transmission. A somewhat similar technique, involving approach grafts between woody and infectious herbaceous hosts, seems to be more effective with *Malus* than *Prunus* (172, 234).

Grafts made at bud break often induce symptoms within a few weeks. Symptoms result sooner from grafts made close to growing points than lower on the stem. After terminal growth has ceased, grafts usually do not induce symptoms until dormancy is broken.

Virus Purification

As with other viruses, purification of stone fruit viruses involves solving a number of problems in logical order as discussed in the following sections.

Selection of host. — The concentration of stone fruit viruses is relatively low in the tissues of many herbaceous plants, which are hardly better than *Prunus* tissues as a source of virus for purification. It is necessary not only to

select a host in which the virus reaches the highest concentration, but also to harvest infected tissue at the optimum time.

For *Prunus* ring spot and related viruses, the inoculated cotyledons of cucumber provide highly infective extracts if harvested 4 to 5 days after being heavily inoculated (228). Inoculation to cucumber cotyledons is tedious and slow, however. Because inoculation is slow, inoculum is best prepared in successive small batches that do not lose significant infectivity before the last batch is used. The inoculum must be highly infectious so that the chlorotic primary lesions are confluent on the inoculated cotyledons. Before a high degree of infection is obtained, two or more transmissions to stock plants with inoculum consisting of only chlorotic lesion tissue may be required. Heavily infected cucumber cotyledons tend to become necrotic with some virus isolates. Necrosis can be prevented or retarded by exposing inoculated plants to day lengths of 18 hours or more.

The conditions under which cucumbers are grown affect their susceptibility and the yield of virus from them. Short days are detrimental, as are long days with low light intensity. Very dark-green cotyledons with very short hypocotyls, resulting from high intensities of artificial light, are relatively insusceptible.

A number of viruses reach a higher concentration in inoculated leaf tissue than in systemically invaded tissue (A. Q. Paulson and R. W. Fulton, unpublished data). Prune dwarf virus, however, reaches a high concentration in systemically infected leaves of *Cucurbita maxima* cv. Buttercup.

Stabilization of infectivity during purification. — If the properties of a virus indicate that infectivity will decrease in extracts within an hour or two, then some method of stabilizing infectivity must be devised. These methods have usually involved antioxidants such as ascorbic acid, sodium thioglycollate, sodium sulphite, cysteine-HCl, or 2-mercaptoethanol, either alone or with an enzyme inhibitor such as sodium cyanide or DIECA. Of these, we have found 2-ME and DIECA the most effective. DIECA, however, inactivates certain viruses, and some lots of the chemical are more detrimental than others. Its use should follow controlled testing.

Because stone fruit viruses may be more stable in diluted than in undiluted extracts, tissue for purification is usually ground in buffer in which are dissolved stabilizers of infectivity. A high-speed mechanical homogenizer seems to be the most efficient way of preparing extracts. Hand grinders are too slow and are not adapted to rapid mixing of homogenized tissue and buffer.

Clarification. — Methods of removing the bulk of the particulate, nonvirus material from extracts must necessarily be mild. Low-speed centrifugation is not sufficient. Much of the particulate cellular matter is adsorbed by hydrated calcium phosphate, which is prepared by mixing 0.1 M solutions of Na_2HPO_4 and CaCl_2 and washing the precipitate thoroughly to remove all but traces of soluble salts (228). The hydrated calcium phosphate and host material sediment readily on low-speed centrifugation. Adsorption is a function of the ionic

concentration; for example, host material, but neither prune dwarf virus nor *Prunus* ring spot virus, is adsorbed from 0.03 M phosphate buffer. Higher concentrations of phosphate result in less host material being adsorbed; lower concentrations result in some virus being adsorbed also. Certain viruses are adsorbed by calcium phosphate from 0.03 M buffer so this method of clarification cannot be used indiscriminately.

Acidification to pH 4.8 to 5.0 with citric acid (503 and A. Q. Paulson and R. W. Fulton, unpublished data) is an effective clarification method. As is true with other treatments, some viruses are lost with acidification, so the method should be used only after testing.

Precipitation of host protein by adding n-butanol to a concentration of 7 to 9 percent is an effective clarification method for some viruses. The n-butanol technique has been used to clarify grape yellow vein virus (=peach yellow bud mosaic virus) (266), but is too drastic for cherry necrotic ring spot virus (R. W. Fulton, unpublished data). Clarification by emulsification with three volumes of cold chloroform has been described for necrotic ring spot virus (14). Emulsification of one volume of extract with two volumes of a 1:1 mixture of chloroform and n-butanol has been used to clarify peach yellow bud mosaic virus (711). Gel-filtration has also been used in purification (270).

Almost all purification methods for stone fruit viruses involve high-speed centrifugation of clarified extracts to concentrate the virus and eliminate low molecular weight contaminants. The times and speeds used have varied somewhat, but are typical of what is theoretically required to sediment small, spherical viruses. Once pelleted, the virus may be resuspended in buffer by gentle shaking or by simply allowing the buffer to cover the pellet overnight at low temperature. The degree of contamination of the virus pellet may be reflected in the rapidity with which it resuspends; badly contaminated virus resuspends more slowly than pure virus. After a high-speed run, there is usually some insoluble, denatured material that does not resuspend; it can be removed by a short run at low speed.

Additional steps to remove host material are usually desirable after centrifugation. Any of the following should remove practically all nonvirus antigenic material from the preparation: density gradient centrifugation (14, 266), zone electrophoresis (570), acidification (503), and precipitation of host protein with the gamma globulin portion of antihost serum (A. Q. Paulson and R. W. Fulton, unpublished data).

Serology

One of the important purposes for purifying any virus is to provide antigen suitable for antiserum production. Few published data indicate that one route of animal injection is superior to another. For a number of stone fruit viruses, intramuscular injection of virus emulsified in Freund's complete or incomplete adjuvant at rather frequent intervals (3 to 4 days) has been effective (232, 512). The amounts of virus injected have usually been, by necessity, relatively small,

probably less than 1 mg. of virus per injection. Such doses usually result in antiserum titers of 1:320 to 1:1280 after 8 to 12 injections. Injections at less frequent intervals than every 3 to 4 days may result in a decrease in antiserum titer between injections (R. W. Fulton, unpublished data).

The various types of serological tests used for other plant viruses are applicable to the stone fruit viruses. The microprecipitin test requires purified virus or well-clarified extracts; clarification by hydrated calcium phosphate has been convenient for this purpose (706).

The serological test most generally convenient is probably the double diffusion test in agar gel. Clarified extracts are not necessary, and a relatively large number of tests may be set up in a short time in one or two petri dishes. Washed Ionagar No. 2 at 0.6 to 0.7 percent, made up with water and with 0.02 percent NaN_3 is added just before solidification to inhibit microbial growth. Wells 6 mm. in diameter, spaced 4 mm. apart are convenient. Templates can be made which will punch a circle of six wells equidistant from a center well. Tests involving diffusion of antigen in agar cannot be applied to long filamentous viruses such as apple chlorotic leaf spot unless the virus is disrupted into small pieces that will diffuse readily.

A problem commonly encountered in serological tests with stone fruit viruses is that test antigen concentrations are not sufficiently high to give a visible reaction. There are a number of ways of coping with this problem. Wells can be spaced more closely in agar gel tests. Antiserum should be appropriately diluted when this is done to avoid reactions at the edge of the antigen well that might be overlooked. Virus can be transferred from a host in which it is present in low concentration to one in which it will reach a much higher concentration within a few days. Virus may be concentrated by high-speed centrifugation from dilute extracts, resuspended in a small volume of liquid and serological tests run with this concentrate, including impurities in agar gel tests.

Serology has not been widely used on virus-containing *Prunus* material. Virus concentrations are low, except during the shock phases of some diseases, or except in young leaves early in the season. The mucilaginous character of ground *Prunus* leaves and the rapidity with which the extract turns brown are also probably detrimental to serology. However, virus is detectable in *Prunus* material (169, 571, 595, 669). Young leaves forced from *Prunus* branches held at 20° to 25° C. have been reported to be a more reliable source of virus for serological tests than leaves produced naturally out of doors (595).

Relationships among viruses infecting *Prunus* have been established by means of serology. Thus, peach yellow bud mosaic virus is the same as tomato ring spot and grape yellow vein viruses (107, 266). Viruses isolated from hops and rose (759) have been identified as *Prunus* ring spot (75). A number of viruses causing line pattern symptoms in plum have been differentiated on the basis of serological unrelatedness (A. Q. Paulson and R. W. Fulton, unpublished data).

CONTROL OF STONE FRUIT VIRUS DISEASES

MAURICE F. WELSH

All control practices must be preventive, because there are no cures for virus diseases that can be applied on a commercial scale. Although preventive measures for long-lived woody plants are characteristically laborious and costly, such measures have been remarkably effective against many of the virus diseases of stone fruits.

The traditional recommendations for prevention of stone fruit virus diseases include: (1) propagating cultivars and stocks from sources that are free from detectable viruses; (2) isolating uninfected plantings from sources of infection; (3) replacing susceptible cultivars with resistant or tolerant cultivars; and (4) minimizing natural spread by suppression of vectors and other agents of spread.

In addition, control by heat therapy has limited uses. Recently there has been increased hope that chemotherapeutic methods can be developed. Indexing techniques contribute to many virus control procedures.

Virus-Free Propagating Materials

The most broadly applicable recommendation for reducing virus incidence in stone fruit crops is the propagation of healthy trees. Its effectiveness is governed only by the extent to which such plantings are subsequently exposed to or protected from natural infection, and inevitably by the availability of suitably indexed propagating materials. Most nurserymen and growers now have access to these materials.

In fruit-growing States and Provinces of the United States and Canada, and in many other countries, certification schemes have been established to provide tree fruit seeds, clonal stocks, and cultivars that are free from all detectable viruses. Such certification programs require the participation of pomologists to verify trueness to type and of plant pathologists to determine that the clones are free from recognized viruses. The administrative details of the schemes vary greatly, but the basic procedures for ensuring adequate virus screening are common to all of them. The customary first step is the indexing of numerous trees of a variety in a search for one that can be certified as free from known viruses. The term usually used is "virus-indexed," which indicates that by the indexing techniques currently in use, no virus infection can be detected. If the cultivar proves to be characteristically virus-infected, the healthiest available material is given heat therapy to eliminate virus infection.

The availability of fully indexed materials of most stone fruit cultivars from the IR-2 Interregional Tree Fruit Repository has obviated the need to derive such materials independently for regional certification schemes, each

of which can use IR-2 materials for establishment of its own nuclear plantings. Such "mother orchards" are protected from reinfection by all practical means and reindexed at regular intervals. In some established schemes, the nurseryman or grower is provided with his full requirements of budwood and seed directly from these plantings. In other schemes, he is required to establish his own bud- and seed-source trees, using propagating materials from the nuclear planting and maintaining these trees under conditions prescribed by the certifying authority. The nursery plantings are inspected at least once during the growing season and usually again at the time of digging. Most administering agencies provide tags or other evidence of certification to nurserymen who satisfy the requirements of the program.

The procedures for indexing stone fruit clones and for heat therapy are outlined in subsequent sections.

Isolation From Sources of Virus Infection

Healthy trees may be protected from infection in several ways. These include roguing diseased trees, eliminating other plants that serve as hosts of the virus, and establishing new plantings remote from existing diseased orchards or other sources of infection. Quarantine measures can also be classified as a means of isolating plantings from sources of infection.

Removal of diseased trees is especially effective against viruses that have low or moderate rates of natural spread and that display characteristic symptoms after short incubation periods. Individual tree roguing has been adequate for such diseases as red suture, peach yellows, and peach rosette. Enforced eradication programs applied to extensive acreages have been necessary to reduce incidence of more rapidly spreading diseases such as phony peach and peach mosaic.

Reservoir hosts of viruses can complicate control by tree removal. The host range of a virus must be determined before tree removal can be recommended. The opportunities for implementing eradication programs are especially prejudiced if some cultivars or kinds of cultivated fruits are symptomless carriers of the virus.

The viruses that cause many of the diseases of cultivated stone fruits are harbored in native plants, most commonly, *Prunus* spp. Eradication of these wild hosts within suitable distances of orchards has been an effective measure for X-disease, peach rosette, and phony peach. Eradication of wild hosts must accompany programs of orchard tree removal. Indeed, for X-disease in fruit districts of eastern North America, eradication of wild chokecherry is more effective than the roguing of diseased orchard trees, apparently because of the patterns of movement of the principal vector.

Isolation of new plantings from older infected ones is especially important for materials used in breeding programs, or for materials serving as sources for nursery propagation. In commercial orchards, there is usually less opportunity to achieve such isolation. However, interplanting of healthy trees with estab-

lished infected ones should be avoided where possible, because it provides maximum exposure to infection for the young trees.

National and regional quarantines and other regulatory measures that are imposed to prevent entry of viruses and their vectors to new countries, regions, or districts, constitute a type of isolation procedure. The administration of national quarantines is described in the section "Quarantine and the Detection of Stone Fruit Viruses in Plant Importation." Many States and Provinces restrict entry of fruit trees from regions of the continent from which new diseases can be introduced. The restriction may be a total prohibition of the movement of certain plant species, or it may involve combinations of inspection, fumigation, indexing procedures, and periods of postentry growth in isolation. Sometimes even more limited quarantine areas may be established, usually for restriction of spread of a specific virus. Such local restrictions have been directed, with considerable success, against phony peach virus in southeastern United States, and against little cherry virus between the two major cherry-growing districts in British Columbia.

Resistant Cultivars

In tree fruit crops, few cultivars have been found that possess total resistance or "immunity" to virus infection. Identification of cultivars with ability to escape natural infection has also been rare but has been demonstrated for Napa Long Stem Bing growing on mazzard rootstocks in California orchards invaded by Buckskin (X-disease virus). Tolerance to infection is much more common. The use of tolerant cultivars, or their development in breeding programs, has been recommended for little cherry, cherry mottle leaf, and peach mosaic. However, tolerance is seldom complete and consistent. Moreover, tolerant cultivars can serve as a reservoir of virus, and, thus as a source of inoculum for neighboring trees of sensitive cultivars.

Suppression of Agents of Natural Spread

Agents that have been shown responsible for transmission of viruses in stone fruits are insects, mites, nematodes, and pollen. Usually there need be concern for only one means of transmission for each virus. Moreover, the insect or mite vectors of a given virus are usually comprehended within one family; frequently within one genus.

Eradication or suppression of insect or mite vectors is an obvious and attractive measure but rarely effective. However, a spray program has been devised to reduce populations of the eriophyid mite vector of peach mosaic virus, and thus reduce the rate of spread of the disease. For control of peach yellows and little peach, spraying to reduce plum leafhopper populations is suggested as a precaution to supplement tree roguing.

The possibility of effectively depressing populations of nematode vectors by use of soil fumigants has been demonstrated for peach yellow bud mosaic. Precautions should be taken against movement of nematode-infested trees from diseased plantings to other orchards or districts.

Pollen transmission of prune dwarf and necrotic ring spot viruses can be prevented in valuable experimental, repository, or mother orchard plantings by deblossoming annually, but can be minimized in commercial orchards only by adequately separating healthy and diseased plantings.

Heat Therapy

Two types of heat therapy have been used successfully to eliminate viruses from stone fruits—exposing scion sticks to high temperatures for short periods or maintaining potted trees at lower temperatures for much longer periods (344, 491).

The higher temperature, short-period exposure is commonly achieved by immersing scion sticks in large volumes of actively circulating water at temperatures between 50° and 52° C. for 10 to 15 minutes. The scion sticks must be cooled quickly, and the buds transferred to clean rootstocks.

Much more success has been attained with lower temperature, long-exposure treatment. Plants are grown in containers, and when well established are placed in a chamber which supplies continuously circulating air at temperatures between 37° and 38° C. Effective periods vary from 2 weeks to the limit of tree tolerance. If growth is maintained in the chamber, tip buds attached to short wedges of shoot can be grafted to clean rootstocks. The wedges are inserted in incisions on the stems of the rootstocks, wrapped with surgical gauze, and protected for several days in plastic envelopes.

An alternative is to remove lateral buds from the heated trees and apply them to clean rootstocks by shield budding or T-budding. This second method depends on heat-induced inactivation of the virus, whereas the propagation of tip buds exploits the reduced ability of viruses to invade tissues laid down during the high-temperature exposure. Trees survive better if given pre-treatment of 1 or more weeks at 33° to 35° C. and if humidity in the chamber is maintained below 60 percent.

Another modification of the lower temperature, long-exposure procedure is the insertion of diseased buds singly into container-grown rootstocks that are known to be uninfected. For 3 to 7 days after budding the trees are placed in the heat chamber for the desired period. After removal, the dormant buds are forced by suitable pruning of the rootstocks, either immediately or after a period of dormancy.

Heat treatment survivals that appear to be freed of virus infection in the first indexing should be reindexed after 2 years to detect previously undetected infection.

Chemotherapy

Relatively few new methods for control of virus diseases of stone fruits have been introduced in the last 30 years. As this handbook is being prepared, there is promise that new types of treatment may be developed. Dimethylsulfoxide (DMSO) has been shown to move quickly and systemically through plant tissues and to suppress the symptoms of several plant virus diseases, including those

of peach mosaic in peach (522). Such materials, acting as antiviral agents themselves, or as carriers for other antiviral agents, may allow the development of curative procedures for some stone fruit virus diseases. Recent demonstrations of the association of mycoplasma-like structures with the occurrence of several yellows type plant virus diseases, and the reported effectiveness of antibiotic applications against such diseases, offer additional hope that cures for such diseases may soon be available.

Indexing

Indexing is used in the search for stone fruit clones that are free from known virus infection, and in periodic checking of virus-indexed clones to ensure that they have not become infected.

The indicator list devised for the IR-2 Repository (p. 22) serves as a basis for all stone fruit indexing programs in North America. All materials submitted to mother plantings should have been indexed on the full indicator range. For indexing of imported materials, the IR-2 list is supplemented by selections from host ranges devised by plant pathologists on other continents; notably by the European Committee for Cooperation in Fruit Tree Virus Research.

For routine reindexing of mother trees in certification programs, modifications in the IR-2 indicator range, or at least of the indexing schedule, are often possible. In most schemes, annual indexing is practiced for ring spot and prune dwarf viruses, either on both woody and herbaceous hosts each year, or alternating between them. Indexing for slow-spreading viruses may be spaced at longer intervals in a rotation schedule.

Specific Recommendations for Control of Viruses in Nurseries

1. Use only virus-indexed budwood, if possible by participation in a regional certification scheme.
2. Minimize risk of seed transmission by obtaining seeds from indexed trees or seedlings certified as having been grown from such seeds.
3. Avoid rebudding of rootstocks on which buds have failed. This doubles the chance of introducing viruses. Moreover, bud failure is often an indication of virus infection.
4. Adjust spacing between different varieties or clones to minimize the possibility of natural root grafting.
5. Segregate tree fruit nursery stock as far as possible from ornamental *Prunus* species that may serve as reservoir hosts of dangerous viruses. Ornamental *Prunus* are seldom as adequately indexed as commercial fruit-producing clones.
6. Rogue abnormal trees in nursery rows as soon as they are observed.

Specific Recommendations for Control of Viruses in Orchards

1. Buy trees from nurseries that participate in certification schemes or that take equivalent precautions in their own independent programs. The orchardist who grows his own nursery stock should observe all precautions recommended above for nurserymen.
2. Avoid as far as possible the interplanting of indexed nursery stock with

established trees that are virus-infected. This is especially important for sour cherries.

3. Be familiar with the symptoms of virus diseases; be prompt in removal of diseased trees, especially if the disease has an appreciable rate of spread; report occurrence of serious diseases to local advisory or regulatory officials; cooperate with them in eradication programs, when they deem them necessary.

4. Know host ranges of serious viruses, and remove wild hosts growing near enough to orchard plantings to be hazardous.

In Breeding and Other Research Programs

1. Use virus-indexed materials for all types of trials to obtain best tree performance and results that can be duplicated.

2. Establish breeding programs with virus-indexed materials to minimize the risk of creating new varieties that are infected. Varieties originated as seedlings can be infected because of seed or pollen transmission. When genetic changes are sought by irradiation or chemical treatment, the selection of clean clones is especially critical.

3. Multiply new cultivars by own-rooting whenever possible; topwork them on certified rootstocks if they cannot be own-rooted. The traditional practice of topworking on unindexed bearing trees to stimulate early fruiting is especially dangerous.

4. Add virus resistance screening to the usual performance tests for new cultivars and rootstocks.

5. Index new cultivars, mutants, and rootstock clones on all appropriate indicators immediately before their release to verify virus freedom.

6. When assembling cultivars and clones from widely separated regions, adhere rigidly to quarantine precautions to avoid introducing new viruses to your country or region.

IR-2, THE INTERREGIONAL DECIDUOUS TREE FRUIT REPOSITORY

PAUL R. FRIDLUND

Between 1935 and 1945, when rapid advances were being made in the identification of virus diseases of deciduous tree fruits, most plant pathologists in this research field recognized the need for assembling and maintaining clones of virus-free deciduous tree fruits which could be used as sources of test materials for research programs. The need for more general use of such materials to improve and standardize all types of tree fruit research and testing programs, and to improve the performance and quality of commercial nursery stock also became increasingly apparent.

Assembling and maintaining numerous virus-free clones by many individuals appeared both unnecessarily duplicative and economically impractical. Furthermore, the prime requisite of adequate isolation from other diseased trees, either by screen or distance, often was unavailable. In other instances, unfavorable local climate might have prevented the survival of some important clonal lines. Additionally, the adequate facilities, experience, labor, and funding necessary for the intensive indexing and maintenance of many clonal lines were usually lacking.

The logical solution appeared to be repositories. Plans for several regional repositories were tentatively devised by scientists within the various regions. However, some of the problems which would be encountered by individuals also remained for regional repositories. After further consideration, a single national repository was deemed advisable, and the plans were formulated by cooperative effort of many scientists from all regions. These plans were enthusiastically supported by the Deciduous Fruit and Nut Tree Research and Marketing Advisory Committee, an advisory committee to the Federal Government, composed of members of the industry.

Organization

The Interregional Research Project IR-2 was initiated on July 1, 1955, and is supported by regional research funds appropriated by the Federal Government under provisions of the Hatch Experiment Station Act of 1887. The headquarters, screenhouses, field plots, and other facilities are located at the Irrigated Agriculture Research and Extension Center, Prosser, Wash., a branch of the Washington Agricultural Experiment Stations system (fig. 1). Supervision is provided by a resident plant pathologist in charge. An isolated repository is maintained in the desert near Moxee, Wash. The latter is adjacent to a facility operated by the Washington State Department of Agriculture with whom irrigation water, labor, and machinery are shared (fig. 2).

The project has four objectives: to assemble virus-free clones of desirable



FIGURE 1.—IR-2 facilities at Prosser, Wash., include a greenhouse and several screenhouses, two of which are shown on the left.

deciduous fruit trees, to verify their virus-freedom by intensive indexing procedures, to maintain the clones in isolated repositories, and to distribute propagating materials to cooperators as foundation clones for research or for release to industry. The objectives also include provision for conducting research on methods of eliminating viruses from infected clones and on indexing techniques and related problems. The term "virus-free" as used herein, means that the plant material is considered to be free from viruses that are detectable by indexing. Cooperators may be defined as all State and Federal research and regulatory scientists.

IR-2 originally limited clonal accumulation and indexing to *Prunus* because only in this genus were the infecting viruses and their indicators understood sufficiently to establish an effective virus-free repository. However, many contemporary studies were being made, including those by an interregional subproject of IR-2, which elucidated some of the missing information relating to the viruses of *Malus* and their indicators. The results have recently allowed IR-2 to assemble and effectively index cultivars of *Malus*. These research results also may have additional value because it now appears that some deciduous tree fruit viruses have a multiple generic host range including *Prunus*, *Malus*, and possibly the Rosaceae in general.

Procedures

Accumulation of clones.—Potentially valuable, *Prunus* candidate clones are either proposed for the repository by cooperating scientists or solicited from them by the technical committee. Virus-freedom, as determined by previous limited indexing, is desirable before submission.

An approved candidate clone is received as budwood from which three trees are propagated on virus-free seedlings in a screenhouse. The remainder



FIGURE 2.—The isolated IR-2 repository site near Moxee, Wash., before tree planting. The buildings in the distance are a part of a facility operated by the cooperating Washington State Department of Agriculture.

of the budwood is used for preliminary indexing of the candidate clone on one or more of the standard index hosts. The hosts selected for this preliminary screening vary because experience with *Prunus* introductions has shown that some varieties and species, particularly from certain areas, are more prone than others to contain specific virus contaminant. The clone is discarded if the initial indexing detects an infecting virus. Meanwhile an attempt is made to find a noninfected source of the clone.

Methods of indexing.—The propagated trees are grown under screen in 5-gallon containers until they are large enough to provide adequate budwood for indexing. One of the three trees of each clone is indexed by budding three inoculum buds into three trees each of 10 *Prunus* indicators (p. 22). Additionally, the propagated trees are indexed by sap inoculation of *Chenopodium quinoa*. The indicator list is not static; it is changed by addition or deletion in response to the results of research and experience (fig. 3).

The period of observation varies with the indicator and the viruses to be detected. Some indicators require only one growing season of observation, whereas others must be retained long enough to inspect them for fruit symptoms.

Virus-infected clones.—If the first clonal propagant is found to be infected, a second tree of the clone is similarly indexed (fig. 4). If the second tree is infected, the clone is rejected, and a search is made for a different healthy source. If no healthy source is found, the rejected clone may be treated with thermotherapy in an attempt to eliminate the infecting virus. However, this procedure is not always successful. Thus, some important clones are not

represented in the repository because no healthy individuals could be found or produced.

Maintenance of virus-free clones.—If the tree under test is found to be virus-free, it is moved to a second greenhouse where it is maintained indefinitely as the nucleus mother tree of that clone. The remaining propagants of the clone are discarded. Some of these nucleus mother trees have been maintained for as long as 11 years in a 5-gallon container without having survival problems.

Usually two subpropagations are made from each nucleus mother tree for orchard-style planting in the isolated repository. Whenever possible the subpropagations are made by rooting softwood cuttings under mist. This prevents chance viral contamination of the clone from infected rootstocks. All trees of peach, mahaleb, and the miscellaneous species, planted in the repository, are on their own roots, as are most trees of apricot, plum, and sour cherry. All trees of sweet cherry, almond, and other varieties impossible to root from cuttings are bud propagated on seedlings grown from virus-free seed produced in the repository.

Reindexing of virus-free clones.—Maintenance indexing of a repository tree begins as soon as it is accepted as a nucleus mother tree or planted as a subpropagation in the repository. All trees are reindexed annually on Shiro-fugen ornamental cherry. This indicator is superior for detecting the latent viruses which presumably would spread most readily to healthy trees. The nucleus mother trees have been reindexed biennially on the 10 prescribed index hosts. The trees planted in the field at Moxee are indexed at less frequent intervals. To date, no virus spread has been detected either into the greenhouses or the isolated field plantings. This information has helped demonstrate the practicality of maintaining virus-free trees in greenhouses and isolated repositories.



FIGURE 3.—A field of 1-year-old, sweet cherry, virus indicator trees at Prosser, Wash.



FIGURE 4.—Trees from the rows shown in figure 3. Virus infected trees are on the *left*; healthy, on the *right*.

Cultivar identification. — Meanwhile, a program of cultivar identification verifies that the clones are true-to-name. The final identifications are made by competent pomologists. Although budwood of introductions to IR-2 usually originates from bearing trees, several inadvertent errors have been discovered.

The best method for verification of a cultivar is to fruit the tree in the field as is done in the repository. Identification in screenhouses is extremely difficult. Consequently, scions from greenhouse-grown trees are top-worked to trees in the field and forced to fruit for identification.

Release of virus-free materials. — The elapsed time from receipt of candidate clones until release to cooperators is at least 5 years. Lists of available clones are distributed periodically. The IR-2 repository began distributing virus-free budwood in 1965. Propagation material has been requested primarily by cooperating plant pathologists, pomologists, and entomologists representing State and Federal research agencies and State regulatory agencies. Considerable interest also has been shown by foreign research scientists, primarily Europeans.

The exact uses made of repository releases are difficult to ascertain. However, it is known that some releases have been used for research in several disciplines to establish virus-free blocks for evaluation or breeding, and as support for nursery improvement programs administered by State regulatory agencies. In the latter case, several States and Canadian Provinces have based the major portions of their programs on IR-2 clones, whereas other States have used these clones

to supplement their own. Interest in most foreign countries has centered on obtaining starts of the virus indicators used by IR-2. This service is provided in cooperation with Agricultural Research Service, USDA, and is helping to standardize research indicators.

Other Results and Observations

IR-2 procedures for virus detection currently are based solely on biologic responses of indicators, primarily because other techniques are not yet sufficiently refined to provide an adequate substitute. Sole dependence upon biologic indexing under the field conditions of one area is not without risks because the local environment may not be suitable for symptom expression of some viruses in some hosts.

About 16 percent of the clones submitted to IR-2 were found to be virus-infected even though most had a previous record of some indexing. *Prunus* ring spot and prune dwarf viruses predominated among the contaminants detected, with green ring mottle viruses next in frequency. The latter virus was never detected in sour cherry accessions, but rather it was found primarily in sweet cherries bred in the West and in peaches bred in one area of the Midwest. The little cherry virus was found in almost all of Japanese flowering cherry cultivars, and the remaining viruses detected were a random assortment in diverse hosts.

Suggestion has been made that all candidate clones should be given routine thermotherapeutic exposure upon receipt. This procedure may have some merit, but it is not without considerable hazard when working with unsuspected diseased clones. Although thermotherapy can now eliminate most of the viruses which infect *Prunus*, all propagative units obtained from treated plants may not be virus-free. Careful indexing using approved methods is required as it is for untreated plants.

Insecticides and miticides have never been used in the isolated repository during the 10 years that trees have been in the field. Although small numbers of virus vectors (e.g., aphids, leafhoppers, and mites) are usually present, particularly in the spring, populations never become dense nor is a particular species observed throughout the growing season. cursory observations suggest that populations of these arthropods are reduced to insignificance by undisturbed biological control. The seasonal presence of dense populations of the parasitic Ichneumonidae, Braconidae, and Asilidae lends circumstantial support for this hypothesis.

CURRENT MINIMUM RANGE OF IR-2 INDICATOR VARIETIES

The following *Prunus* cultivars were selected as the principle indicators for detection of the viruses listed within the environment of Prosser, Wash. Most viruses may also cause symptoms in one or more of the other indicators. All

indicators are inoculated in the field except *Prunus tomentosa* and *Chenopodium quinoa*, which are inoculated in the greenhouse at approximately 22° C. Different principle indicators for some viruses may be preferable or necessary in other environments. The reader is referred to the host range of a virus in question if a substitution appears advisable.

P. serrulata cv. Shiro-fugen: prune dwarf, *Prunus* ring spot.

P. serrulata cv. Kwanzan: Green ring mottle.

P. avium cv. Bing: Albino, black canker, freckle fruit, mottle leaf, rasp leaf, rusty mottle, short stem, spur Bing, sweet cherry rough fruit, twisted leaf, Utah Dixie rusty mottle, xylem aberration.

P. avium cv. Sam: Little cherry, necrotic rusty mottle.

P. armeniaca cv. Tilton: Apricot pucker leaf, apricot ring pox.

P. persica cv. Elberta: Peach asteroid spot, peach blotch, peach calico, peach mosaic, peach mottle, peach rosette, peach yellows, peach wart, phony, red suture, rosette mosaic, stubby twig, weak peach, X-disease, yellow bud mosaic.

P. cerasus cv. Montmorency: Pink fruit.

Prunus hybrid cv. Shiro plum: Plum line pattern, plum white spot.

P. domestica cv. Italian Prune: Prune mottle; prune dwarf.

P. tomentosa seedlings: Bark splitting of Montmorency.

Chenopodium quinoa: Apple chlorotic leaf spot, cucumber mosaic, tobacco mosaic.

QUARANTINE AND THE DETECTION OF STONE FRUIT VIRUSES IN PLANT IMPORTATIONS

ROBERT P. KAHN

Quarantines are regulations promulgated by governments to prevent or delay man from inadvertently upsetting the balance of nature to the detriment of the agriculture of a region or country. Man's previous experience in disrupting this system of checks and balances has resulted in devastating losses to plant and animal life such as the introduction of rabbits into Australia, measles into Africa, Klamath weed into Canada, the United States, and Australia, and both giant snails and hoof-and-mouth virus into many countries.

Viruses that are known to infect *Prunus* throughout the world include some of particular quarantine significance to the United States and Canada. Methods and indicators used to detect these viruses are discussed in this chapter. Literature citations are presented as sources of more detailed information about specific points or viruses rather than as surveys of the literature.

Quarantine

Legal basis. — The legal basis for quarantine may be found in (1) international plant protection conventions such as the Food and Agriculture Organization's International Plant Protection Convention (404), (2) intergovernmental agreements such as the European and Mediterranean Plant Protection Organization (199, 200), (3) Federal regulations issued by ministries or departments of agriculture such as USDA's Plant Quarantine Act of 1912 (677), and (4) State or provincial regulations.

The government regulations issued by ministries or departments of agriculture have been reviewed and abstracted for most countries (676). In general, these regulations (1) specify requirements of import permits, (2) require phytosanitary certificates, (3) require certificates of origin, (4) stipulate inspection upon arrival, (5) prescribe treatment upon arrival to eliminate a risk, and (6) prescribe quarantine or postentry quarantine, depending on the nature of the risk.

Quarantine status of plant importations. — Quarantine regulations of various countries utilize one or more of four classifications for plant importation with reference to their quarantine status: (1) absolute prohibition, (2) quarantine, (3) postentry quarantine, and (4) restricted entry.

Absolute prohibition is used when the risk of introducing pests or pathogens is so great that importation should be denied even to government services. Absolute quarantines are invoked when safeguards are inadequate and isolation from commercial crop production is not feasible.

Quarantine is used for plants imported from a country where hazardous pests or pathogens are known to occur. Admission of plants under quarantine is

usually reserved for government services and is not available to the general public. Safeguards consist of trained personnel, reliable methods of pest or pathogen detection, phytosanitation, and quarantine facilities (337).

Postentry quarantine status is usually assigned to plants for which hazardous pests or pathogens have been reported from some, but not all, foreign countries, and the importation originates from a country from which these pests or pathogens have not been reported. Postentry quarantine is usually reserved for government services, institutions, and qualified individuals whose facilities meet postentry quarantine requirements. Safeguards consist of inspection upon arrival and during a specified postentry period, usually for at least two growing seasons, on the premises of the importer.

Restricted entry status is usually assigned to all other plant introductions. "Restricted" means that plants are subject to inspection and treatment, if necessary, upon arrival at a port of entry or inspection station.

Quarantine regulations for Prunus

1. *International*. — A survey of the abstracts (676) of quarantine regulations of countries where stone fruits are of agricultural importance shows that the genus *Prunus* is almost always under quarantine or postentry quarantine. However, the reasons for promulgating these regulations vary among countries. Most European countries regulate *Prunus* (or Rosaceae) because of San Jose scale insects and/or fire blight bacteria. Some of the European countries also include regulations against virus diseases of fruit trees but such regulations usually do not name specific virus diseases with the exception of sarka (plum pox) virus. In some instances, *Prunus* plants are under regulation if they originate in soils previously used for grapes or from areas where golden nematode or the potato wart fungus is known to occur.

2. *United States*. — *Prunus* importation into the United States is regulated by the Nursery Stock, Plant, and Seed Quarantine 37 (677). *Prunus* species, including ornamentals, are prohibited because of a "diversity of plant diseases" from all countries with the following exceptions: (1) The USDA may import these prohibited species from any country for experimental or scientific purposes. (2) Vegetatively propagated root stocks may be imported if they originate from nurseries in foreign countries which are certified by the plant protection service of the originating country to have been grown from parent plants that were *tested* and found free of all diseases of plant quarantine significance including those caused by viruses. (A list of certified nurseries may be obtained by writing to the Plant Protection and Quarantine Animal and Plant Health Inspection Service, Federal Center Building, Hyattsville, Md. 20782).

Prunus imported for scientific purposes enter the United States under quarantine at the U.S. Plant Introduction Station, Glenn Dale, Md., to determine whether the *Prunus* is infested with pests or infected with pathogens of quaran-

tine significance. These are defined as hazardous organisms or infectious agents that (1) are not known to occur in the United States, (2) are not widely distributed in the United States, or (3) have not been identified.

3. *Canada.* — Regulations governing importations of *Prunus* into Canada from countries other than the United States (2) are similar to the U.S. regulations. However, in addition, Canadian regulations require that *Prunus* introductions from the United States to Canada must be accompanied by a State or Federal certificate stating that the source was inspected during the growing season and is believed to be free of virus diseases, as may be designated from time-to-time, such as little cherry, peach mosaic, or phony peach. Also, fumigation may be required (or entry refused) for nursery stock received as balled-and-burlapped if *Xiphinema americanum* or other harmful nematodes are found in the shipment.

Prunus introductions from foreign countries (except the United States) otherwise prohibited, enter Canada for scientific purposes through the quarantine station at Saanichton, British Columbia.

Quarantine Policy Governing the Release of Virus-infected Plant Introductions. — Federal quarantine regulations, as discussed previously, exclude plants infested with pests or infected with pathogens of quarantine significance. A problem arises, however, as to the proper course of action, when an introduction is infected with a virus that is not of quarantine significance. The problem is minimized if the importer experiences a conflict of interest between the benefit of importing new germ plasm and the risk of introducing a pathogen. The problem takes on added significance when the importer fails to experience a conflict between benefits and risks — an attitude that fosters a disregard for phytosanitary aspects of plant introduction. Consequently, the USDA, in cooperation with State quarantine officials and virus specialists, formulated a policy governing the release of foreign plants infected with viruses that are not considered of quarantine significance.

In general, it is the Department's policy to exclude plants infected with viruses of quarantine significance, and to ask the State quarantine official to determine the admissibility of plants infected with a virus that already occurs in his State. Some factors that influence this determination at the State and Federal level are: (1) Quarantine facilities and phytosanitation awareness of the importer, (2) the presence of nursery stock certification programs or virus-free clone collections at the location of the importer, (3) the potential importance of the introduction to the development of the stone fruit industry in the State, (4) the degree of isolation from commercial production, (5) the possibility that the foreign source of a virus already in the State might be a more severe strain, and (6) opinions of virologists. The decision-making process is essentially a weighing of risks against benefits (337). In practice, quarantine officials consider each case on its individual merits.

The situation concerning *Prunus* is complex. On one hand, some *Prunus* breeders expressed more-than-usual concern about the loss of some sources of germ plasm merely because the plants were virus-infected. On the other hand, quarantine officials in some States with *Prunus* nursery stock certification programs, were equally concerned about jeopardizing virus-free foundation plantings located at the same institution as the breeder's plots.

Consequently, before formulating a policy for *Prunus*, the USDA sent questionnaires in 1963 and 1964 to stone fruit virus specialists and State quarantine officials. The responses showed that almost all participants concurred in the Federal quarantine policy, which excluded viruses of quarantine significance. However, 90 percent of the virus specialists and 70 percent of the quarantine officials in 14 States, that account for 93 percent of the stone fruit production, also favored the exclusion of foreign sources of viruses already established in the United States such as *Prunus* ring spot virus (unless the *Prunus* was of exceptional importance to the stone fruit industry).

Consequently, it is the USDA policy to destroy *Prunus* introductions infected with virus whether or not the virus is of quarantine significance with the following exception. If a *Prunus* introduction is infected with a virus that is not of quarantine significance and the introduction is judged by the USDA to be of unusual importance to the improvement of the stone fruit industry, the introduction may be admitted into a State with the approval of the State quarantine official. (Frequently, one of the conditions specified is that the importation be subjected to heat therapy and reindexing). In Canada, important clones are heat-treated immediately upon receipt (before indexing) if the source is suspect. Otherwise, these clones are heat-treated if preliminary indexing is positive.

Viruses Infecting Stone Fruits

Listing of viruses. — Now that we have considered some of the principles of plant quarantine and the regulations pertaining to *Prunus*, it would be convenient to compile a list of *Prunus* viruses from which those of quarantine significance might be selected. However, compiling such a list is fraught with difficulty. The listing of stone fruit viruses and virus diseases is not only complicated by synonymy and double-virus infections but by the fact that some naturally occurring viruses of stone fruits do not even have a stone fruit in the virus name. For the sake of discussion, the Review of Applied Mycology (RAM) (now Review of Plant Pathology) list of plant virus names (439) can be used as a starting point although the RAM list — and probably any other list that might be compiled — is not universally accepted by stone fruit virus workers. The RAM list contains 232 entries for common names of stone fruit viruses or virus diseases that currently can be associated with 55 viruses, as follows:

<i>Stone fruit</i>	<i>Number of viruses with a stone fruit in name of virus</i>	<i>Number of common names in index</i>
Almond	0	4
Apricot	5	13
Cherry	23	107
Nectarine	0	0
Peach	17	56
Plum	6	26
Prune	3	5
<i>Prunus</i>	1	21
	<hr style="width: 10%; margin: 0 auto;"/> 55	<hr style="width: 10%; margin: 0 auto;"/> 232

These figures include strains and synonyms but not the Latin nomials although these are also included in the index.

In addition to the 55 viruses in the RAM list, we should add the viruses that occur in *Prunus* but which do not have a stone fruit in the name. Among these are apple chlorotic leaf spot; tomato bushy stunt; tobacco mosaic; cucumber mosaic; and the nematode-transmitted, polyhedral (NEPO) viruses such as arabis mosaic, raspberry ring spot, and tomato black ring viruses. Also, viruses that have been described since the list was published should be added.

To be complete, the list should also contain the paired names of viruses which incite a virus disease that is different from the disease incited by either virus alone, such as (1) arabis mosaic virus + prune dwarf virus and (2) raspberry ring spot virus + prune dwarf virus. These pairs have been implicated as incitants of cherry Eckelrade, Pfeffinger, and rasp leaf (European) diseases.

Finally, in considering any listing of viruses one should not be shackled to the concept of looking only to the genus *Prunus* as sources of stone fruit viruses; for example, apple chlorotic leaf spot virus has been found in *Amelanchier*, *Sorbus*, pear, plum, peach, and raspberry. *Prunus* ring spot virus has been found in hops, roses, and apples. Cucumber mosaic, tobacco mosaic, and the NEPO viruses have wide host ranges in crop and wild species.

Viruses of quarantine significance in the United States and Canada.—Among the viruses of quarantine significance, and therefore subject to Federal regulation by the United States and Canada, are: (1) plum pox (186, 528, 638), (2) cherry leaf roll (170), (3) arabis mosaic (103, 104, 280), (4) raspberry ring spot (104, 280), and (5) tomato black ring (104, 280) viruses; also, apricot apoplexy (479), which may be incited by a virus. None of these are known to occur in North America.

Of these viruses, plum pox is of the greatest concern because it spreads rapidly in commercial stone fruit areas in eastern Europe, it destroys quality of fruit, and reduces the yield. Arabis mosaic, tomato black ring, and raspberry

ring spot viruses are important because they are spread by nematodes and have broad host ranges in many nonrosaceous crops and wild species.

Among the viruses already present but not widely distributed in the United States, and either not present or not widely distributed in Canada, and, therefore, subject to State or Federal regulation or both are: (1) peach mosaic, (2) peach yellows, (3) peach phony, (4) peach rosette, (5) X-disease and strains, and (6) tomato ring spot, peach yellow bud mosaic strain. In addition, little cherry (K & S) virus although widely distributed as a latent infection in flowering cherries is of concern because of its potential severity in sweet cherry growing regions of the United States and Canada.

Because Federal regulations exclude viruses of quarantine significance and both State regulations and State or Federal policy exclude almost all other viruses, the primary objective of the plant introduction and quarantine stations at Glenn Dale, Md., and Saanichton, British Columbia, is detection. For all practical purposes, a philosophy of "what's in a name" prevails in routine testing. Priority is given to detection rather than identification. Identification does not necessarily come into the picture unless the *Prunus* introduction is of extreme importance to the stone fruit industry and a decision must be made concerning the admission of a virus-infected plant. Of course identification is a prerequisite to determining whether the virus represents a new strain or new virus.

Methodology

In quarantine stations, stone fruit viruses are detected in foreign plants by transmitting an infectious agent that incites virus diseaselike symptoms in one or more indicator plants. The indicators are woody *Prunus* species or cultivars or herbaceous nonrosaceous plants recommended by stone fruit virus specialists. Transmission to woody plants is effected by some form of grafting and to herbaceous plants by mechanical transmission.

Mechanical transmission. — Mechanical transmission is accomplished by rubbing carborundum-dusted indicator plants with an inoculum prepared from leaf, petal, root, or pollen extracts. *Prunus*-to-*Prunus* mechanical transmissions have not proved useful on a routine basis. Transmissions have been inconsistent presumably because of the exacting requirements of proper age of inoculum tissue, age of *Prunus* indicator plants, and the necessity of incorporating additives to the inoculum to suppress inhibitors. However, *Prunus*-to-herbaceous plants (nonrosaceous) have been extensively used for testing a few stone fruit viruses such as *Prunus* ring spot (225, 374), prune dwarf (225, 374), tomato bushy stunt (16), tomato black ring (34, 104, 280), and plum pox (345). The most commonly employed *Prunus*-to-herbaceous plant indexing test is the *Prunus*-to-cucurbit test (93, 473) for the detection and identification of *Prunus* ring spot and prune dwarf viruses. (See section under "Techniques of Investigating Stone Fruit Viruses.") At Glenn Dale, the following herbaceous varieties are used as indicators: Improved Long Green Cucumber, White Bush Squash,

and Buttercup Squash. High greenhouse temperatures either mask symptoms or are not conducive to infection. Consequently, because young inoculum tissue is a requirement for routine indexing and high greenhouse temperatures are detrimental, the testing is usually confined to the period between February and May.

Graft transmission. — Grafting is accomplished by budding the foreign *Prunus* to woody indicators or by double budding both to an appropriate seedling rootstock. The buds to be indexed are obtained from a tree that was derived from a bud cut from an imported scion and budded on a domestic seedling rootstock. For convenience, the buds subsequently produced on this tree are referred to as "foreign buds." However, they only stem from a foreign bud. They were actually produced in a quarantine screenhouse at Glenn Dale. Two foreign buds are inserted in the usual T-cut on each of two indicator trees. If the foreign buds do not remain alive at least 3 weeks, the indicator trees are rebudded. A bud contact period of a few days to 2 weeks has been reported as necessary for the transmission of several viruses (222, 388). The ornamental *Prunus* species, such as *P. laurocerasus zabeliana* which are incompatible when budded, are either approach-grafted to container-grown indicators or they are not admitted.

The incubation period, or the period that indicators are observed for symptoms, varies with the indicator and the viruses to be detected. Some indicators require one growing season whereas others require a period long enough for fruit development. Foliage is observed for at least 2 years after the year of budding.

Woody indicators for stone fruit viruses. — At the Glenn Dale and Saanichton Introduction Stations, a compromise must be reached between the total number of indicators that have been recommended to detect the viruses that infect stone fruits throughout the world and the practical limitations imposed by the availability of facilities, land, personnel, and funds. Perhaps the most that one can do is to include the indicators described in the previous section for North American viruses plus those recommended in the foreign literature for viruses not known to occur in North America — keeping in mind that stone fruit viruses occur outside of the genus *Prunus* so that apple and pear indicators (201) might be included. Certainly the very least that one can do is to use virus-free seedlings of commercial stone fruit varieties. The former approach is too conservative to be practical and the latter, too liberal to be safe.

The manner in which this compromise has been reached for the Glenn Dale and Saanichton Introduction Stations, the IR-2 Repository in Prosser, Wash., and stations in Europe has been summarized in table 3. The lists vary among geographic areas depending on the relative importance of the various viruses and the differences in commercial cultivars. An underlying rule-of-thumb in selecting cultivars as indicators, where two or more are equally sensitive to a virus, is to select the one that has the greatest commercial production in the

area where indexing occurs. If a new virus should appear on the scene, there is an advantage in using an important commercial cultivar as an indicator. The new virus might be latent in the indicator, while causing severe damage to the commercial cultivar. For example, in eastern Europe, the Pozegaca plum (*Prunus domestica*) is an ideal indicator because it not only detects and dif-

TABLE 3.—*Minimum list of stone fruit virus cultivar indicators used or recommended in the United States, Canada, and Europe*
[+ = used or recommended; (+) = alternative; — = not used]

	United States		Canada Saanichton, British Columbia ³	Europe ⁴
	Glenn Dale, Md. ¹	Prosser, Wash. ²		
<i>Prunus indicator</i>				
Cherry:				
Bing	+	+	+	+
Mazzard F12/1	—	—	+	+
Lambert	—	—	+	+
Sam	+ ⁵	+	+	+
Montmorency	+	+	+	+
Flowering cherry:				
Shiro-fugen	+	+	+	+
Kwanzan	+ ⁶	+	+	+
Apricot:				
Tilton	+	+	+	+
Wenatchee	—	—	+	(+)
Peach:				
Seedlings	—	—	—	+
Elberta	+	+	+	—
Fairhaven	—	—	(+)	—
Almond:				
Peerless	+	—	—	—
Plum and prune:				
Shiro	+	+	+	+
Cambridge	—	—	(+)	+
Pozegaca	+	—	+	+
Italian Prune	+	+	—	+
Prune d'Agen	—	—	—	+
<i>Prunus tomentosa</i>	—	+	—	—

¹ Host Range Committee, Interregional Stone Fruit Virus Research Group.

² Interregional Project IR-2.

³ Post Entry Quarantine Station, Saanichton.

⁴ European Committee for Cooperation on Fruit Tree Virus Research.

⁵ Some tests conducted at the Fruit Tree Experiment Station, Wenatchee, Wash., by E. L. Reeves and P. W. Cheney.

⁶ Effective 1970.

ferentiates between plum line pattern and plum pox viruses but also it is of commercial importance. In North America, Pozegaca is not of commercial importance. At this writing Pozegaca is grown only in the indexing plots at Glenn Dale, Prosser, and Saanichton. Shiro plum, which is of commercial importance, will detect but not differentiate both plum line pattern and plum pox viruses. Therefore, in the United States, Pozegaca is not expected to replace Shiro as an indicator but should be useful when virus identification is required.

In an indexing program based on 10 indicators, the testing of 50 *Prunus* introductions would require more than 5,000 separate budding operations. To prepare indicators (unless these are purchased), 2,000 buddings are required (10 indicators \times 2 replications \times 2 buds \times 50 tests). To prepare one check per four indicator trees, another 500 buds must be inserted. To index 50 *Prunus* introductions, 2,000 foreign buds must be budded on indicator trees (10 indicators \times 2 replications \times 2 buds \times 50 tests). Assuming an 80-percent foreign bud-take, another 500 buddings may be required to insure a 3-week contact period.

At Glenn Dale, the *Prunus*-to-cucurbit mechanical transmission test is used to screen introductions prior to indexing on woody plants. Unless the introduction is of unusual horticultural importance, there is no point in entering the clone into the more expensive woody indexing test if it cannot pass the *Prunus*-to-cucurbit test.

Some results of virus detection and identification tests have been published for the Glenn Dale (338, 339) and Saanichton (715) Introduction Stations.

Identification. — Although the primary objective of indexing tests at a quarantine station is detection, there are circumstances when additional testing is warranted in order to identify the virus detected. For example, the identification of a detected virus would be prerequisite (under State and Federal policies as discussed previously in this section) to determine whether a tree is infected with nothing more than a common virus already widely distributed in the U.S. stone fruit growing areas. Fortunately, many of the symptoms on indicator plants are sufficiently diagnostic at least for typical strains to permit identification at the same time as detection on the basis of host range and symptoms.

In addition to host range and symptoms, other tests may be employed to facilitate identification. Serology has been used to identify some viruses such as *Prunus* ring spot and prune dwarf (14, 75, 233, 571, 738), cherry leaf roll (169), tomato ring spot, peach yellow bud strain (107), and the NEPO viruses (104, 280). Cross-protection has been used to determine relationships of *Prunus* viruses (103, 107, 732). Synergism can also be employed (317). Comparisons of symptoms incited by "unknowns" and "knowns" on various hosts are also useful. However, neither cross-protection, synergism, nor comparisons on *Prunus* hosts in the field are used at Glenn Dale because these tests require the maintenance of known cultures of stone fruit viruses in living *Prunus* hosts — a practice at variance with the policies of phytosanitation at quarantine

stations. The electron microscope has been used for characterizations of some *Prunus* viruses (108, 281, 345, 512, 737). The determination of properties such as thermal inactivation point, dilution end point, and longevity in vitro, although widely employed in virus characterization, is limited in stone fruits to those viruses that are mechanically transmitted (14, 16, 107, 345, 512). Finally, viruses may also be characterized by modes of transmission such as insects and mites (625, 717), seed (219, 235, 238), nematodes (104, 280), and pollen (261, 708, 725) in addition to sap and grafting.

VIRUS DISEASES OF ALMOND AND APRICOT

ALMOND VIRUS BUD FAILURE

GEORGE NYLAND

Causal Virus

Almond bud failure virus (ABFV).

Synonyms

Drake almond bud failure (744); peach mule's ear (745); wild leaf (606). Peach willow twig (649, 650) has some symptoms similar to almond virus bud failure in peach and at least some of the trees diagnosed as willow twig were infected with ABFV (S. M. Alcorn and George Nyland, unpublished data). Several strains of *Prunus* ring spot virus (PRSV) also induce bud failure and willow twiglike symptoms in peach.

History and Distribution

Almond virus bud failure as a distinct disease of almond was first reported in 1947 on Drake and Nonpareil almonds (637). In symptomatology, it resembled a nontransmissible almond bud failure (739, 742) believed to be a genetic disorder. A peach disorder, possibly related to almond virus bud failure, was named mule's ear in 1955 (745). Mule's ear resembles a condition in peach described and named wild leaf in 1932 (606) and probably can be equated with it. However, there is a wild leaf condition in Carolyn peach that is nontransmissible, but it is known only from this variety (H. K. Wagon and George Nyland, unpublished data). The almond calico strain (AICs) of PRSV seems to be associated consistently with almond virus bud failure and peach mule's ear, where this has been determined, but its causal relationship to the two diseases has not been clearly established. Whether the peach disease willow twig should also be considered a synonym of almond virus bud failure is still open to question because so little is known about the virus or viruses that are associated with the diseases.

Almond virus bud failure occurs in many older almond orchards and in some young orchards planted with infected trees in the central valley of California. Its widespread distribution in almond was not recognized until recent years. Natural spread of the disease is strongly indicated in at least one orchard of Nonpareil in Colusa County, Calif., and in several other sites including one in the Winters district of Yolo County and another in Stanislaus County.

Economic Hosts

The disease occurs naturally in several cultivars of almond and has been produced by inoculation in Drake, Nonpareil, Peerless, Jordanolo, Mission (Texas), and Ne Plus Ultra. In peach, the mule's ear disease has been seen in

nine clingstone cultivars, three freestone cultivars, and in many seedlings of Lovell peach. At least three nectarine cultivars are known to show symptoms: Quetta, Early LaGrande, and Late LaGrande. Sweet and sour cherry and Shiro plum are carriers of ABFV as are mazzard and mahaleb seedlings (H. K. Wagon and George Nyland, unpublished data).

Symptoms

Almond virus bud failure is characterized by failure of blossom and leaf buds to grow and the constant association of the AICs of PRSV spot virus with the disorder in the field. Often no buds are produced in the axils of leaves of the portions of current season shoots produced late in the season. Many lateral buds that are formed die and are cast prior to budbreak or they may remain attached to the shoot for several months. The terminal bud may be the only surviving bud on the previous season's growth or a few basal buds may also survive (fig. 5). Often the terminal bud fails to grow, in which case the shoot, over a period of weeks or several months, dies back to the uppermost live lateral bud. Over a period of years, annual growth from only the terminal bud and from a few basal buds of many twigs produces a dense brushy growth of predominately bare shoots with few lateral spurs in the portion of the tree affected (fig. 6). Affected twigs usually have shorter internodes than unaffected ones of the same tree or of healthy trees.

Symptoms of almond calico are seen in the leaves of some cultivars e.g., Nonpareil, IXL, Ne Plus Ultra, Peerless, Jordano) that also show virus bud failure symptoms. Drake and Mission almonds in stabilized infections rarely show calico leaf symptoms, but almond calico virus is readily recovered from trees of these cultivars that show bud failure symptoms. Other leaf symptoms that have been associated with almond virus bud failure in almond are a slightly darker green, an erect rather than drooping posture, and retention of leaves until very late in the fall.

Few blossoms and fruits are produced on affected branches after bud failure symptoms become obvious. Not infrequently the disease is more severe on some branches of a tree than on others although the infectious agent can be transmitted from any portion of the tree. Symptoms of almond calico are often more prominent on the branches showing the most pronounced bud failure.

The symptoms of almond virus bud failure in peach have been referred to as mule's ear (745) because of the erect leaves near the terminals of slender willowy shoots (fig. 7). These leaves are retained on the tree until long after normal leaves have dropped in the fall. Failure of lateral buds of diseased shoots to grow the spring following the year they are produced and the absence of buds in the axils of leaves produced during the latter part of the growing season are additional symptoms. Symptoms of almond calico are more or less evident in leaves produced during the spring in peach trees with mule's ear symptoms. Calico symptoms fade and may disappear entirely during the summer, but

almond calico virus is invariably associated with the disease (H. K. Wagnon and George Nyland, unpublished data).

Fruit symptoms in peach trees infected with almond virus bud failure are very striking (fig. 8). The cheeks of some fruits remain green and flattened, and the stem and styler ends are rounded and ripen with normal color. Other fruits may be irregular in shape and ripen later than normal fruits. Fruits with symptoms are seen usually on branches infected during the current season. In old infections, few fruits are produced. The disease can reduce the crop significantly in some trees. Usually only limited portions of the trees show leaf symptoms; these are most evident late in the season. Normal pruning removes most of the diseased shoots so that the disease in peach may be easily overlooked during the first half of the growing season.

Transmission

ABFV has been transmitted by grafting to Drake, Nonpareil, Peerless, and Jordanolo (744, 745) and to Mission and Ne Plus Ultra (George Nyland, unpublished data) almond cultivars. Mission and Ne Plus Ultra do not develop as conspicuous symptoms of bud failure as the other cultivars, but the virus can be recovered from them. Virus from almond trees with symptoms of bud failure was transmitted to peach that subsequently developed mule's ear syndrome (745) and to sweet and sour cherry, Shiro plum and mahaleb and mazzard cherry seedlings. The symptoms of mule's ear in peach and bud failure in almond are produced 2 to 3 years after inoculation.

Mechanical transmission of virus to the common herbaceous hosts of PRSV is readily effected from almonds with bud failure or from peach with mule's ear. The virus obtained has been returned to *Prunus* hosts and has induced symptoms typical for almond calico but not as yet the complete almond virus bud failure—mule's ear syndrome (George Nyland, unpublished data).

Remarks

The AICs of PRSV is invariably associated with both almond virus bud failure and peach mule's ear wherever the diseases have been found. However, although studies with pure cultures of this strain in almond and peach are in progress, they have not yet shown conclusively that the AICs is causally associated with the two diseases (George Nyland, unpublished data). The cherry rugose mosaic strain of *Prunus* ring spot virus also induces bud failure and calico leaf symptoms in several almond cultivars in addition to other symptoms (fig. 9).

Distortion of the leaves and a more pronounced mosaic associated with the cherry rugose isolates in almond helps to differentiate rugose mosaic from almond virus bud failure in field trees of almond.





FIGURE 5.—Almond virus bud failure: *Left*, Drake almond branch showing the effect of 3 years of bud failure. On four shoots produced the previous year, only two terminal leaf buds, three lateral leaf buds, and one blossom bud grew; *right*, Gaume peach branch showing failure of terminal and lateral buds and willow shoots with upright leaves (mule's ear symptoms). Many leaves have no macroscopically visible buds in their axils.



FIGURE 6.—*A*, Drake almond tree affected with almond virus bud failure, showing rigid, upright leaves, bare twigs, and absence of fruit; *B*, comparable unaffected tree showing normal growth and a good crop of fruit.



See legend on p. 40.



FIGURE 7.—Almond virus bud failure: *A*, Gaume peach tree inoculated with tissue from Peerless almond infected with almond bud failure virus (ABFV). Center shoot shows erect leaves or mule's ear symptoms, that remain on the tree long after normal time of leaf fall; *B*, shoot from Gaume peach tree showing calico leaf symptoms.

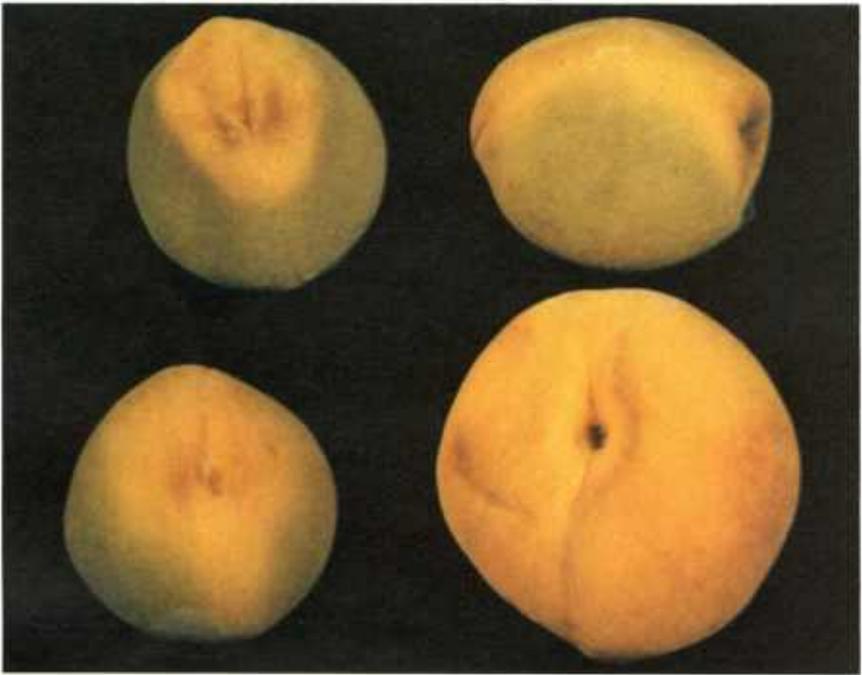


FIGURE 8.—Almond virus bud failure. Peach fruit showing delayed ripening of flattened cheeks.



FIGURE 9.—Failure of buds of Nonpareil almond induced by cherry rugose mosaic strain of *Prunus* ring spot virus, resembling almond virus bud failure and noninfectious almond bud failure.

APRICOT PUCKER LEAF

BRYCE N. WADLEY

Apricot pucker leaf virus was found in 1957 in several orchards in Washington County, Utah (686). No additional infected trees have been found, indicating that natural spread has not taken place and that the disease is probably of minor importance. Transmission by grafts was successful to apricot, peach, Manchu cherry, and Marianna plum. Attempts to infect sweet cherry, sour cherry, desert almond, Kwanzan flowering cherry, and mahaleb cherry were not successful.

Symptoms first appear as chlorotic areas in the middle of young, developing apricot leaves. As the leaves enlarge, they develop a marked puckering (figure 10). Much of the chlorotic tissue often becomes necrotic and falls out, giving the leaves a tattered appearance at maturity. Fruit symptoms are mild and consist of dark-green to reddish-brown spots on the surface of immature fruits. Slightly deformed fruits are found occasionally. Symptoms were observed in Moorpark seedling trees and in Perfected, Riland, Royal, Tilton, Moorpark, and Sunglo.

Leaf symptoms were obscure and generally absent in Elberta and Gleason Early Elberta peach cultivars. A few slightly deformed leaves and a few young leaves with mild vein chlorosis were seen in infected peach trees growing in the greenhouse. No fruit symptoms were seen in peach. Leaf symptoms were usually as severe in Manchu cherry as in apricot, but no symptoms were detected in Marianna plum. Transmission was readily achieved to host plants by budding



FIGURE 10.—Puckering, chlorotic spots and patterns, and tattered effect due to loss of tissue on leaves of Moorpark apricot seedling affected by apricot pucker leaf.

or tissue grafts, and the virus was recovered from inoculated apricots, peaches, Manchu cherries, and Marianna plums the season following inoculation, regardless of whether symptoms developed or not.

Symptoms of apricot pucker leaf somewhat resemble those of peach mosaic and of apricot ring pox in apricot leaves. Both of these diseases have been found in the same general area as pucker leaf. However, when symptoms were compared in greenhouse tests, those caused by apricot pucker leaf virus did not resemble those caused by the other viruses.

APRICOT RING POX

A. J. HANSEN, C. L. PARISH, and T. S. PINE

Causal Virus

Apricot ring pox virus (ARPV).

Synonyms

Apricot ring spot (83); apricot pox; ring pox. Local forms have been named after typical symptoms, such as tatter leaf and pit pox.

History and Distribution

Apricot ring pox was reported first from Colorado (83) and has since been found in Washington (554), British Columbia (405), Utah (682, 685), and California (501). It is now known to occur in all the apricot-growing areas west of the Rocky Mountains, but there is no substantiated report of its occurrence outside this region.

Economic Hosts

Apricot (*Prunus armeniaca*), all cultivars. Some are symptomless carriers of ARPV (521, 559).

Other Hosts

The only known natural hosts of ARPV are apricot, western chokecherry, hybrid plums (*Prunus salicina* x *P. simonii*), and sweet cherry. The virus can be experimentally introduced into certain cultivars of almond, nectarine, Japanese plum, peach, sour cherry, desert peach, Bessey cherry, and black cherry. Abundance plum (*P. salicina*), Shiro plum (hybrid), Italian Prune (*P. domestica*), American plum, and bitter cherry are immune to ARPV (696 and P. W. Cheney and C. L. Parish, unpublished data).

Symptoms

Symptoms of apricot ring pox occur in the fruit, leaves, and current season twig bark of affected trees (85, 164). The first leaves to expand in the spring may be without symptoms. Obscure spots develop in successively formed leaves, particularly in the rapidly growing shoots. Simultaneously, veinbanding, chlorotic spots, streaks, and rings appear at random and increase in size and number (fig. 11). These symptoms become increasingly pronounced until midsummer and are especially conspicuous in years of below normal temperature (164). When the entire border of a spot or ring becomes necrotic, the center falls out, producing holes with a ragged or angular outline. On the current season twigs of some cultivars, reddish spots appear at about mid-season and eventually become necrotic.

Fruit of affected trees appear healthy until the pit-hardening stage. Then, discolored or necrotic areas begin to develop on the fruit surface, under the skin, or inside the flesh. These areas expand and coalesce, forming reddish-purple

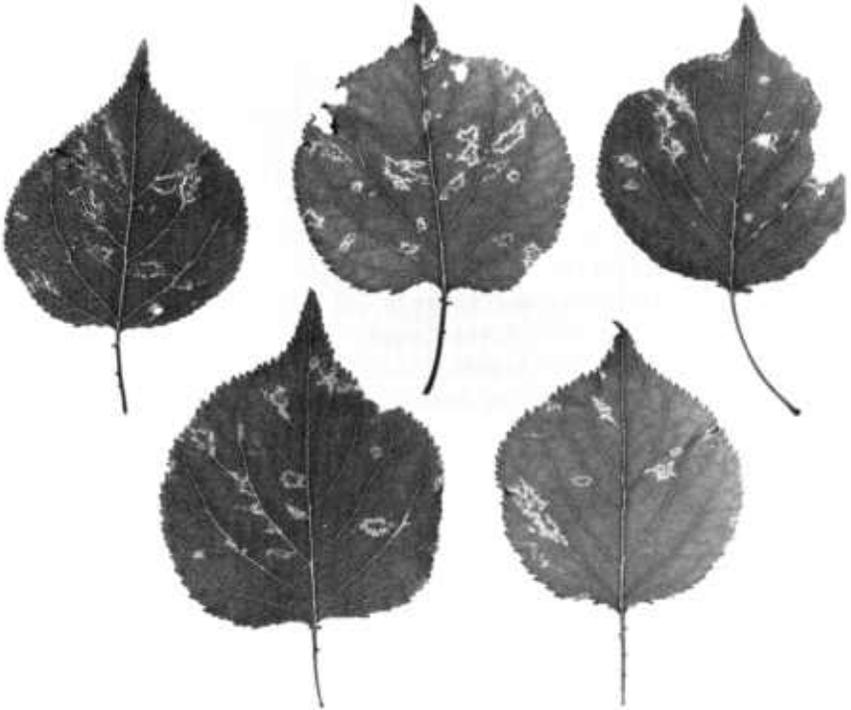


FIGURE 11.—Chlorotic rings and ragged holes caused by apricot ring pox virus (ARPV) in apricot leaves.

or black spots, rings, pits, and arcs. As the fruit reaches the soft-ripe stage, the discolored areas may become slightly raised and may be surrounded by concentric lines or radial cracks (fig. 12). In some fruits, the discoloration and necrosis extend deep into the flesh, where dry black pits are formed. In other cases, symptoms are very mild, consisting of a light scurf, which may disappear altogether by harvest time. The variation of symptoms which is found in field trees is caused mainly by differences among virus strains and among reactions of apricot cultivars.

Distribution of poxed fruit in diseased trees tends to be irregular; some branches may produce fruit that are severely affected, whereas others produce mostly healthy fruit. During the summer following infection, only a few fruits may develop characteristic symptoms, but in subsequent years 30 to 100 percent of all fruit is affected.

A marked fruit drop has been observed in British Columbia and Washington at about the time other fruit symptoms begin to appear, but this symptom has not been reported from south of the Washington-Oregon border. Under Colorado growing conditions, developing fruit appears healthy until approx-

imately 2 weeks before ripening. After that time, protuberances develop on the fruit and are followed by typical ring pox symptoms (83).

Of the major commercial apricot cultivars, Tilton and Wenatchee (Wenatchee Moorpark) develop severe pox and leaf spots when infected by most forms of ARPV. Royal (Blenheim), Reliable, Sunglo, and Blenril are usually symptomless. Responses to inoculation with ARPV in many other local and foreign apricot cultivars have been cataloged and published (521, 559).

Foliage symptoms of apricot ring pox in plum are similar to those in Tilton and Wenatchee apricots but are generally less severe. Plum fruit remains unaffected (609, 696).

Various strains of ARPV have been distinguished on the basis of symptoms in Tilton, Wenatchee, Royal, and Perfection. The common strain of ARPV induces in apricot fruit a superficial black scurf or purple-to-black rings and arcs, which may be elevated but never penetrate to the seed. Leaf symptoms are generally present in Tilton but not always in Wenatchee or hybrid plum. The common strain predominates from central Washington southward into California and eastward into Utah and Colorado.

The pit pox strain induces black cylindrical plugs which penetrate the fruit flesh and reach the seed in most cases. These plugs are very pronounced in Tilton, Riland, Perfection, and Wenatchee, but leaf symptoms are often absent in these cultivars. In Bing sweet cherry, the pit pox strain tends to induce leaf

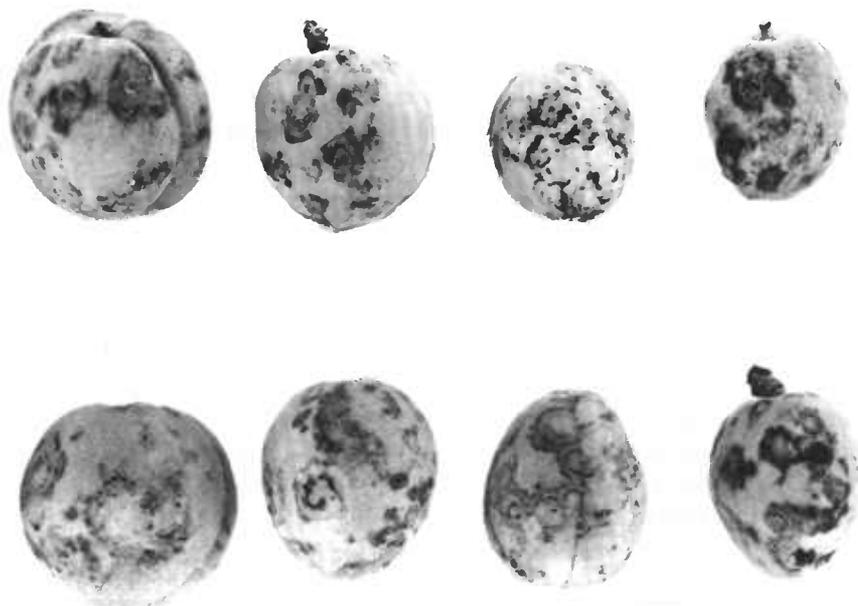


FIGURE 12.—Apricot ring pox. Discolored and necrotic rings and spots on Wenatchee apricot fruit.

twisting similar to that caused by the cherry twisted leaf virus. This strain has been found in Utah (B. N. Wadley, unpublished data), in the Yakima Valley and north of Wenatchee in Washington, and in British Columbia. Experimental transmission of the pit pox strain is more difficult than the common strain, and field symptoms vary more from year to year. A strain of ARPV reported from California (501) and Utah (B. N. Wadley, unpublished data) differs from common ring pox and from pit pox in causing leaf and fruit symptoms in Royal, which is a symptomless host of all other known forms of the virus. Isolates of the recently discovered spur cherry virus induce symptoms similar to those of apricot ring pox in Tilton (E. C. Blodgett, unpublished data).

Transmission

Common apricot ring pox is easily graft-transmissible to healthy apricot trees in budwood, scions, fruit, and leaf tissue (559). The pit pox strain is less easily transmitted; five to seven buds should be used in each index tree. Indicator trees infected through bud grafts usually develop symptoms the following year, although an incubation period of 2 years has been observed (83, 216). The virus is not seed-transmitted (151).

The pattern of natural spread of ARPV suggests that the virus is arthropod-transmitted (415) but no vector has been reported. Native chokeberry often is naturally infected, even in locations far from apricot orchards, and this host is the most likely source of infection in British Columbia.

ARPV has not been transmitted to herbaceous plants, has not been purified directly from *Prunus* tissues, and has not been detected with the electron microscope. Therefore, nothing is known about its chemical or morphological characteristics. The virus was not inactivated in *Prunus* material exposed to 38°C. for 21 days or to 50° C. for 20 minutes (P. R. Fridlund, unpublished data).

Control Measures

Although no spectacular regional outbreaks of apricot ring pox have been reported, the disease takes a constant toll in many western apricot-growing areas. Until the natural vector of ARPV is discovered, and an adequate control is established, the only means of preventing further spread of this disease is the removal of affected trees. The present program is complicated in areas where orchards are composed of mixed plantings of several apricot cultivars, or of apricots and sweet cherries, because some of them may serve as symptomless carriers of the virus. Trees infected with ARPV are commercially worthless if they are a cultivar that develops fruit pox. Even if they do not exhibit symptoms, they are reservoirs of the virus. In either case, infected trees should be removed. Known virus-indexed stocks and scions should be used for new plantings. Where native chokecherry occurs near apricot orchards, it should be removed.

Remarks

Diagnosis of apricot ring pox is made difficult by the varying degrees of symptom development caused by strains of ARPV and by symptoms caused by other viruses that closely resemble those of ring pox. *Prunus* ring spot virus

(516), peach mosaic virus (523), and pucker leaf virus (686) cause leaf symptoms in apricot which are almost identical to those caused by ARPV, but no fruit symptoms develop. Because these viruses may be present in apricot material suspected of carrying ARPV, it is essential to index for ARPV in a host combination that produces fruit symptoms regularly (e.g., Tilton or Wenatchee). Sarka (plum pox), a virus disease spreading at present in Europe, has symptoms similar to apricot ring pox in some hosts (528). It can easily be distinguished from apricot ring pox in Italian Prune, which has not become infected when inoculated with ARPV. Ring poxlike fruit symptoms have been found on plum in England (195) and on one apricot cultivar in Australia.

The relationship between ARPV and cherry twisted leaf virus (CTLV) has not been investigated sufficiently. Extensive work in British Columbia (414, 415) indicated that a close relationship exists between the two viruses. A large percentage of ARPV isolates were capable of inducing twisted leaflike symptoms in Bing sweet cherry and, conversely, CTLV isolates from cherry induced ring pox symptoms when budded into Wenatchee apricot. Similar results have been obtained in Tilton apricot with isolates from Lambert sweet cherry from the Kettle Falls area of Washington. The data available at present are not sufficient to determine whether one virus causes both diseases or whether two closely associated viruses are involved in this syndrome.

VIRUS DISEASES OF PEACH

ASTEROID SPOT

H. E. WILLIAMS, BRYCE N. WADLEY, and H. K. WAGNON

Causal Virus

Asteroid spot virus (ASV).

Synonyms

Utah Dixie rusty mottle; stipple spot (in Texas).

History and Distribution

Asteroid spot in peach trees was reported first from California in 1938 (161) although the disease had been observed in Texas and California prior to that time (162). Later the disease was recognized in peach in Colorado, Oregon, Utah, Washington (578), and Mexico (162). Two affected trees reported in North Carolina were traced directly to infected nursery stock originating in Texas (142). The virus is known to occur in native desert peach trees in Nevada and California (699). A disease of sweet cherries in the Dixie area of southern Utah was reported first in 1944 (573) and later described as Utah Dixie rusty mottle (583, 586). In 1949, what appeared to be a related disease was found generally distributed in peaches growing near the infected cherries (583). A virus, transmitted from these naturally infected peach trees, produced symptoms of Dixie rusty mottle in sweet cherry and asteroid spot in peach.

Economic Hosts

Peach, nectarine, and sweet cherry.

Other Hosts

Susceptible species are almond, desert almond, apricot, Japanese apricot, anzu apricot, mahaleb cherry, Manchu cherry, desert peach, European plum, Japanese plum, Damson plum, *Prunus bokhariensis*, *P. angustifolia*, and *P. spinosa* (162, 682, 699; and T. S. Pine, unpublished data).

Symptoms

Peach and nectarine. — This disease was named for the small, star-shaped spots that develop in infected peach leaves (fig. 13). Leaf symptoms appear early in the spring on fully expanded leaves and become more pronounced as the season progresses, particularly during periods of high temperatures. The spots begin as small, translucent, light-green flecks in the fully expanded leaves. Affected leaves gradually turn yellow, but the spots remain yellowish green, thus reversing the color contrast. Spots vary in size, the size being inversely proportional to the number present. Occasionally some of the larger spots have green centers and are thus really rings. In some leaves, large, angular, chlorotic



FIGURE 13.—Asteroid spot. Yellowish green angular or star shaped spots on yellowed peach leaves.

blotches form along the veins. The chlorotic spots sometimes become necrotic and drop out. The size, number, and character of the leaf spots varies among cultivars and between isolates of the virus. Progressive defoliation of affected trees often is encountered in the orchard, the older leaves dropping first. In severe cases, peach and nectarine trees may be almost completely defoliated by the end of summer. Affected trees show symptoms annually, but they are usually more severe during the first year of infection.

White-fleshed peach and nectarine cultivars tend to develop more pronounced symptoms than yellow-fleshed cultivars. No fruit symptoms have been reported but heavy defoliation seems to reduce the quality and quantity of fruit on affected trees.

The presence of strains of ASV has been suggested (162, 699). The isolate of ASV from desert peach (699) produces a much more severe reaction in peach (e.g., Lovell and J. H. Hale) than isolates collected from other sources

(699 and T. S. Pine, unpublished data). This isolate caused some dying of twigs on inoculated limbs of peach trees, a symptom not found after inoculation with other sources of the virus.

Sweet cherry.— Asteroid spot in sweet cherry was described originally as Dixie rusty mottle, but the symptoms are distinct from those induced by other viruses of the rusty mottle group. Symptoms vary considerably between cultivars of sweet cherries and are influenced by climatic conditions (high temperatures). The foliage appears healthy until 4 to 7 weeks after full bloom and then small necrotic spots begin to appear in the leaves. A few days after the appearance of leaf spots, many of the leaves begin to develop premature senescence, similar to that which occurs with the rusty mottle diseases. As the chlorophyll degenerates, islands of green remain on a background of yellow (fig. 14). The yellow leaves may be cast within a week, and partial to complete defoliation may result by mid- to late summer. Late fall symptoms differ from those produced earlier in that necrotic spots become larger and are surrounded by a halo of yellow tissue with a green or reddish-brown outer margin (fig. 15). Sometimes the necrotic spots are absent, but the yellow tissue and colored rings remain.

Lateral buds and small branches of susceptible sweet cherry cultivars are usually killed by ASV. Larger limbs also may be killed, and severely affected trees may die in 2 to 5 years. Necrotic areas often form in the flesh of maturing

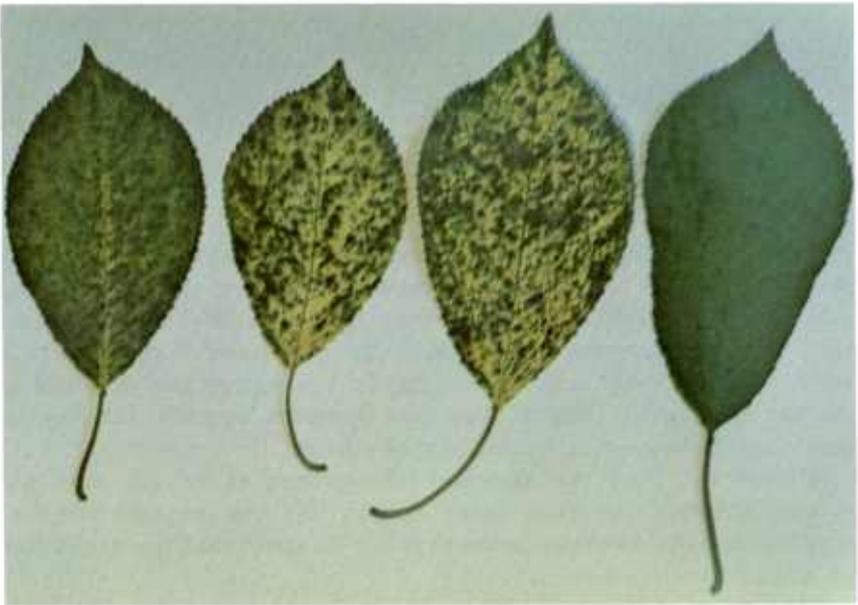


FIGURE 14.—Early season symptoms of asteroid spot on Lambert sweet cherry leaves—small necrotic spots and green islands on a yellowing background.



FIGURE 15.—Late fall symptoms of asteroid spot on Lambert sweet cherry leaves—large necrotic spots surrounded by halo of yellow tissue and outer green margin on a yellow background.

fruits (fig. 16). The percentage of deformed fruits may be high enough to cause complete loss of the crop. The heavy defoliation causes a reduction in fruit size, a delay in maturity, and a lowering of fruit quality. Both leaf and fruit symptoms in sweet cherry are most severe when summer temperatures are high.

All sweet cherry cultivars are hosts of ASV, although some are more susceptible to damage than others. Lambert, Bing, Deacon, and Van are more seriously damaged than Black Tartarian, Napoleon, or Windsor (682). Some mazzard seedlings develop symptoms whereas others do not.

Apricot. — Symptoms of asteroid spot in apricot vary with cultivar and strain of the virus, but they are essentially the same as those in peach. In a collection of foreign and domestic apricot cultivars, ASV caused only delayed foliation and retarded growth in many cultivars, but the virus was easily recovered in peach or plum 4 years after inoculation (T. S. Pine, unpublished data). Some cultivars in the collection underwent premature defoliation without displaying foliar symptoms whereas others, such as Tilton and Royal, developed symptoms as severe as those in peach and dropped most of their leaves by late summer. Some apricot cultivars (e.g., Moorpark) may not develop symptoms until the second year of infection.

The apparent presence of strains of ASV also can be detected in apricot. Isolates of the virus from naturally infected Santa Rosa plum trees caused only

mild symptoms in Tilton, but an isolate from an unknown peach cultivar growing in a desert area of San Bernardino County, Calif., caused very severe leaf symptoms and defoliation in Tilton (T. S. Pine, unpublished data).

Other hosts. — Symptoms caused by ASV in Japanese plums also vary with cultivar and virus strain but generally resemble those in peach (fig. 17). Defoliation is less common on plum than on peach and cherry. Symptoms in inoculated French (Agen) prune and damson plum were obscure. ASV may cause faint symptoms in almond but is known to remain latent in some cultivars. The virus is completely latent in both desert almond and desert peach.

Transmission

ASV has been transmitted only through tissue grafts. The incubation period varies from 4 weeks to 2 years, depending on the host cultivar and the time of inoculation. The occurrence of ASV in widely separated geographic areas and its recovery from native *Prunus* spp. is indicative of natural spread but no vector is known.

Control Measures

Nursery trees should be grown only from inspected and indexed source trees. Trees grown under provisions of official certification programs are highly



FIGURE 16.—Small necrotic spots and green islands on yellowing leaves and necrotic spots on maturing fruits of Napoleon sweet cherry affected by asteroid spot.

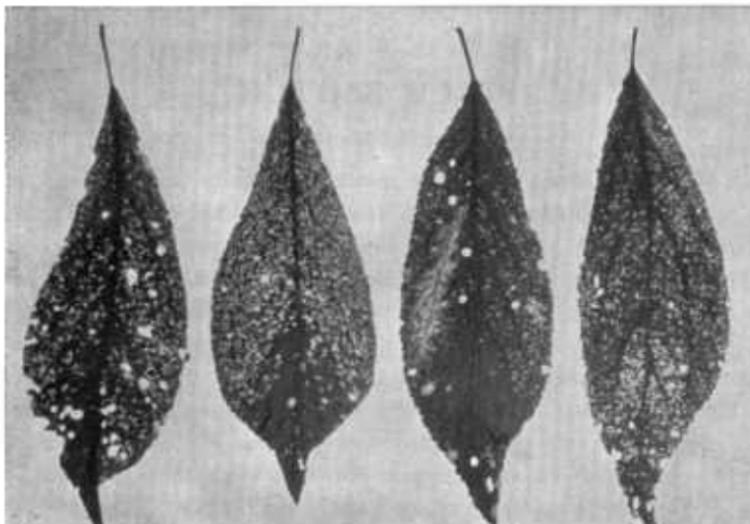


FIGURE 17.—Asteroid spot. Small chlorotic, angular or star shaped spots on leaves of Late Santa Rosa plum.

recommended as planting stock. Diseased trees should be removed promptly in those areas and orchards where infected trees occasionally appear. In areas where the disease is widespread, and severe forms of the virus cause extensive damage, attention should be directed toward the feasibility of an eradication program.

Remarks

Asteroid spot cannot be considered economically important although ASV occasionally causes the loss of fruit trees in limited areas. The more important losses, which are usually overlooked, result from reduced yield and poor quality of fruit from trees infected by mild forms of the virus. Leaf symptoms of asteroid spot may be confused with those of *Prunus* ring spot, plum line pattern, peach mosaic, apricot ring pox, and probably others, but the severe defoliation of index peach seedlings inoculated with ASV is diagnostic.

PEACH BLOTCH

CARL W. NICHOLS

Peach blotch is a virus disease of minor importance that has been reported from Ontario (730, 731), California (691, 694), Italy (168, 592), and possibly from Yugoslavia (592). It very likely occurs in many other peach-growing areas but has not been reported because of the minimal damage it causes.

The primary symptom of peach blotch is a well-defined, pale-green to yellow-green area in the leaves (fig. 18). These areas usually are large, irregular blotches scattered over the leaf blade, but smaller, angular spots may occur. Occasionally the chlorosis (light shades of green) is limited to bands along both sides of the midrib and basal portions of the main veins, or to one large chlorotic spot on one side or both sides of the midrib. While the margins of the chlorotic areas are usually well defined, sometimes they are "feathered out" along the lateral veins. The usual pattern of chlorotic blotches on a normal green background may be reversed, and dark-green islands occur on a chlorotic background. Both patterns may occur on the same leaf. The scorched margins and necrotic leaf spots reported in Canada (731) have not been observed in California. In California, the symptoms are usually limited

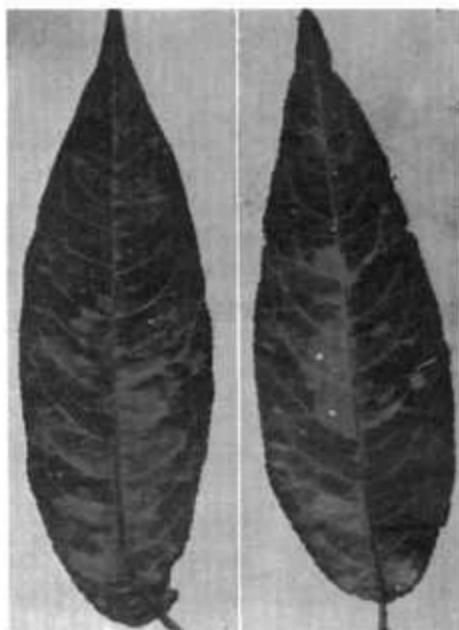


FIGURE 18.—Peach blotch. Pale green blotches on peach leaves.

to leaves that are produced during the early and late parts of the growing season (694). Mild symptoms of peach blotch have been detected late in August in the terminal leaves of nursery stock propagated in the spring of the same year with buds from virus-infected peach trees (691).

Peach is the only known host of this virus. Disease symptoms have been observed in many peach cultivars in the orchard and have been produced on others following graft inoculation. Apparently all peach cultivars that are infected by the virus will develop symptoms, but the intensity of the symptoms may vary widely (730, 731).

PEACH CALICO

H. KEITH WAGNON, JACK TRAYLOR, and HAROLD E. WILLIAMS

Causal Virus

Peach calico virus (PCV).

Synonyms

The term "peach calico" was first applied to peach variegation, a nonvirus disorder, but later was reserved for use with peach calico virus disease (55).

History and Distribution

In the United States, the disease is known to occur only in California (689, 694), Idaho (55, 71), and Washington (554). It also has been reported in Australia (506), Bulgaria (671), and New Zealand (132). Although PCV is widely distributed, its incidence always has been low.

Economic Hosts

Peach and nectarine are the only known hosts. All cultivars of these fruits are believed to be susceptible.

Symptoms

Symptoms of peach calico occur on the leaves, on stems of current year shoots, and on the fruit. The most conspicuous symptoms are those found on leaves, particularly those which develop during the spring and early summer. Occasionally, certain shoots will produce symptomatic leaves throughout the growing season (fig. 19). Usually, an affected twig will have both affected and non-affected leaves.

The first leaf symptoms usually appear as the leaves unfold, and consist of a mottle or mosaic pattern of green, pale green, yellow, cream, and white. As the affected leaves age, the pale-green areas change to creamy yellow and even papery white. The areas of the leaf lacking green pigmentation may follow along each side of the midrib, often forming a symmetrical pattern. However, in many instances, the pattern is spectacular in its nonuniformity. In extreme situations all, or the greater part, of some individual leaves may be completely devoid of green pigment. These leaves frequently become necrotic along their margins during periods of hot weather.

Characteristic stem symptoms usually are confined to twigs having leaf symptoms. Creamy white streaks or blotches, often with pink or reddish margins, occur on stems of seriously affected shoots.

Fruits on affected branches may begin to show symptoms when they are less than an inch in diameter. These symptoms are irregular patches of creamy white or bleached tissue showing through the pubescence. As the fruit matures, the affected areas may become orange or reddish. Affected fruits are smaller, shorter, and more nearly round than healthy fruit.

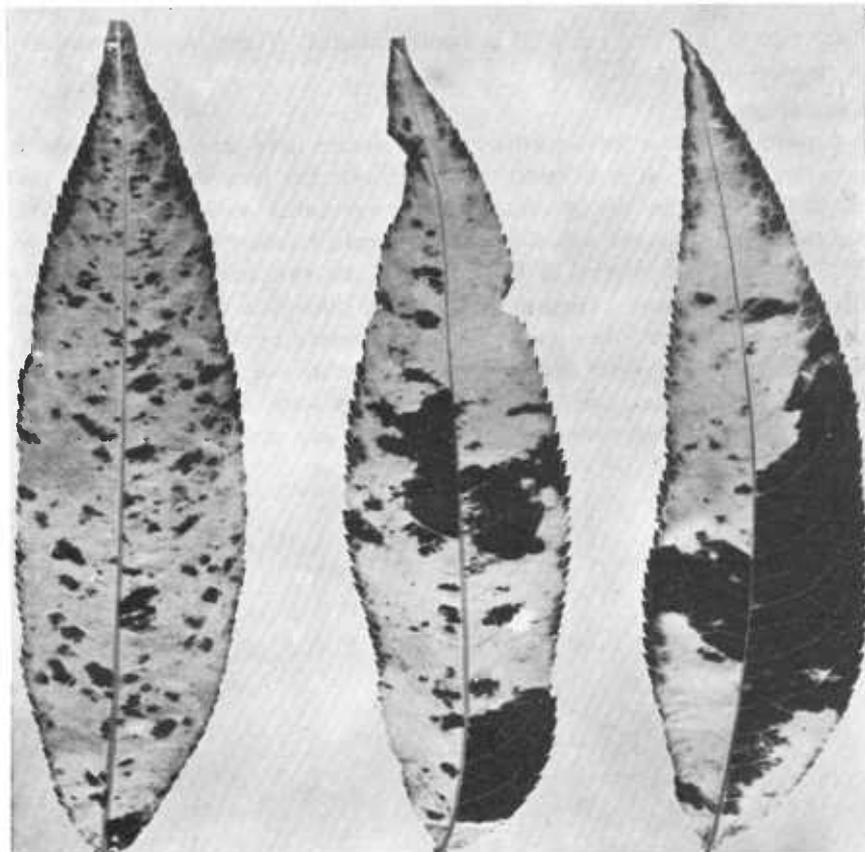


FIGURE 19.—Peach leaves affected by peach calico, showing mosaic patterns of pale green, yellow, cream, and white.

Limited inoculation tests have failed to induce symptoms in Manchu cherry and the following cultivars: Bing sweet cherry, Montmorency sour cherry, May Duke semisweet cherry (*Prunus avium* × *P. cerasus*), Moorpark apricot, Italian Prune, and Ne Plus Ultra and Peerless almonds.

Information on virus morphology and serological relationships is lacking. Heat treatments in which growing plants were maintained for 40 days at 99° F. had no apparent effect on the virus (132).

Transmission

Transmission of PCV has been accomplished only by grafting. Because the virus moves very slowly in a tree, evidence of transmission is best attained by the forcing of dormant or adventitious buds near the inoculum.

Control Measures

The potential hazard is in the propagation of nursery stock with infected scions. Once PCV invades the apical meristem of a shoot, all subsequent

shoot growth from that meristem is usually infected. Therefore it is important to use virus-indexed budwood.

Remarks

Limited studies on the occurrence of the disease have provided no evidence of natural spread. It is believed that the disease has been increased and distributed through the use of contaminated propagating materials. The erratic occurrence of symptoms indicates that there may be more than one strain of PCV. Mature trees infected by PCV, for the most part, have not been severely affected by the disease. Because PCV spreads through a tree at such a slow rate, natural spread of the virus in an orchard would be difficult to detect and would not be of economic importance. The similarity of symptoms and occurrence of affected twigs in orchard trees causes some confusion with other diseases, such as peach variegation, peach blotch, and almond calico.

PEACH MOSAIC

T. S. PINE

Causal Virus

Peach mosaic virus (PMV).

History and Distribution

In 1931, peach mosaic was found simultaneously in peach trees in Texas (322) and Colorado (76, 77). Subsequently the disease was discovered in peaches in Arizona, Arkansas, California, New Mexico, Oklahoma, and Utah, and in the States of Baja California, Chihuahua, and Coahuila of the Republic of Mexico (325). Today it is economically important only in western Colorado and southern California. It has been confused with peach calico, sarka, and other plum virus diseases in Europe (528), but there is no substantiated report of its occurrence outside the United States and Mexico. The widespread incidence of peach mosaic in the Rio Grande Valley indicates that it has been present there for a long time (325).

Economic Hosts

Peach and nectarine.

Other Hosts

The virus has been transmitted to peach from naturally infected almond, tangut almond, apricot, ansu apricot, Japanese apricot, Manchu cherry, David peach, flat peach, American plum, Bokar plum, beach plum, chickasaw plum, damson plum, European plum, flatwoods plum, hog plum, hortulan plum, Japanese plum, Mexican plum, myrobalan plum, and wildgoose plum. Western sand cherry and Korean cherry (not true cherries) and Klamath plum can be infected experimentally and will develop characteristic symptoms. PMV was recovered from desert almond, desert apricot, and sweet cherry when these species were grown on infected rootstocks but could not be recovered if they were grown on their own roots or nonsusceptible rootstocks (82, 148, 158, 325).

Peach mosaic virus has not been found infecting, or has not been transmitted to, fenzl almond, capollin cherry, bitter cherry, black cherry, hollyleaf cherry, mahaleb cherry, pin cherry, sand cherry, sour cherry, sweet cherry, western chokecherry, oriental flowering cherry, or *Prunus kansuensis*.

Peach mosaic virus was successfully inoculated into and recovered from 269 cultivars and 62 numbered selections of peaches and nectarines (158). Most freestone cultivars are severely damaged but clingstones tolerate the virus with little injury. Based on symptoms produced in J. H. Hale, peach cultivars can be divided into three comparative classes. The most important commercial cultivars are thus classified, in table 4, according to the relative severity of their reactions to a severe strain of PMV. Forty-two horticultural plum cultivars

TABLE 4.—*Classification of commercially important peach and nectarine cultivars according to the relative severity of their reaction to peach mosaic virus*

Severely damaged cultivars	Moderately damaged cultivars	Slightly damaged cultivars
Alamar	Babcock	Andora
Ambergem	Cardinal	Carolyn
Blake	Coronet	Cortez
Dixired	Early Babcock	Gaume
Earlihale	Early East	Halford No. 2
Earlired	Jerseyland	Johnson
Elberta	Keystone	Nemaguard
Fay Elberta	Merrill Fiesta	Paloro
J. H. Hale	Redglobe	Peak
Le Grand (nectarine)	Redskin	Phillips
Loring	Richaven	Springtime
Maygold	Redtop	Stuart
Merrill 49'er	Southland	Sullivan No. 1
Ranger	Sunhaven	Sullivan No. 2
Redcap	Suwanee	Sullivan No. 4
Redhaven	Wiser	Suncrest
Regina		Vivian
Rio Oso Gem		
Sunhigh		
Triogem		

and 103 apricot cultivars tested were found to be hosts for at least one strain of PMV, but not all susceptible cultivars developed foliar symptoms (78, 526).

Symptoms

Peach.—Diagnosis of peach mosaic often is difficult because the virus exists in many forms which vary in virulence, and because peach cultivars differ in tolerance to the virus (325). Symptoms of peach mosaic consist of blossom break, retarded foliation, mottling and deformity of leaves, fruit deformity, and abnormal tree growth. Symptoms in some cultivars may be so mild that they are almost masked. Some virus cultures cause a pronounced shock stage during the first year of infection, whereas others show no shock the first year but increased symptom development the second year (520). In subsequent years, affected trees usually enter a chronic stage of infection during which symptoms are present in less severe forms (143).

Affected peach cultivars that normally produce large, solid-pink blossoms (e.g., Fay Elberta, Rio Oso Gem) often show an unusual color break in the petals. Irregular streaks, spots, or lines of white break up the pink so that the petals appear mottled (fig. 20). The affected petals sometimes become crinkled and dwarfed. This symptom can be confused with a similar color break in peach



FIGURE 20.—Petals from peach tree infected with peach mosaic virus, showing color breaking.

petals caused occasionally by the *Prunus* ring spot virus. The number of geminate buds produced on trees infected by PMV is substantially increased (514).

Leaf growth and new twig growth on affected trees is retarded during the spring and early summer. The degree of retardation depends on the virus form present and may involve the whole tree or only one or two branches (fig. 21). Many of the early leaves are shed, but those formed later remain on the tree. The retarded condition becomes less pronounced as the growing season progresses until, by late summer or autumn, the tree presents an almost normal appearance. Reduced twig growth (shortened internodes) leads to a stubby growth habit with the leaves crowded together to form rosettes (fig. 22). Some severely affected trees are seriously dwarfed.

An important symptom of peach mosaic is the development of variously shaped chlorotic patterns in the leaves during the spring and early summer. These discolorations differ in size and form from tiny flecks to bold blotches, streaks, or vein feathering (fig. 23). Severely affected cultivars may show all these patterns at the same time. Cultivars infected with a mild strain of PMV, or very tolerant cultivars, may show only vague veinlet clearing or yellow flecking in the leaves. Leaf patterns of peach mosaic appear in both the early formed leaves, which tend to drop, and those formed later. Many leaves on severely affected trees are small, narrow, crinkled, or irregular in outline. Chlorotic areas frequently become necrotic and fall out, resulting in a leaf shot



FIGURE 21.—Peach mosaic. Retarded growth on part of a severely affected peach tree.

hole. As the season progresses, the foliage of affected trees becomes darker green, and the leaf patterns become less distinct. The color change to darker green is part of the normal development of the peach mosaic syndrome (326) and probably results from high summer temperatures (119).

Many peach cultivars, particularly freestones, develop severe fruit symptoms that make the fruit unmarketable. The surface becomes rough or bumpy (fig. 24) when fruit on diseased trees has exceeded an inch in diameter or at the stone-hardening stage. The bumps appear as raised areas of normal tissue surrounded by depressions caused by failure of tissue to develop. Frequently, the suture area is deformed. As the season advances, the bumpy character of the fruits intensifies; the size of affected fruits fails to increase as rapidly as normal fruits. Occasionally, affected fruits lose some of the bumpiness just prior to maturity, but they never reach normal size or shape. Fruit of cultivars that do not develop such severe symptoms tend to ripen several days to a week or more later than healthy fruit. The flavor of affected fruit is only slightly inferior, but the reduced size and irregular shape make it unmarketable.

Plum.—Horticultural plum cultivars are highly susceptible to PMV but only occasionally are they found naturally infected (526). Symptoms in cultivars of *Prunus domestica* (fig. 25) and *P. salicina* usually are the same types found in

peach. Infected trees rarely show delayed foliation, dwarfing, or fruit injury. Leaves of some affected trees are deformed but not as seriously as those of peach trees. The severity of leaf symptoms in plum tends to remain more constant, year after year, than in peach.

Apricot.—Natural infections of peach mosaic occur more often in apricot than in plum. Symptoms in most apricot cultivars are less severe than those in peach, but some may be severely affected (fig. 26). The more sensitive cultivars develop the same leaf patterns, leaf distortion, and short internodes associated with peach mosaic in peach. Trees severely infected by PMV tend to be less vigorous, produce less fruit, and have a higher percentage of sunburned fruit than healthy trees.

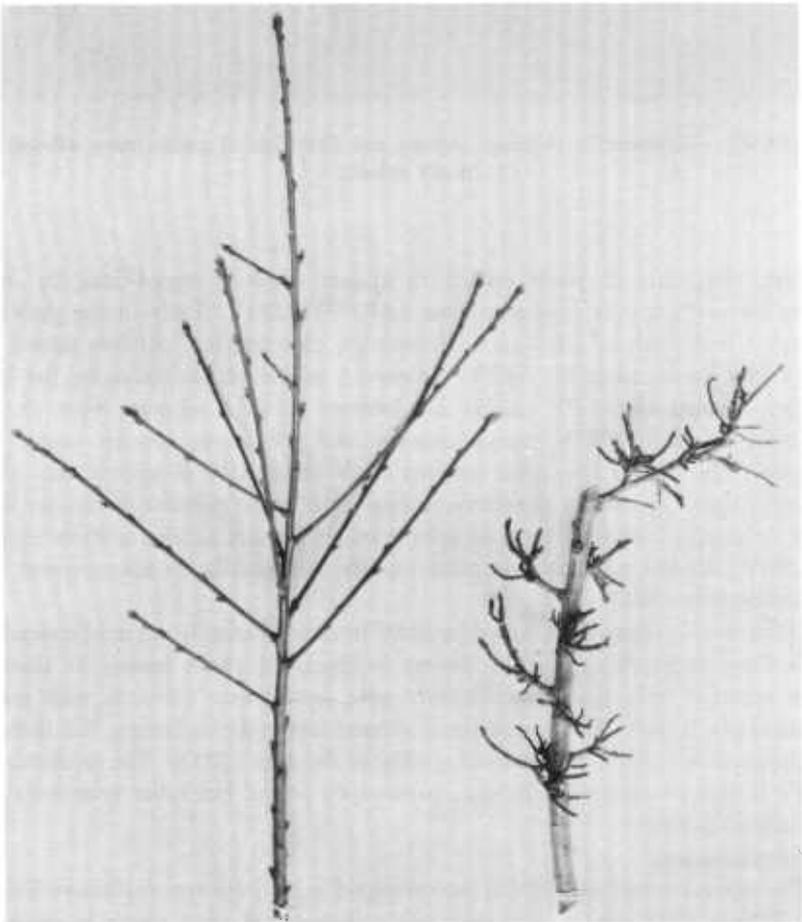


FIGURE 22.—Peach mosaic. Stubby twig growth on affected branch, *right*; normal growth on healthy branch, *left*.

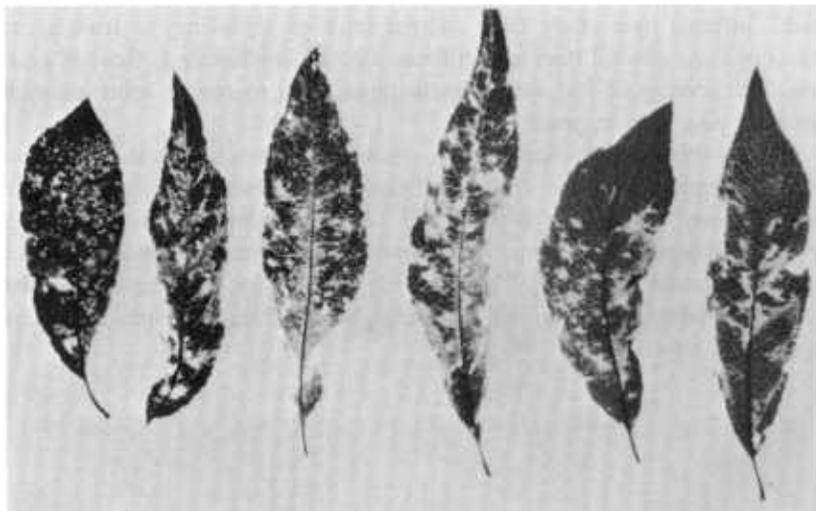


FIGURE 23.—Variations in chlorotic spotting and distortion of peach leaves affected by peach mosaic.

Field diagnosis of peach mosaic in apricot often is complicated by dual infections with apricot ring pox virus (ARPV) (523). Early in the growing season it is difficult to distinguish between the chlorotic leaf patterns caused by PMV and those caused by ARPV. However, as the season advances, the leaf patterns caused by ARPV redden and become necrotic, whereas those caused by most strains of PMV remain constant and the leaves become cupped or twisted. There is no synergism between PMV and ARPV in apricot trees with respect to severity of leaf symptoms or fruit pox. Some cultures of *Prunus* ring spot virus cause mosaic patterns in apricot leaves that are similar to those caused by PMV, but the patterns fade more rapidly and usually do not reappear the following year (516).

Other hosts.—Symptoms caused by PMV in almond usually are mild compared with those in peach (fig. 27). Severe outbreaks of peach mosaic in almond have occurred only when the almonds were grown near orchards with many infected peach trees. Fruit on diseased almond trees may be bumpy, but there is no appreciable reduction in yield or quality of the nuts (325). The incidence of PMV is high in native *Prunus* spp., particularly plums, but foliar symptoms are not always present.

Transmission

The natural vector of PMV is the eriophyid mite *Eriophyes insidiosus* Keifer and Wilson (351, 747). The mite was collected first from peach in southern California and subsequently from several *Prunus* spp. in California and in other States where PMV is present. Range of the vector, however, does not coincide

with the present range of peach mosaic; the vector has been collected from flowering peach trees as far north as Madera County in California's Central Valley, 200 miles outside the limits of the known range of PMV. It has been found in Georgia, Indiana, and Mississippi, where peach mosaic has never been observed. The vector feeds and reproduces beneath the closely adhering leaf-bud scales, seeming to prefer the retarded or stunted buds that are commonly found on peach trees late in the summer. However, the densest populations are found in April in buds that have not yet expanded. On native plums, the mite has been found not only under the scales of dormant or retarded buds but also reproducing in leaf axils and among rudimentary leaves of expanding buds on vigorously growing shoots. Bud retardation is characteristic of peach mosaic in peach, hence the persistence of the vector is favored in diseased trees. A single mite is capable of transmitting PMV from diseased to healthy peach seedlings. Transportation in air currents is probably their means of dissemination.

PMV apparently spreads whenever the foliage is present and green; i.e., from late March to October (335). When trees are infected before the leaves are expanded in the spring, either by the vector or by artificial means, symptoms usually develop in 21 to 30 days; if infection takes place later in the year, symptoms do not appear until the following spring (326). Natural spread of PMV readily occurs from peach to peach and sometimes from peach to apricot and

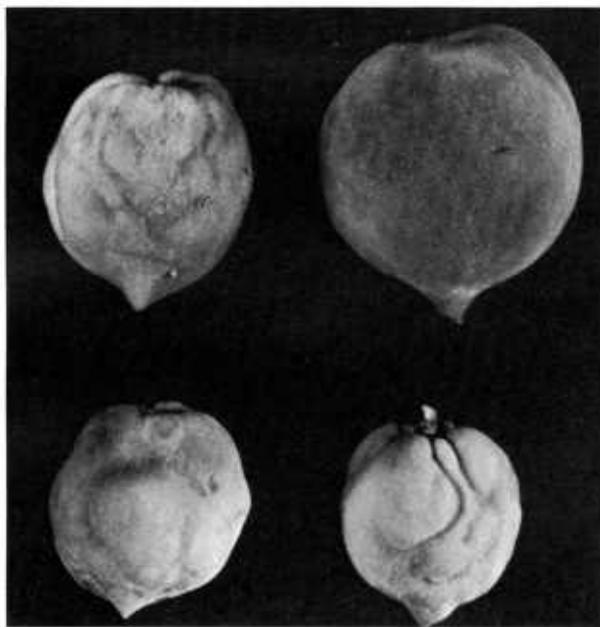


FIGURE 24.—Fruit from peach trees affected by peach mosaic, showing reduced size, bumpy surface, and uneven color development.



FIGURE 25.—Chlorotic spotting and distortion of leaves of French (Agen) prune affected by peach mosaic.

almond but rarely from apricot or almond to peach (163). This is probably because the vector travels to hosts other than peach or plum and feeds on them for short periods but does not colonize (reproduce) on them.

PMV has not been mechanically transmitted in expressed juice. The virus has been transmitted in tissue grafts, using diseased fruit, leaf pieces, shootwood and bark, and rootwood and bark (159). The virus is not transmitted by dodder (32).

Because PMV has not been transmitted to any herbaceous plant and has not been purified from *Prunus* tissue, nothing is known about its chemical or physical properties. Based on differential symptoms in *Prunus* spp., cross-protection, and consistent recovery from natural infections, it has been possible to show that definable strains of PMV occur in nature (81, 144, 149, 154, 155, 160, 327, 520). The "hairy break" or mild strain of PMV has maintained its identity through 25 years of research on peach mosaic. It provides good protection against reinoculation as long as the challenging severe cultures of PMV are obtained from peach trees, but it fails to protect completely against cultures derived from other *Prunus* spp., especially plum (both *P. domestica* and *P. salicina*) (79, 160, 520). The peach severe strain of PMV produces symptoms in plum but not in almond or apricot. The plum strain, originally obtained from French prune and Kelsey plum, causes symptoms in almond, apricot, and peach.

Control Measures

Peach mosaic has been contained within relatively limited areas since 1935 through a program of diseased-tree removal conducted by the Plant Protection

Division, USDA, in cooperation with the States, counties, and pest control districts concerned. Infected areas are under Federal-State quarantines, and tree removal is coordinated with nursery inspection to prevent spread of the disease to noninfected areas. The use of tolerant peach cultivars in areas where roguing of infected trees is used as a control measure is undesirable because of greater difficulty of diagnosis, especially where the milder virus strains are present. On the other hand, tolerant cultivars are desirable in areas where roguing is not feasible and peach growers must operate in the presence of the disease.

Peach mosaic virus was not inactivated by heat treatment (385). Chemotherapy of peach trees infected by PMV has been attempted on a limited scale without conclusive results (522).

Remarks

Because native plums seem to be the preferred host of the PMV vector and because isolates of the virus from plums have a wider host range than those from other *Prunus* spp., it seems plausible that this disease probably originated in the native plum species of the Rio Grande Valley and "escaped" to its economic hosts, peach and nectarine. Long-range dissemination of PMV and its vector from southwestern Texas (or northern Mexico) probably was in cuttings of native *P. americana* and *P. munsoniana*, both valued for their fruit. Local natural spread then took place wherever the vector and the virus were present. The contagious nature of peach mosaic and the efficiency of the vector are shown by the fact that only seven diseased trees were found in Colorado in 1931, but 30,467 were known to be diseased in 1935. During the first 5 years of the tree-removal program in California, 204,193 diseased peach trees were destroyed.

Peach mosaic virus has seriously limited the culture of many popular freestone cultivars (e.g., J. H. Hale and Elberta) in the southwestern United States. The

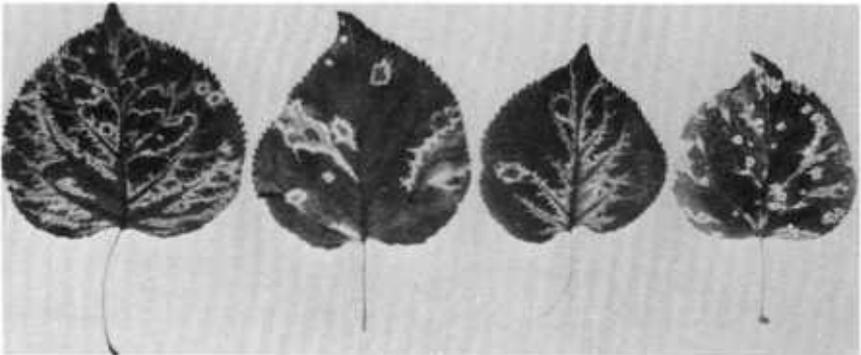


FIGURE 26.—Chlorotic patterns in leaves of Royal apricot affected by peach mosaic.

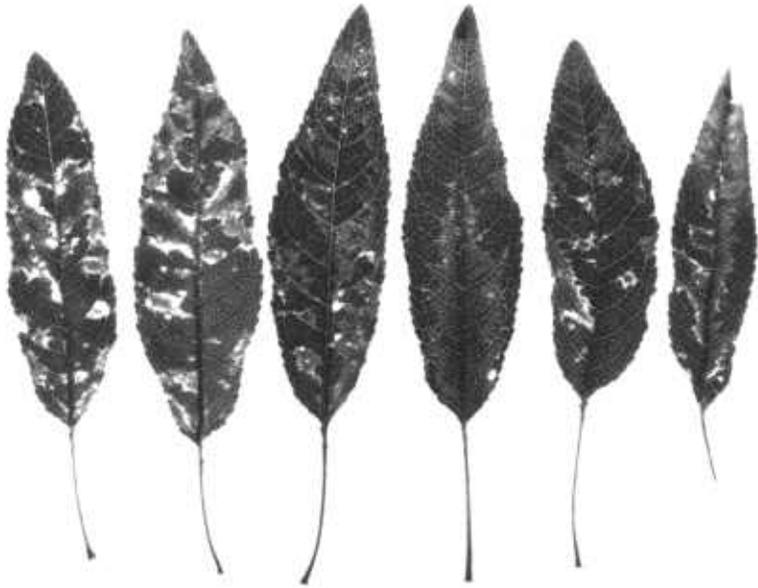


FIGURE 27.—Chlorotic spots and patterns in leaves of Ne Plus Ultra almond affected by peach mosaic.

more tolerant cultivars, on which marketable fruit can be produced, are often an economic threat to the industry in this region because of their ability to act as reservoirs of virus for spread to susceptible cultivars. Control of the virus vector, combined with diseased-tree removal and continued vigilance in the selection of virus-free budwood for propagation, should continue to reduce the incidence of peach mosaic.

PEACH MOTTLE

A. W. HELTON

Peach mottle was found in a few Elberta peach trees in Idaho between 1939 and 1943. The causal virus was transmitted by grafting to peach cultivars J. H. Hale, Elberta, Slappey, and peach seedlings; to sweet cherry cultivars Bing, Napoleon, Lambert, Black Republican, and mazzard seedlings; to sour cherry cultivar Montmorency; and, to May Duke cherry (61). Trees inoculated in the fall usually showed symptoms of peach mottle the following spring. No natural spread of the virus has been reported, and the disease is of minor economic importance.

Leaves of affected trees are slightly dwarfed and coarsely mottled, with the margins tending to roll and pucker (fig. 28). Naturally infected trees are ragged in appearance and generally lighter in color than healthy trees. Growth is reduced but not enough to cause rosetting. Foliar symptoms in artificially infected peach trees are similar but vary markedly in severity among cultivars. No symptoms of peach mottle have been noted on fruits or blossoms, but infection at an early age often results in stunting or death. Foliar symptoms tend to fade as the growing season progresses.



FIGURE 28.—Peach leaves showing mottling and puckering caused by peach mottle virus.

Symptoms in artificially infected sweet cherry trees range from severe leaf mottle and necrosis, shoot necrosis, and tree stunting in Bing, to a chlorotic veinlet clearing in Lambert. Symptoms in Napoleon resemble those in Bing but are less severe. Symptoms in Black Republican are similar to those in Lambert but are somewhat more severe. Peach mottle virus in Montmorency sour cherry causes variable leaf mottling, twig dieback, and formation of gum pockets on the twigs, which are sometimes accompanied by stem swellings or cracks in the bark. Infected May Duke trees produce leaf mottling and vein clearing, stunting, and dieback of terminal shoots.

Infected trees should be removed. Only disease-free propagating materials should be used for replanting.

Peach mottle virus induces severe symptoms in Black Republican and Montmorency and is therefore distinguishable from the cherry mottle virus which produces only mild symptoms in these hosts. Peach mottle is distinguished from peach mosaic in that peach mosaic virus does not infect cherries.

PEACH ROSETTE

GLENN KENKNIGHT

Causal Virus

Peach rosette virus (PRV).

History and Distribution

Peach rosette was observed first in Georgia in 1881 (613, 614). Subsequently, it was found in Alabama, Arkansas, Illinois, Indiana, Kansas, Kentucky, Mississippi, Missouri, Oklahoma, South Carolina, Tennessee, Texas, and West Virginia (423 and author's unpublished data).

Economic Hosts

Peach; Japanese plum and plum hybrids; and wild plum species (*Prunus angustifolia*, *P. hortulana*, and *P. munsoniana*) and hybrids.

Other Hosts

Almond, apricot, flowering almond, damson plum, western sand cherry, David peach, wild plums (*Prunus mexicana* and *P. injucunda*), Manchu cherry, mazzard cherry seedlings, mahaleb cherry, sour cherry, sand cherry, common chokecherry, and red maple. Tobacco (*Nicotiana glutinosa*), tomato (*Lycopersicon esculentum*), and periwinkle (*Vinca rosea*) have been experimentally infected.

Symptoms

Peach.—The development of early spring symptoms in affected trees is both rapid and striking. Leaves on part or all of a diseased tree are normal in size but yellow and either folded inward or arched backward. Occasionally, the first symptoms are the reddening of leaf veins and early defoliation. New shoot growth has extremely short internodes. As leaves are formed on this growth, they are appressed into distinct rosettes (fig. 29). Leaves on older portions of the tree are shed by early summer, leaving only tufts of young leaves near the tips of naked shoots. Blossoms on affected branches rarely develop or set fruit. Most affected trees die during the first year of infection. Occasionally, an affected tree will leaf-out the following spring but it dies quickly.

Plum.—Japanese plum cultivars and hybrids (e.g., Kelsey Ogon [Botan], Maynard, and Red June) are very susceptible to natural infection by PRV. Rosetting is less conspicuous than on peach, and affected trees may live 2 to 3 years after the appearance of symptoms. Some diseased plum trees are noticeably stunted. Inoculated damson plum trees develop symptoms similar to those in Japanese plum. An Italian Prune tree (*Prunus domestica*) inoculated with one source of PRV developed symptoms similar to those seen in Japanese plum, but other Italian Prune trees inoculated from other virus sources showed no signs of infection. The virus was transmitted from peach to Green Gage plum and back to peach without the plum becoming permanently infected.



FIGURE 29.—Elberta peach tree affected by peach rosette showing severely rosetted growth of all shoots.

Wild plum.—*Prunus angustifolia* is the principal native plum host of PRV. Leaves on affected portions of trees often are larger than healthy leaves and tend to become reddish rather than yellow. Rosetting of terminal leaves is much less noticeable than on peach; shortened internodes are less prevalent, and there is less premature defoliation. Most affected trees die the year of infection; only a few live more than one full year. Naturally infected *P. injucunda* is known in only one location in Georgia (360). Some affected seedlings of this species die quickly while others become stunted and live up to 5 years. They produce dense green foliage in the spring and the leaves turn red in summer. Some clones of *P. injucunda* are symptomless carriers of PRV.

Other hosts.—Symptoms in almond are similar to those in peach (613). Shoots of affected Royal apricot are weak and have shortened internodes, but rosetting is not as severe as on peach. Growth may be stunted, and the leaves often develop a yellow-green mottle. On Moorpark apricot, only stunting and leaf mottle may appear (423). Wilson apricot develops very mild symptoms, but there is a tendency toward proliferation of shoots on the main branches which results in a witches'-broom effect (661). Leaves on affected trees are slightly yellow and the petioles turn red. Some apricot seedlings, however, may show very severe symptoms after inoculation with PRV.

The growth of mazzard cherry trees infected by PRV is markedly reduced, and the leaves are smaller and more rolled than healthy ones. The leaves are

yellowish and tufted, as on peach. Terminal buds fail to become dormant (425). Symptoms resembling those of peach rosette were seen in seedlings of sour cherry trees growing near infected peach (660). Affected sand cherry trees develop a witches'-broom growth on the young shoots, and the leaves are smaller and lighter green than those on healthy plants, but the trees are not killed (425). In Georgia, when scions of an eastern chokecherry tree were grafted to a diseased peach tree, they developed marked symptoms of peach rosette, but similar scions of Bing and Black Tartarian sweet cherry, Montmorency sour cherry, *Prunus serotina*, *P. mahaleb*, and *P. capuli*, did not develop symptoms (author's unpublished data).

Red maple trees, naturally infected by PRV, were found in Georgia (360). The trees had developed shortened internodes, resulting in dense, rosetted foliage. On periwinkle, PRV causes rosetting similar to that on peach. The virus causes wilting and death of rapidly growing tomato and tobacco plants (390).

Peach rosette virus has not been purified. The virus in budsticks was inactivated by hot water treatment, for 8 to 10 minutes at 50° C. (387) and in bark patches for 18 minutes at 50° C. (359). The virus is known to move downward more rapidly than upward in diseased plants and will not pass a bark ring (358). Neither peach yellows virus (386) nor phony virus protects peach trees from infection by PRV, and no synergism between these viruses has been observed. During host range studies, it was shown that apparently there are three forms of PRV: one that causes overt symptoms in Marianna plum, a second that infects Marianna plum without causing symptoms, and a third that will not infect Marianna plum (author's unpublished data). The virus apparently was inactivated in some peach seedlings by immersing the roots for 4 hours in 5 to 25 ppm of several tetracycline compounds (357). Similar treatments with other antibiotics were not successful.

Transmission

Natural spread of PRV from wild plums to commercial plums and peaches is well known although no vector has been identified (425). Attempted transmissions with sap and insects were unsuccessful (424). The virus can be transmitted through tissue grafts from diseased to healthy peach or plum (615). A bud-contact period of 8 to 14 days is necessary for transmission of PRV from peach to peach (388). A single bark patch graft (3 cm. sq.) from diseased plum or peach resulted in 80 percent transmission to 1-year-old peach seedlings but seven patches the same size were needed to give 20 percent transmission to 3-year-old peach trees. The virus was transmitted through dodder (*Cuscuta campestris*) to periwinkle, tomato, and tobacco (390). The minimum incubation period of PRV in Burbank plum, approach grafted to diseased peach, was 3 weeks but for transmission between diseased red maple trees and healthy Methley plum (360), was 3 years.

Control Measures

Eradication of infected orchard trees and wild *Prunus* growing near orchards, as soon as symptoms are observed, has proved effective. If diseased trees are allowed to remain in the orchard, peach rosette has been observed to reach epidemic proportions, affecting whole orchards, in 3 to 5 years. Even under these conditions, natural spread can be reduced to a few trees within 1 to 2 years by prompt diseased-tree removal in the spring (425).

Remarks

The presence of large populations of native *Prunus* hosts, particularly *P. angustifolia*, which act as reservoirs of virus for spread to peach and plum, continue to make the disease a threat to industry. Occasionally, winter-killing of peach and cupping of leaves due to aphids on wild plums have been mistaken for peach rosette. Zinc deficiency in peach is sometimes called rosette although the symptoms do not resemble closely those caused by PRV.

Because PRV seems to be inactivated by certain antibiotics (357), peach rosette possibly may be caused by one of the mycoplasmlike organisms and not by a virus. This opinion has significance because peach rosette is one of the yellows-type diseases that is not mechanically transmitted in expressed juice, is inactivated during hot-water treatment, spreads rapidly in the field, and is readily transmitted through dodder. However, it is not known to be transmitted by leafhoppers. Until more information is known on the subject of mycoplasmas in plants, peach rosette will continue to be classified as a virus disease.

PEACH STEM PITTING

SRECKO M. MIRCETICH and H. W. FOGLE

Causal Virus

Peach stem pitting virus (PSPV).

History and Distribution

In 1960, Christ (140) reported a new peach disorder in New Jersey and attributed it to scion-stock incompatibility, although the disorder later was identified as peach stem pitting (470). In 1966, Barrat (23) reported the occurrence of an obscure disorder characterized by wood pitting in young orchard and nursery peach trees. The authors (unpublished data) have transmitted peach stem pitting from diseased to healthy peach trees in the greenhouse and nursery by means of bud- and root-patch grafts.

Pitting of stems of *Prunus* spp. other than peach has been reported from British Columbia (419), Maryland (469, 470), Pennsylvania (631), and Oregon (113). The syndrome, as it occurs in British Columbia, is described under "Xylem aberration" (412).

Field surveys in 1967 and 1968 established the presence of stem pitting in peach in New Jersey (140), West Virginia, Pennsylvania, Maryland, Virginia (24, 396, 470), Ohio (334), Delaware (M. Sasser, personal communication), North Carolina (C. N. Clayton, personal communication), South Carolina (192), Michigan (A. L. Jones, personal communication), and California (J. H. Weinberger, personal communication).

Economic Hosts

Peach stem pitting has been observed in 26 commercially important peach cultivars and in peach seedlings (24, 140, 192, 396, 470). The same disease was observed in naturally infected nectarine trees (470).

Symptoms

Peach trees affected by PSPV have the general appearance of being girdled (fig. 30). The terminal growth is brachytic. Leaves droop, curl upwards lengthwise, and may be chlorotic. In late summer, leaves may become increasingly chlorotic, often turning red or purple in the fall. Intensity of leaf color may vary with the stage of disease, cultivar and climatic conditions during the growing season. Unfolding of leaf buds in the early spring is delayed, but leaf abscission in the fall occurs earlier than on comparable unaffected trees. Fruits on affected trees may have insipid flavor, fail to size properly, be more highly blushed than normal, and ripen and drop prematurely.

Marked enlargement of the trunk occurs at ground level or below. Occasionally, trunk enlargement in younger trees does not extend beyond the root transition zone. This enlargement usually is noticeable as soon as foliar symptoms are recognized and becomes more marked as the disease progresses. The



FIGURE 30.—Peach stem pitting. *A*, Four-year-old peach seedling showing typical drooping leaves with upward rolled margins; *B*, comparable healthy peach seedling.

enlarged portion has very thick, spongy bark that is often two to four times the thickness of bark in a comparable unaffected tree (fig. 31, *A*, *B*). The thick bark is usually detected at or below ground level. Removal of the bark from the affected lower trunk reveals pits and grooves accompanied occasionally by enations and necrotic areas within the cambium (fig. 31, *C*). Corresponding ridges of inner bark are usually imbedded in the wood. Considerable variation in symptom expression, particularly in the extent of pitting, enation, and cambial necrosis, is found between cultivars—for example, Redhaven and Redskin develop severe pitting, enations of assorted sizes, and necrosis of the vascular cambium in the enlarged trunk areas (figs. 32, 33), whereas Elberta develops short and very shallow pits in the wood. Only two cultivars, not yet positively identified, among 26 surveyed developed neither pitting nor disoriented woody tissue, even though the rootstock was severely affected (fig. 32, *C*). However, these two cultivars showed foliar symptoms. It has not been determined that these cultivars are symptomless carriers.



The wood pitting appears first below the ground level, then gradually spreads into the roots and short distances into the aboveground trunk. The tissue within the woody cylinder of the affected lower trunk may be highly disorganized and may have wide xylem rays and inclusions of large phloem islands (fig. 33). Lignification of the xylem cells appears to be incomplete, resulting in a weak woody cylinder. Trees with a high proportion of such weakened tissue break easily at ground level, exposing highly disorganized tissue in the woody cylinder (fig. 34). The tendency to break is directly related to a high proportion of disorganized woody tissue. The trees which are prone to break show fewer normal annual growth rings than disorganized ones in the woody cylinder. These trees are usually very stunted and have poorly developed root systems. Affected peach trees that have developed a large number of normal annual growth rings followed by a few disorganized ones (fig. 34, *B*), have excellent



FIGURE 31.—Trunk symptoms in peach infected by peach stem pitting virus: *A*, Diseased trunk—note thick bark (*tb*) and initial stage of wood pitting (*p*) extending to the ground level (*gl*); *B*, healthy trunk—note normal bark (*nb*) and smooth woody cylinder; *C*, pitting in woody cylinder accompanied by necrosis of vascular cambium (*nvc*).



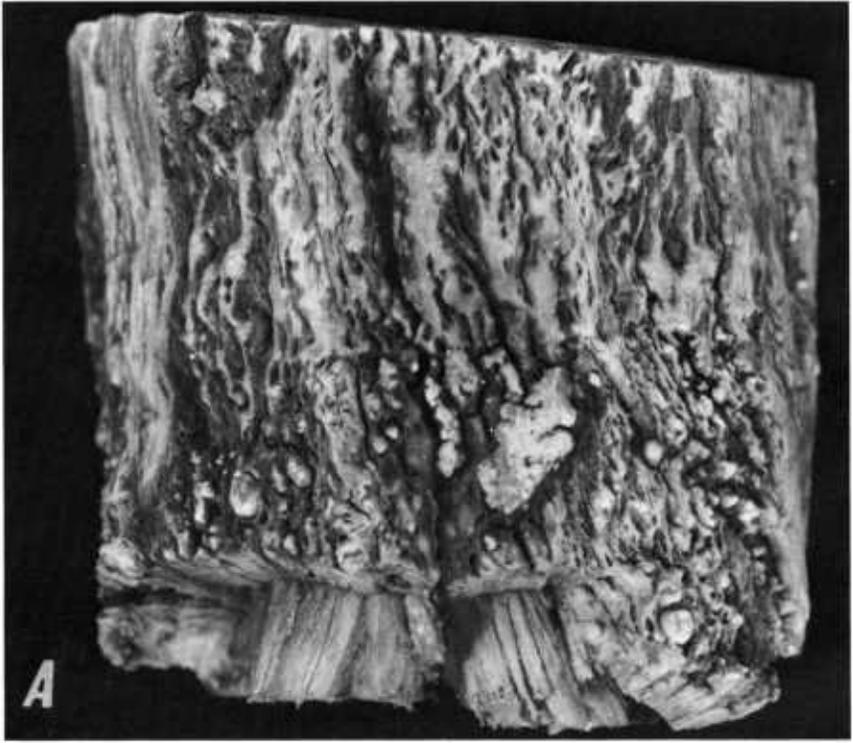
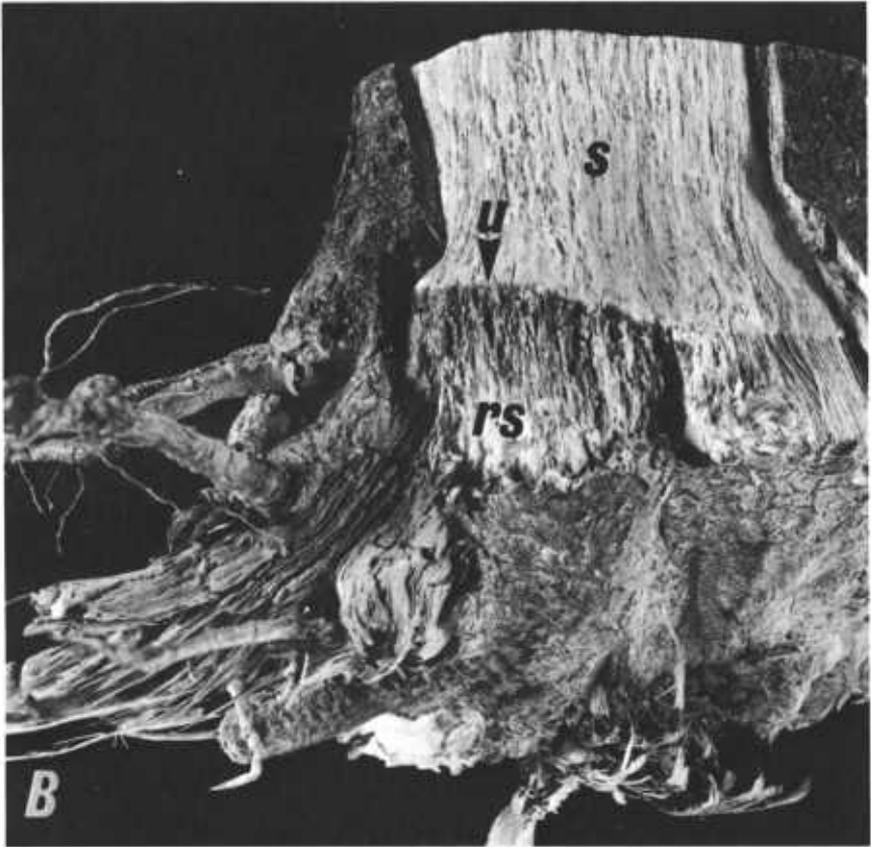


FIGURE 32.—Trunk portions of 3 peach cultivars affected by peach stem pitting, showing variation in symptom expression: *A*, Seven-year-old Redhaven tree with severe wood pitting and enation; *B*, 20-year-old Elberta tree on undetermined rootstock—note mild pitting on the scion (S) and distinct line of the union (U) formed by differential pitting of the scion and rootstock (RS); *C*, 3-year-old tree of an unidentified cultivar—note the scion portion (S) is free of pitting, whereas the rootstock (RS) is severely pitted.

anchorage. Lateral roots are usually pitted when they develop from an affected trunk (fig. 34, *C*). These roots may separate easily from the trunk, creating a socketlike depression. Feeder roots of affected trees are usually decayed. The severity of feeder root necrosis is determined by the stage of the disease. Foliar and lower trunk symptoms in naturally infected nectarine trees are similar to those in naturally infected peach trees.

Transmission

PSPV has been transmitted from infected peach to peach seedlings through tissue grafts and field soil (authors' unpublished data). Elberta and Sunhigh seedlings, inoculated by chip buds and root patches from naturally affected peach trees, developed stem pitting in the greenhouse within 18 months and



in the nursery within 11 months. Natural spread was observed in a densely planted, 5- by 15-foot peach seedling orchard. Typical spread occurred within the row from infected trees to adjacent trees 5 feet away. The number of infected trees doubled within 1 year. However, the mode of spread and possible vectors are not known.

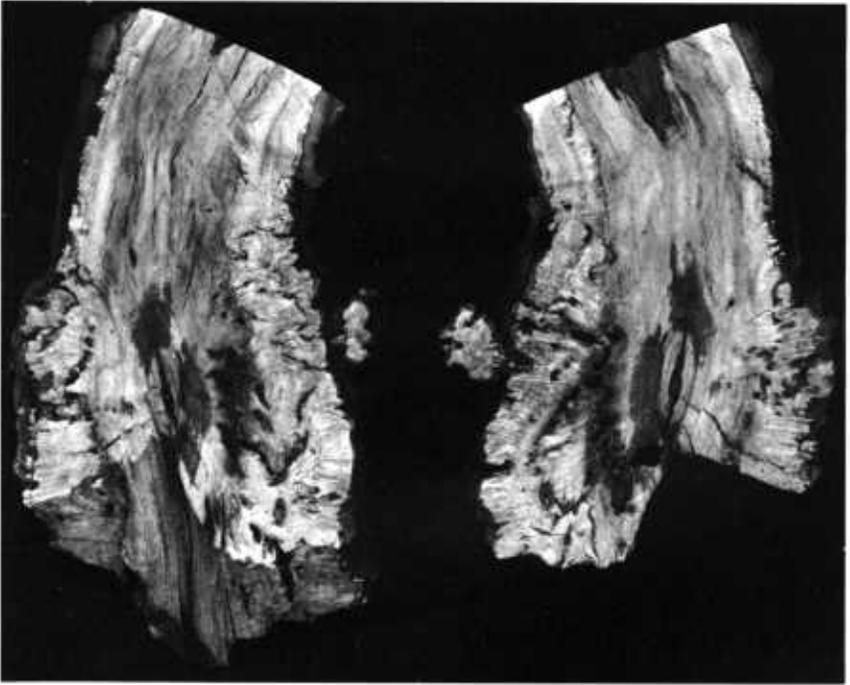


FIGURE 33.—Longitudinal section through lower trunk of 7-year-old Redskin tree severely affected by peach stem pitting. Note wood enations of various sizes and large phloem inclusions within disorganized woody cylinder.

Control Measures

Infected trees should be removed promptly, and only budwood and seed from healthy trees should be used for propagation. Good cultural practices may be helpful in combating this disease. Because a disease similar to peach stem pitting is found in many *Prunus* spp., eradication of wild *Prunus* in the vicinity of commercial orchards is advisable.

Remarks

Severe incidence of this disease in certain commercial peach and nectarine orchards in southern Pennsylvania, Maryland, and West Virginia indicates that stem pitting may become a limiting factor for peach production in these areas. In some surveyed orchards, planted in 1966 and 1967, 12 to 25 percent of the trees were affected by the disease. In several plantings of 4- to 13-year-old trees, over 75 percent of the trees showed typical symptoms of the disease. Affected peach trees become commercially worthless. Younger trees may die within 2 or 3 years, but older trees may continue to produce fruit of inferior quality for some time. Recovery of affected trees has not been observed.

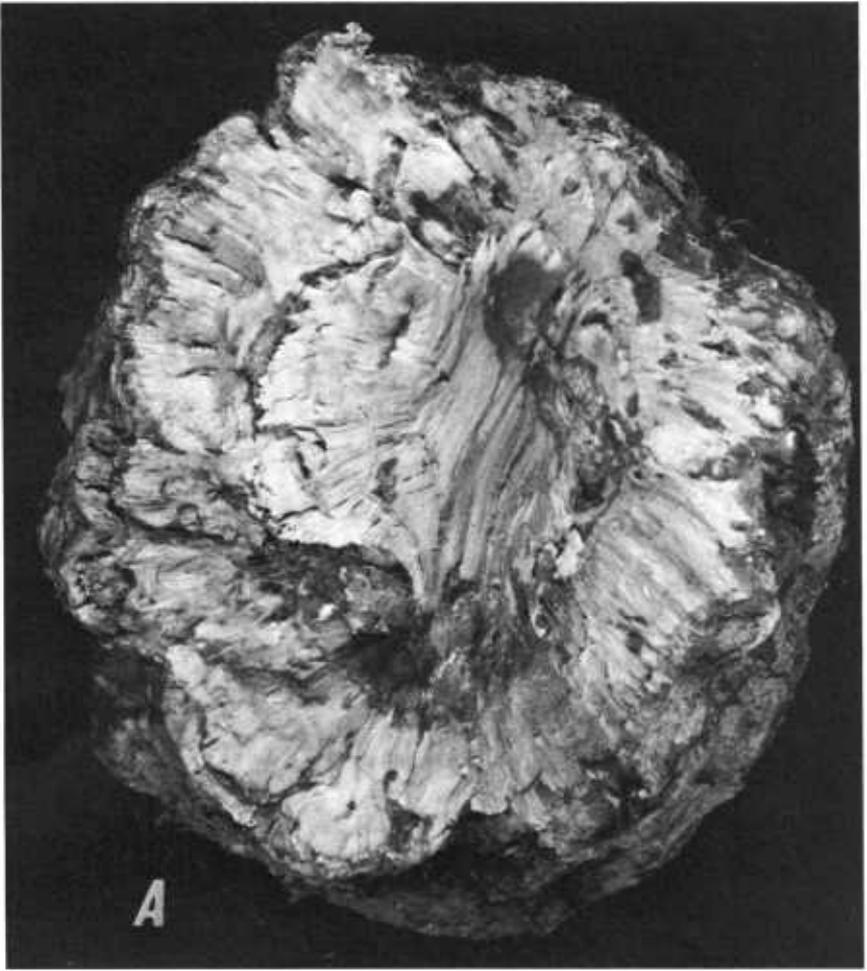
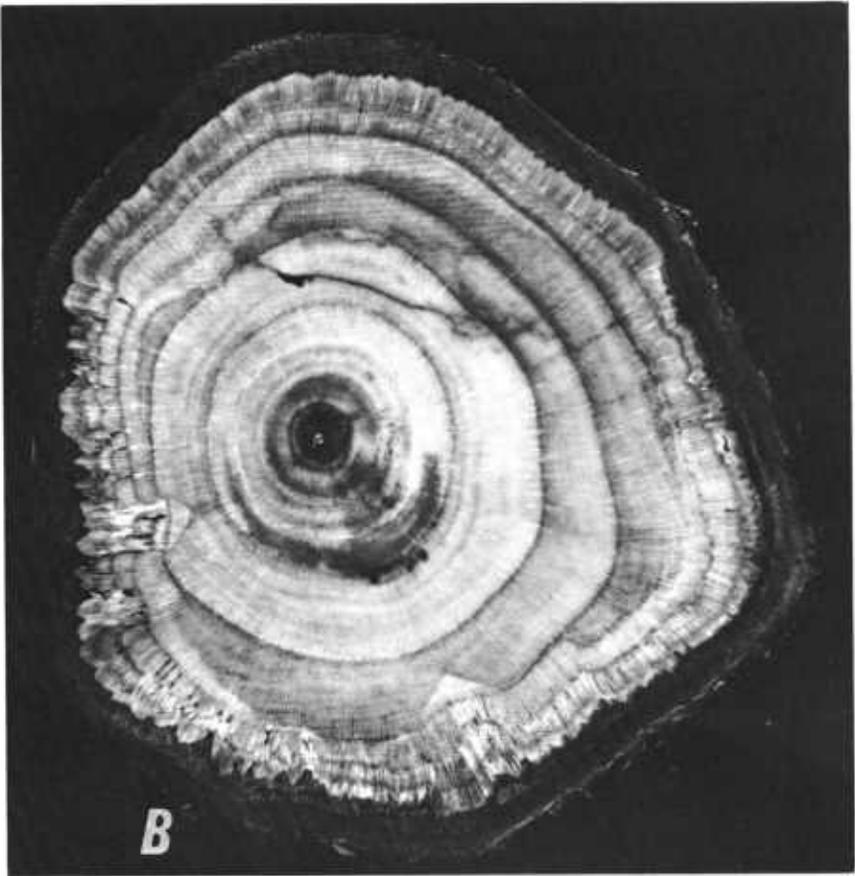


FIGURE 34.—*A*, Seven-year-old Redskin peach tree, in an advanced stage of peach stem pitting, broken off at enlarged area of affected lower trunk. Note highly disorganized woody tissue throughout the trunk. *B*, Cross section through affected trunk of 7-year-old Redhaven peach tree. Note that only outer annual growth rings have disorganized xylem tissue. *C*, Root of peach tree affected by peach stem pitting. Note thick bark, pitted woody cylinder and outer annual growth rings composed of disorganized xylem tissue.



See legend on p. 85.

A disorder resembling peach stem pitting has been observed in a number of *Prunus* species other than peach. The following *Prunus* spp. and interspecific hybrids are known to be affected by various forms of wood pitting in nature: sour cherry seedlings and Montmorency and North Star (419, 469); sweet cherry seedlings and Windsor (R. F. Stouffer, personal communication) and Bing (419); Shiro-fugen flowering cherry (631); mahaleb cherry (469); European plum Stanley, Late Smith and NY H-2 selection (469), Bradshaw (631), and Italian Prune (113); Japanese plum, Abundance, Santa Rosa (631) and Burmosa (469); apricot, open-pollinated seedlings of several cultivars (419, 469); cherry plum-sloe plum hybrid seedlings (469); Chinese wild peach, open-pollinated seedlings (469); hybrid Japanese plum, Redglow (469), Shiro, and Wickson (631). In some naturally infected European plum trees on myrobalan plum, the rootstock portion was without any pitting, whereas the scion was severely affected (469).

PEACH WART

EARLE C. BLODGETT

Causal Virus

Peach wart virus (PWV).

Synonyms

This disease was first called blister (46) but usually it is known as wart or peach wart.

History and Distribution

In 1939, Blodgett (46) described a peach disease present in Idaho that was first recognized in 1938. In 1951 (70), it was recorded in Idaho, Oregon, and Washington. Since then it has been reported in California (635) and New Mexico (139). It has not been noted in any other country. Wartlike symptoms have been observed on specimens of peach fruits from many areas of the United States, but these probably were due to effects of environment, insects, or other factors.

Economic Hosts

Peach (*Prunus persica*).

Other Hosts

The virus may be transmitted by bud inoculation to sweet cherry cultivars Black Republican, Royal Ann (Napoleon), and Lambert (769). Fridlund (220) tested a wide range of possible indicators including Bing, Lambert, and Sam sweet cherry; Kwanzan and Shiro-fugen flowering cherry; Montmorency sour cherry; Tilton apricot; and Shiro plum; plus 64 cultivars and selections of peach and nectarine. All were infected by PWV.

In a later paper, Lazar and Fridlund (393) gave evidence that Shiro plum, when inoculated with PWV, exhibited characteristic and abundant leaf symptoms after 4 weeks when grown under temperature-controlled greenhouse conditions. Fridlund (personal communication) stated that seedlings of Manchu cherry (*P. tomentosa*), when used in the greenhouse, are also regarded as good indicators for PWV.

Symptoms

The first symptoms in peach trees appear on the very young fruits, shortly after the calyxes have fallen, as bleached bumps or raised welts on or near the styler end and often involve half or more of the fruit (51, 554). The fruits are both dwarfed and misshapen if the disease is severe, but slightly affected fruits are nearly normal in size. The surface of affected tissue varies from light tan to dull red and may be rough with warty outgrowths conspicuously raised, or it may be rather smooth (fig. 35). The fruits of some cultivars less severely affected exhibit a cracked and russeted appearance. Gum is usually present and

may be abundant. The warty tissue is rather superficial, but the underlying tissue is coarse and filled with gum pockets. In some cases, the warty tissue is very hard and bonelike, but usually it is tough and leathery. Frequently, the warts occur in ringlike patterns near the styler end.

In the study of peach wart, certain variations were found and described by such names as "smooth wart," "crease wart," and "beady wart." In tests by Blodgett, it appeared that smooth wart was transmissible as such, that crease wart was not caused by a virus but was associated largely with certain cultivars (Rio Oso Gem in particular), and that beady wart was probably caused by insects. Typically, there are no leaf symptoms and infected trees show no reduction in size, vigor, or production.

Black Republican, Napoleon, and Lambert sweet cherries, when infected experimentally, show leaf mottling in variable degrees, a tendency to dwarfness, and dieback. Other *Prunus* spp. and cultivars (see "Other Hosts") tested by Fridlund (220) showed that Bing and Sam sweet cherry, Tilton apricot, and Shiro plum produced mild symptoms, whereas Lambert sweet cherry, Kwanzan and Shiro-fugen flowering cherry, and Montmorency sour cherry did not, although all were carrying the virus. The 64 peach and nectarine cultivars were rated according to symptoms and placed into six groups ranging from no symptoms to very severe. None of the four nectarine cultivars showed fruit symptoms, and none of the 64 cultivars in the test showed foliage symptoms. Fridlund reported that clingstone peaches showed only mild fruit symptoms.



FIGURE 35.—Peach fruits affected by peach wart, showing warty outgrowths on immature fruit.

Transmission

Peach wart virus may be transmitted by conventional budding, branch approach grafting, and by "budding" 1-year-old nursery tree trunks with wedge-shaped pieces of infected fruit tissue (56). Inoculation in the fall may produce fruit symptoms the next spring.

In tests by Fridlund (222), PWV was transmitted from infected inoculum buds to peach within 90 to 110 hours contact at 26° C. This time interval was in his rate II group which, besides wart, included cherry mottle leaf, cherry twisted leaf, apricot ring pox, green ring mottle, and Napoleon latent viruses.

A limited number of tests indicates that the virus is not seed transmitted (65, 219) and no natural vector is known. However, healthy trees may become diseased naturally in orchards (E. C. Blodgett, unpublished data).

Normally, to obtain proper index readings, fruit must be observed on the peach indicator. Lazar and Fridlund (393) determined that Shiro plum is a useful indicator for this virus, and, through its use, the testing time can be greatly reduced. Fridlund (personal communication) regarded seedlings of *Prunus tomentosa* as good indicators.

Control Measures

Diseased trees should be removed as soon as identified, and budwood for propagation should be taken only from known healthy trees.

Remarks

Peach wart is rare, but the potential threat of spread is ever present. Fruit from infected trees is of no value.

PEACH YELLOWS

T. S. PINE and R. M. GILMER

Causal Virus

Peach yellows virus (PYV).

Synonyms

Little peach and probably red suture (p. 133).

History and Distribution

Yellows first appeared as an unknown malady of peach trees on the farm of Judge Richard Peters near Philadelphia in 1791 (513). The disease was described in some detail by William Prince in 1828 (542). The history and distribution, and much of the early research on peach yellows, was reported by E. F. Smith during 1888–94 (612, 614, 616). Although severe outbreaks of the disease occurred during the 19th and early 20th centuries, it has been of minor importance in recent years (437). Peach yellows has been found in the Atlantic Coastal States and the Appalachian States, from Massachusetts to the Carolinas, and in Ontario, Canada, and southward through Michigan, Illinois, Indiana, Kentucky, and Tennessee. It has never become established west of the Mississippi River or in the South.

The little peach disease, described first by Taft in 1896 (641), is encountered more often today than peach yellows. It is found in approximately the same area as peach yellows and on the same hosts (109, 117).

Economic Hosts

All cultivars of peach and nectarine, apricot, and almond.

Other Hosts

All *Prunus* spp. that have been tested were found susceptible to infection, including: American plum, David peach, myrobalan plum, European plum, dwarf flowering almond, hortulan plum, Japanese apricot, wildgoose plum, Japanese plum, and Manchu cherry. The plum cultivars Abundance, Chalco, and Chabot (*P. salicina*), and myrobalan rootstocks are symptomless carriers of the little peach strain (435).

Symptoms

Peach yellows.—In early spring, the leaf buds of an affected tree unfold prematurely, and many normally latent buds fail to remain dormant. Leaves produced from these buds are dwarfed and yellow. The tree rapidly develops many slender, branched, willowy shoots that grow upright from the main limbs, thus giving the tree a bushy appearance (fig. 36). Leaves on the abnormal shoots are narrow, chlorotic, and develop red spots; they roll inward and tend to droop downward. Terminal dieback of twigs and branches is common in advanced stages of the disease. Severely affected trees die 2 to 3 years after the onset of

symptoms, but those with mild infections may live 6 years or longer. Inoculated greenhouse trees develop vein-clearing in the tip leaves, and the leaves tend to droop or become sickle shaped. Such trees usually die 1 to 2 years after infection.

Fruit produced on trees infected by PYV matures several days to 3 weeks before healthy fruit and may be larger than normal (fig. 37). The fruit is of poor quality, usually with a bitter taste (43). In cultivars that normally develop



FIGURE 36.—Peach tree affected by severe peach yellows, showing dense type of growth and small leaves.

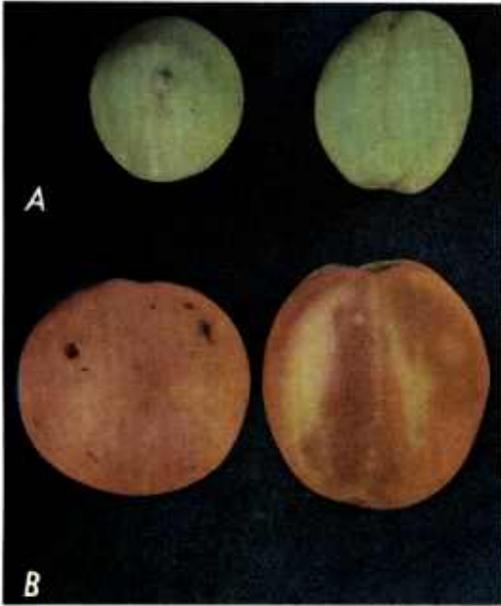


FIGURE 37.—Peach yellows. *A*, Normal immature fruits; *B*, comparable fruits affected by peach yellows, showing early ripening.

red coloring in the skin and around the pit, the skin is abnormally highly colored, spotted with red and purple, and the flesh is streaked or marbled crimson with a pronounced red coloring around the pit (392).

PYV causes symptoms in apricot, almond, American plum, hortulan plum, David peach, almond cherry, and Manchu cherry that are the same as those produced in peach. In Abundance plum, German prune, wildgoose plum, and Japanese apricot the symptoms are the same but milder than those in peach. Some cultivars of plums and plum hybrids are symptomless carriers of the virus.

Little peach.—Infected peach trees have distinctly greener foliage than healthy trees during the early part of the growing season and appear more compact than normal. Proliferation of leaves on short lateral branches or spurs, produced on 2- and 3-year-old wood, leads to a bushy appearance. The branches are more upright than on healthy trees but are not as long and willowy as those on trees with peach yellows. Leaves on affected trees are noticeably leathery in texture and tend to droop or roll downward; they become progressively chlorotic as the growing season advances but do not develop the red spotting common on trees with peach yellows. Symptoms are usually evident first on a single branch or portion of a tree but spread to the whole tree within 2 or 3 years. Fruit produced on affected trees matures several days to 3 weeks after healthy fruit and is reduced in size. The pits are small and the kernels are either underdeveloped or fail to germinate. The fruit generally has an insipid flavor. Symptoms on susceptible plum and apricot cultivars are similar to those on peach but usually are milder.

Designation of little peach virus as a strain of PYV is based on cross-protection; peach trees infected with little peach are immune from infection by PYV, and trees with peach yellows are immune from infection by the little peach strain (386, 387). However, exceptions to this pattern have been recorded, in which the fruit on one branch of a diseased tree matured earlier (peach yellows), whereas fruit on another branch matured later (little peach) than fruit on healthy trees (43). When healthy bearing trees are inoculated simultaneously near the growing points with one bud infected with PYV and the other with the little peach strain, the yellows symptoms appear first but the little peach symptoms eventually become dominant (435). When peach seedlings are simultaneously inoculated vertically in sequence with buds from both sources, the bud placed in the top position determines the disease syndrome (388).

Peach yellows virus, including the little peach strain, is inactivated by treatment with hot water—dormant trees by immersion for 10 minutes at 50° C. and buds by immersion for 3 to 4 minutes at 50° (387). Because the virus has not been purified, nothing more is known about its physical or morphological characteristics.

Transmission

PYV and the little peach strain are transmitted through tissue grafts or by the plum leafhopper (*Macropsis trimaculata* Fitch) (384, 388, 434, 614); they have not been transmitted mechanically in expressed juice, are not pollen transmitted, and are not seedborne (612). The incubation period of the diseases in field trees is usually 1 to 3 years but is less than 60 days in greenhouse plants (388, 435). The virus moves downward more rapidly than upward in infected trees (383). Hence, inoculations near the top of a tree cause symptoms to appear much earlier than infections occurring at or near the ground level. The incubation period of the virus in the leafhopper vector varies from 7 to 26 days, with an average of 16 days (282, 283). Because plums are the preferred host of the plum leafhopper, and because some plum cultivars are symptomless carriers of PYV, it has been assumed that natural spread of these diseases is largely due to transfer of the virus from plum to peach in the vector (436).

Control Measures

Diseased trees should be removed as soon as they are discovered. Because some plums and plum hybrids are either symptomless carriers of the virus or develop only mild symptoms, they should not be planted near peach orchards. Control of the plum leafhopper is essential.

Remarks

Although peach yellows and little peach are not economically important today, the cyclic nature of these diseases in a large peach-growing region suggests that growers and agricultural technicians should be continually aware of their presence and prepared to take appropriate action should the disease incidence increase or the vector become prevalent.

Designation of peach yellows and little peach as virus diseases is based on the best evidence gathered by the workers who originally described them. These investigators concluded that a virus was the causal agent because no other pathogenic entity could be implicated and because transmission could be obtained only through tissue union or by means of an insect vector. Reevaluation of the whole group of "virus yellows" diseases, exemplified by the aster yellows disease, suggests that peach yellows may result from infection by one of the pleuropneumonia-like organisms (531, 608). However, until more evidence is available on this subject, peach yellows and little peach will continue to be classified as virus diseases.

PHONY

L. C. COCHRAN and LEE M. HUTCHINS

Causal Virus

Phony virus (PV).

Synonyms

In Georgia, the name "phony peach" was sometimes used by growers.

History and Geographic Distribution

Phony was first observed in two peach trees at Marshallville, Ga., about 1885 by Samuel Rumph, who discovered and introduced the Elberta peach. The dwarfed specimens were called "phony trees" by Rumph and were regarded as a curiosity. Their number increased, and, by 1915, thousands of peach trees were affected in a limited district in central Georgia. Illustrated papers calling attention to and describing the disease appeared in 1920 (482) and 1928 (318).

Research into the cause of phony was begun in 1921 by the Bureau of Plant Industry, USDA. A fairly exhaustive, well-illustrated account of the research and other information was published in 1933 (323). Additional information may be found in other publications of that period (320, 321).

In 1928, phony was conclusively shown to be caused by a virus (319). At that time the disease had spread over a large part of central Georgia and a smaller area in eastern Alabama. A Federal quarantine was promulgated in 1929 which restricted the movement of peach and nectarine nursery stock and peach roots from affected districts. Also, in 1929, a Federal-State cooperative program for systematic survey and removal of phony-diseased peach trees was inaugurated. By 1932, about 35 million orchard peach trees had been inspected. The disease was found in all the South Atlantic States below Virginia, in all the Gulf States, in Tennessee and Arkansas, and in a few locations in the southern parts of Oklahoma, Missouri, and Illinois (672). Subsequent surveys added isolated cases in Indiana, Kentucky, Maryland, and Pennsylvania. The disease has now been eradicated from these last four States and from some other States in the lightly affected area. The disease has not been positively identified elsewhere in this country or abroad.

Economic Hosts

All cultivars, races, and hybrids of peach (*Prunus persica*), including peach trees propagated on rootstocks of other species, such as almond, apricot, Japanese apricot, David peach, and hortulan plum.

Other Hosts

Seedlings of apricot, Japanese apricot, chickasaw plum, and Mexican plum. Trees of all the abovenamed species on their own roots are also susceptible to experimental infection through root grafts with root pieces from phony-diseased

peach trees. Peach is generally used as the test plant for verifying the presence of phony virus in the other species.

Symptoms

The symptoms of phony are most striking when the trees are in full foliage and in good vigor. Affected trees develop shortened internodes, profuse lateral branches and flattened dark-green leaves, producing a compact appearance of dense, luxuriant foliage. Decided dwarfing of new growth results and the periphery of the upper crown tends to take on a uniform, rounded appearance (fig. 38). Phony trees may live many years. However, after several years the wood becomes brittle, and there may be notable dying back of the terminal twigs and branches. Vigorous, new terminal growth cannot be forced. As large limbs die or are broken down, they are not replaced and the tree takes on a ragged appearance. Even on such trees, the foliage continues to be greener and flatter, characteristic of the disease. In spring, phony trees flower and leaf out earlier than normal ones of the same cultivar, and they hold their foliage later in the fall (fig. 39).

The fruits on phony trees are well formed and ripen a few days earlier than healthy fruits, but they are reduced both in size and in number per tree (fig. 40). They may be somewhat poorer in flavor than normal fruits, but they are generally more highly colored. Seeds from phony peaches average smaller than normal seeds, but they give excellent germination and produce normal seedlings.

Infection by PV does not induce spindling growth or rosetting; does not cause yellowing, rolling, or spotting of leaves; and does not induce lesions, galls, or hypertrophies.

Identification of the disease in the affected tree is made from the aspects of the tree as a whole and should not be attempted from detached specimens. Phony trees are most easily identified in cultivated orchards, where comparison with vigorous healthy trees of the same age and cultivar can be made. Identification of the disease in wild seedlings and in orchards of mixed cultivars may be difficult unless the symptoms are very pronounced. Pruning, fertilizing, and cultivating, in keeping with good horticultural practice, will induce vigor and growth that intensify symptoms and bring out strong contrasts between the diseased and the healthy trees.

No truly symptomless carriers of PV have been found, but some species, such as chickasaw plum, are much less strikingly affected than peach. Such species are also less strikingly affected when growing under competitive conditions in natural thickets than under good cultural conditions. Visual identification of the disease in native thickets is unreliable and highly uncertain. The occurrence of PV in chickasaw plum growing in thickets near the sites of old phony-affected peach orchards is also high (165).

Phony virus can be inactivated in dormant peach nursery trees by immersing the trees for 40 minutes in water maintained at 48° C. (330).

Chemical identification test.—A simple, rapid chemical test, easily applied

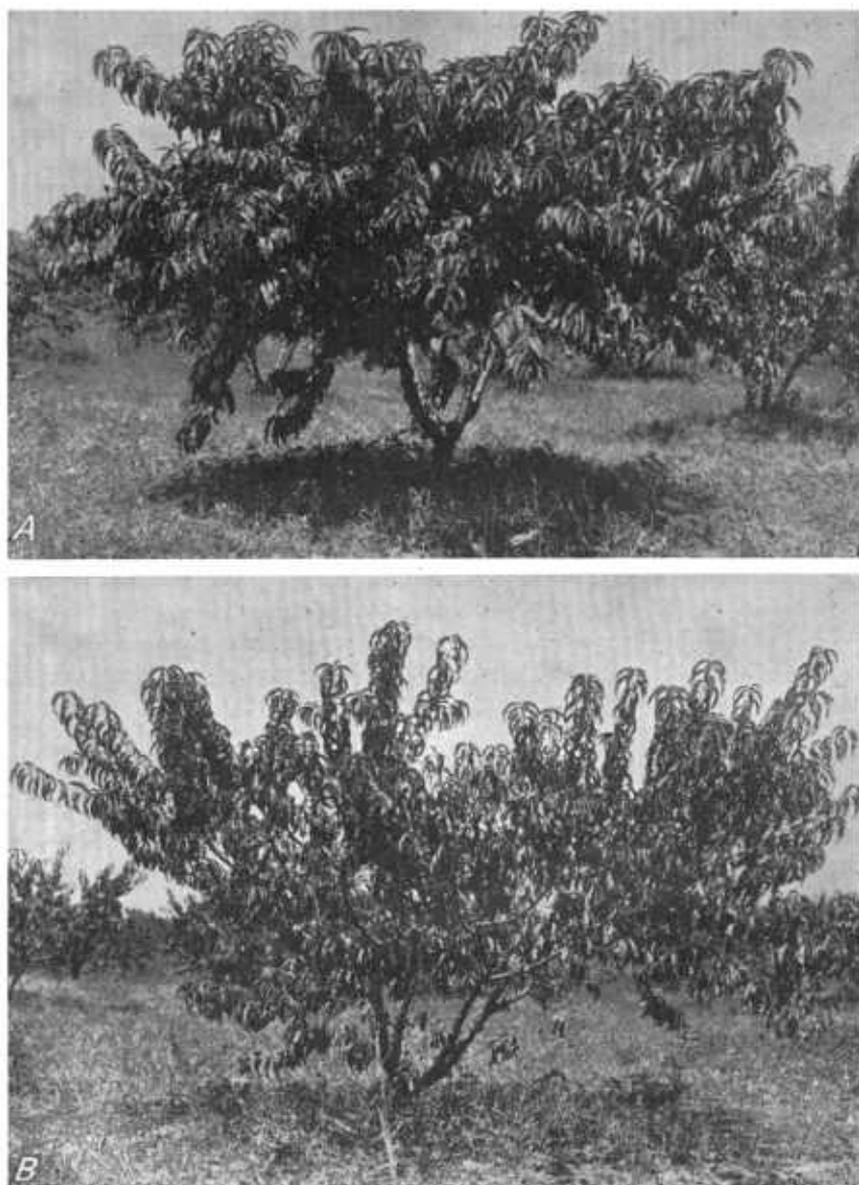


FIGURE 38.—*A*, Six-year-old Elberta peach tree affected by phony, showing the dense shadow cast by the heavy, appressed foliage; *B*, comparable healthy tree in the same orchard.

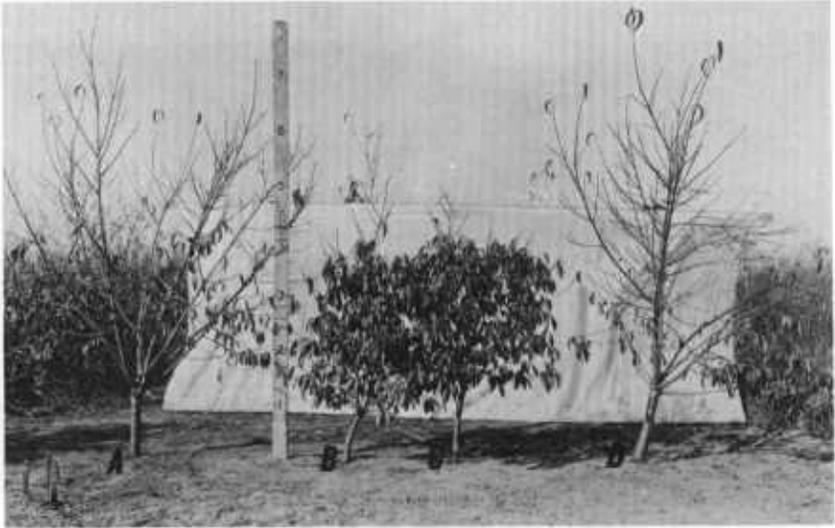


FIGURE 39.—Four nursery peach trees of same age: *A* and *D*, Healthy trees; *B* and *C*, trees affected by phony as a result of root-graft inoculation, showing dwarfing and retention of foliage.

in the laboratory or field, can be used for confirming identification of the phony disease of peach (323). The test reagent is prepared by acidulating 100 cc. of absolute methyl alcohol with 15 to 20 drops of concentrated, chemically pure hydrochloric acid.

For the test, select unblemished, whole-root sections 4 to 6 inches long, with a woody cylinder $\frac{1}{2}$ to $\frac{3}{4}$ of an inch in diameter remote from malformations or injuries. Remove the bark and cut entire transverse sections of the woody cylinder 0.5 to 1.0 mm. thick. Immerse these in 5 cc. of the reagent in a shallow container. Before the test, the wood should be fresh, nearly white, and in good condition. If numerous, well-distributed purplish spots up to 2 mm. in diameter appear against a background of whitish or faintly purple wood after a few minutes in the reagent, a positive test for phony is indicated (fig. 41). Parallel tests should be run on roots of known healthy trees for comparison. If acidulation is too strong or if the sections are allowed to remain too long in the reagent, the entire surface of the sections (either healthy or phony) may be purple.

In well-developed cases of phony, each suitable root will give a good positive test throughout the year. In old cases of the disease, easily visible brown flecks may occur in the wood sections. These will usually turn purple in the reagent, and numerous additional purple spots will develop during the test. Only the wood gives the test; the bark shows no reaction. In some cases, phony stem

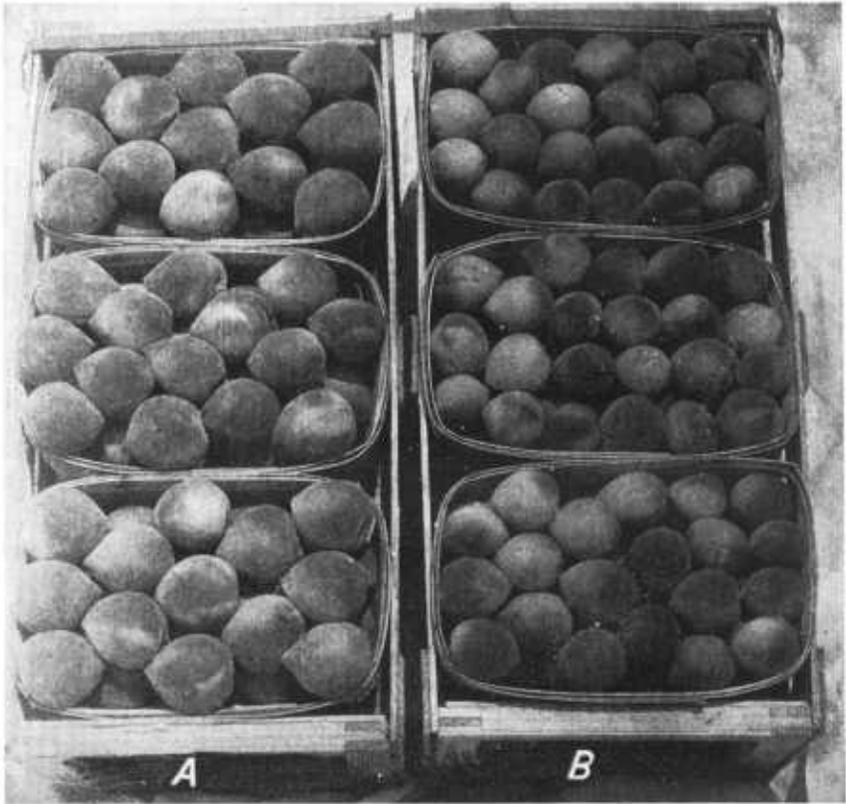


FIGURE 40.—*A*, Commercial pack of best-grade Hiley peaches from a healthy tree; *B*, similar pack of fruits from a tree affected by phony, showing smaller size of fruit.

wood may show a positive reaction to the test, but it is much less satisfactory than rootwood.

Prunus species other than peach give a somewhat variable reaction. Therefore, the chemical test is not considered definitive in them.

Transmission

The phony disease is easily transmitted when roots of a diseased tree are grafted onto roots of a healthy tree. If large root pieces are used as inoculum, the incubation period may be only a few months, whereas with small pieces, 3 to 6 inches in length, symptom production requires 18 months or longer. Transmission also can be effected by grafting diseased scions or root pieces into the tops of trees. It is imperative that the inoculum contain infected wood along with bark and that this wood be kept alive until a wood union has formed between the inoculum and the inoculated tree.

Heel-spur scions (a bark shield subtending a short spur in place of a bud)

were used effectively for transmitting phony from tops to tops (328). The freshly cut heel-spur bark shields are inserted in a T-cut in the same manner as with budding. The shield placed directly against the cambium makes a quick union which keeps the virus-infected wood in the spur alive until a new union is formed. Straight scions cut to a long bevel inserted into a T-cut can also be used but were less effective than the heel-spur type. The woody cylinder of scions whipgrafted onto trees usually dies before a wood union is completed even though the bark cylinder lives and the scion grows into a shoot. Such scions were ineffective in transmitting phony.

When trees are inoculated in the roots, PV becomes distributed throughout the root system, and the inoculated trees become characteristically phony, but the virus does not move into the tops (328). If an infected scion is grafted onto seedling roots at the ground level and develops into a new top, the virus is carried along in new growth and becomes distributed throughout the top from which it can be transmitted at will. If one arm of a growing tree is infected through inoculation, PV moves rapidly to the roots. It may also be carried to tissues of the inoculated tree immediately adjacent to the inoculation point, but it will not become generally distributed to other parts of the tops.

PV is not generally well distributed in the tops of naturally diseased trees. This fact indicates that insect vectors may feed on lower branches from which the virus is translocated to roots. Roots contain approximately 20 times the amount of living wood as tops (198). This larger amount of living wood supports a greater amount of virus and, because the wood is interconnected, it facilitates virus distribution. Living wood in trunks and branches exists mostly as islands in medullary rays. PV is generally better distributed in the tops of plum trees than in peaches.

No strains of PV have been demonstrated. However, six species of Cicadellidae have been reported as vectors of PV under experimental conditions (673, 674, 675). These are *Draeculacephala* sp., *Homalodisca triquetra* (F.), *H.*



FIGURE 41.—Chemical identification of phony in transverse sections of woody cylinder of peach root: *Left*, before test; *right*, after test, showing color reaction.

insolita (Walker), *Oncometopia undata* (F.), *Graphocephala versuta* (Say), and *Cuernia costalis* (F.). All are members of the subfamily Tettigellinae. All are large, long-lived species, which overwinter as adults. Once they acquire the virus, they can transmit it any time during the remainder of their lives. On peach, they feed principally on the woody cylinder of 1- to 2-year-old twigs and small branches. It is assumed that they place the virus directly into the wood, but it is not known how the virus gets to the roots.

Control Measures

The phony disease reached a peak in the mid-1940's, and its incidence has steadily declined since that time. Control is effected through a coordinated program of roguing, general use of wide-spectrum insecticides, selection of new orchard sites away from old, phony-affected orchards, and removal of wild plums from the vicinity of orchard sites. The roguing program is carried on jointly by the Agricultural Research Service, USDA, State regulatory officials, and growers.

Remarks

Phony virus has unusual specific tissue relationships. Early work showed that PV is largely limited to the roots of naturally infected trees; symptoms in the tops are induced by effects of the virus on growth-controlling systems produced in the roots. Later work showed that the virus is restricted to the woody cylinder (324) but could exist to a limited degree in the tops. If diseased trees are developed from infected scions, PV is carried to all portions of such trees. Where trees are infected by grafting diseased scions on branches, PV moves rapidly downward, where it invades all portions of the roots. It also remains in the inoculum scion and any growth from it. Sometimes it will invade other branches near the inoculation point but does not invade other portions of the tops of trees. If trees are infected by grafting diseased roots onto healthy roots, PV will move throughout the root system but will not move into the tops of the trees. General restriction to the roots is the result of restriction to living wood and not the result of killing of the virus in tops exposed to high summer temperatures.

Phony is first in importance among virus diseases of peach in Georgia and some other Southern States. Over two million phony-diseased trees have been removed by control agencies since 1929. Previously, an estimated million orchard trees had been ruined by the disease and removed by growers in central Georgia.

Phony does not cause early death of the affected trees, but it induces marked dwarfing of new growth and of fruits, which results in a smaller crop of peaches, most of which are undersized. The longer the tree has the disease, the more pronounced this effect becomes. The bulk of the fruit from phony trees falls in the smaller commercial grades or is graded out entirely. Trees that contract the disease before bearing age never come into profitable bearing, and mature trees that have had the disease several years produce no commercial crop of fruit.

The incidence of phony disease reached its peak in the 1940's. At that time, where the disease was prevalent and spread was rapid, it was not uncommon for half the trees in an orchard to be phony by the fifth year in the orchard and 99 percent at 10 to 12 years. During the last 15 years, with coordinated control, the incidence of phony in well-cared for orchards has become negligible. Local hot spots have developed in orchards in areas not receiving the standard insect-control spray programs.

“PRUNUS” RING SPOT GROUP

GEORGE NYLAND, R. M. GILMER, and J. DUAIN MOORE

Causal Virus

Prunus ring spot virus (PRSV).

Synonyms and Relationship

Peach ring spot, peach necrotic leaf spot, necrotic ring spot, tatter leaf (in part), lace leaf, cherry ring spot, and stone fruit ring spot are synonyms of *Prunus* ring spot. Some of the isolates distinct enough (144) to be called strains of PRSV are those that incite recurrent necrotic ring spot, necrotic ring spot, cherry rugose mosaic, almond calico, and Stecklenberger disease. Typical apple mosaic virus, rose mosaic virus, some virus isolates that incite plum line pattern, and initial mosaic symptoms in hops show relatedness to PRSV; some or all could be considered strains of PRSV. Each of these strains can be recovered from naturally infected plants and identified. Variants that may be designated as isolates or forms exist within each strain. Strains may be grouped as serotypes (232). Serotype is a designation applied to virus groups that share a minor proportion of common antigens (343). Thus, apple mosaic virus (AMV) and rose mosaic virus (RMV) have a large proportion of common antigens, and necrotic ring spot virus (NRSV) and Danish line pattern virus (DLPV) have a large proportion of common antigens (232), but AMV and RMV could be designated one serotype and NRSV and DLPV another serotype of PRSV. The names for the diseases within the groups could be retained. The final designation of particular isolates or groups of isolates of PRSV associated with specific diseases as related strains or serotypes or as separate but related viruses remains a matter to be settled in the future.

History and Distribution

PRSV occurs as many strains or forms that incite different diseases and is the most commonly encountered virus in cultivated species of *Prunus* and some other rosaceous plants. The virus was first named peach ring spot virus (156), but earlier reports probably were also concerned with one or more strains (653, 679). PRSV was soon recognized to be a common contaminant of stone fruit virus cultures (144), and transmission of it through seed was demonstrated (123, 145, 147). One of the viruses first transmitted from sour cherry to cucumber by sap (446, 473) was later identified as PRSV (227). Once the virus was sap-transmitted some of its physical, chemical, and biological properties were studied (228, 231, 232, 233, 452).

PRSV and prune dwarf virus (PDV) both are spread by pollen from plant to plant of sour cherry in the orchard (236). PRSV is carried in pollen (196, 708, 725) and can infect seeds when infected pollen is applied to emasculated flowers, even on healthy plants (666, 708). Seed and pollen trans-

mission and propagation of infected cultivars account for the worldwide occurrence of PRSV. In spite of the general distribution and the means of spread of PRSV, realistic nursery improvement programs have been exceptionally successful in some areas in controlling the diseases incited by the many forms of this virus. Spread is rapid in sour cherry in the Midwestern and North-eastern States, but relatively slow in sweet cherry, prune, and cling peach in the Western States. Where natural spread is rapid, clean stock programs must be combined with maximum isolation for control.

No arthropod vector of PRSV is known, nor has the place of origin of the virus been established. PRSV has been isolated from native stands of pin cherry in Wisconsin and from *Prunus fasciculata* in California, but native stands of wild *Prunus* are not known to be generally infected in the United States or Canada. Most evidence points to the Middle East or Western Asia as the place of origin. Logically, there is where a search should be made for a vector. The virus has many properties and characteristics of NEPO viruses which have nematodes as vectors.

PRSV is a serious disease in the fruit-tree nursery causing poor bud and scion "takes" and reducing growth of the trees (443, 449, 515, 518). It also reduces growth and yield of trees in the orchard (378, 449, 508), but with some species the effects may not be as great as with others (376, 537).

Economic Hosts

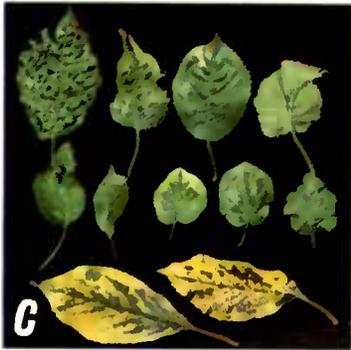
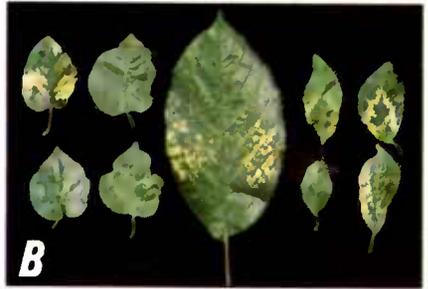
Almost all cultivated species and cultivars of *Prunus* that have been tested are susceptible to one or more strains of PRSV (37, 152, 242, 475). Burbank and Abundance plums were reported resistant to infection (474, 475) as was *P. cistena* (242). However, later reports showed them to be susceptible to some strains or isolates (761, 240). Symptoms induced by several strains in some economic hosts are shown in figures 42 and 43.

Other Hosts

PRSV has a very wide host range among wild species of *Prunus* and herbaceous plants (37, 152, 175, 225, 242). Some *Prunus* and herbaceous hosts have been used successfully to separate certain viruses from a mixture with PRSV (7, 225, 447).

Symptoms

Shock symptoms followed by chronic symptoms are produced after infection in most hosts of the virus if they are not already infected with a latent isolate or strain. Shock symptoms recur in some hosts intermittently in years subsequent to infection and like chronic symptoms seem to be influenced by environmental factors. The virus is extremely variable and exists as a multiplicity of forms each slightly different in its effects on a standard host range. The differential tolerance of host species and varieties, the variability of the virus, and the effects of environment result in unlimited variations in symptoms encountered in the field and in the test plot. General types of symptoms can be classified as chlorosis, necrosis, leaf deformity, and stunting. Chlorosis occurs in patterns of



rings, lines, bands, spot mottle, and mosaic. Buds, leaves, shoots, large branches, and roots may become necrotic. Epinasty, twisting, rugosity, and enations are leaf symptoms sometimes associated with infection. Entire plants may be dwarfed or only portions of them may be stunted. Necrosis usually occurs only during the initial acute stage. It may affect leaves, shoots, and shoot terminals and may occur as small or large cankers on the limbs or trunks. In many hosts, other conspicuous symptoms also become less obvious after the first year or two of infection. In some hosts infected with certain strains of the virus (e.g., cherry rugose, almond calico, and recurrent ring spot), visual symptoms occur annually and can be classed as chronic symptoms. In such cases, symptoms are usually more severe a year or two following infection than they are later. Symptoms on specific hosts will be discussed for each strain of the virus.

Necrotic ring spot strain (NRSs).—This strain can be classed as the ordinary variety of ring spot and typically produces in its hosts shock or acute symptoms followed by recovery and the absence of obvious visual symptoms. The extent of recovery varies with the host, the isolate of the strain, and environmental conditions. In peach trees, when infection occurs sometime during the previous season, budbreak is retarded, many flower and leaf buds die when partially open or before, and previous season's twigs are killed, or cankers form at the nodes or extend vertically either below or above the point of infection on branches that are not completely killed. Emerging leaves may show only a few obscure rings or chlorotic areas or may develop severe necrosis or necrotic spotting of chlorotic areas (fig. 44). Leaves are often deformed as a result of unilateral necrosis, and many leaves are cast when symptoms are severe (fig. 45). Some forms of the strain kill small trees or trees up to 3 or 4 years old the year following experimental inoculation or following natural infection in the orchard (152).

As the season progresses, new growth of peach is essentially without symptoms, except that in the nursery and orchard leaves of current season shoots that are

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FIGURE 42.—*Prunus* ring spot. Symptoms incited by three strains of *Prunus* ring spot virus (PRSV) on several *Prunus* hosts. *A*, Recurrent strain (RRSs): *upper left*, depressed rings on Montmorency cherry leaves; *upper right*, chlorotic spots and patterns and mosaic on mahaleb cherry leaves; *bottom*, yellow Montmorency cherry leaves similar to those with sour cherry yellows. *B*, Almond calico strain (AICs): *Left*, Calico, chlorotic patterns, and mosaic on mahaleb cherry leaves; *center*, calico on sweet cherry leaf; *right*, chlorotic patterns and mosaic on Shiro plum. *C-F*, Cherry rugose mosaic strain (CRMs): *C*, *Top row*, sweet cherry leaves showing rugosity, lace leaf, and chlorotic blotch; *center row*, *left*, two Montmorency cherry leaves with rugosity and ring spots; *right*, three mahaleb cherry leaves with calico, mottle, and necrotic spots; *bottom row*, yellow Montmorency cherry leaves similar to those with sour cherry yellows. *D*, Chlorotic patterns in Shiro plum. *E*, Bare limbs and dieback in Napoleon cherry. *F*, Closeup of Napoleon tree showing shoot development from latent buds, chlorotic blotches in leaves, and leaf necrosis.

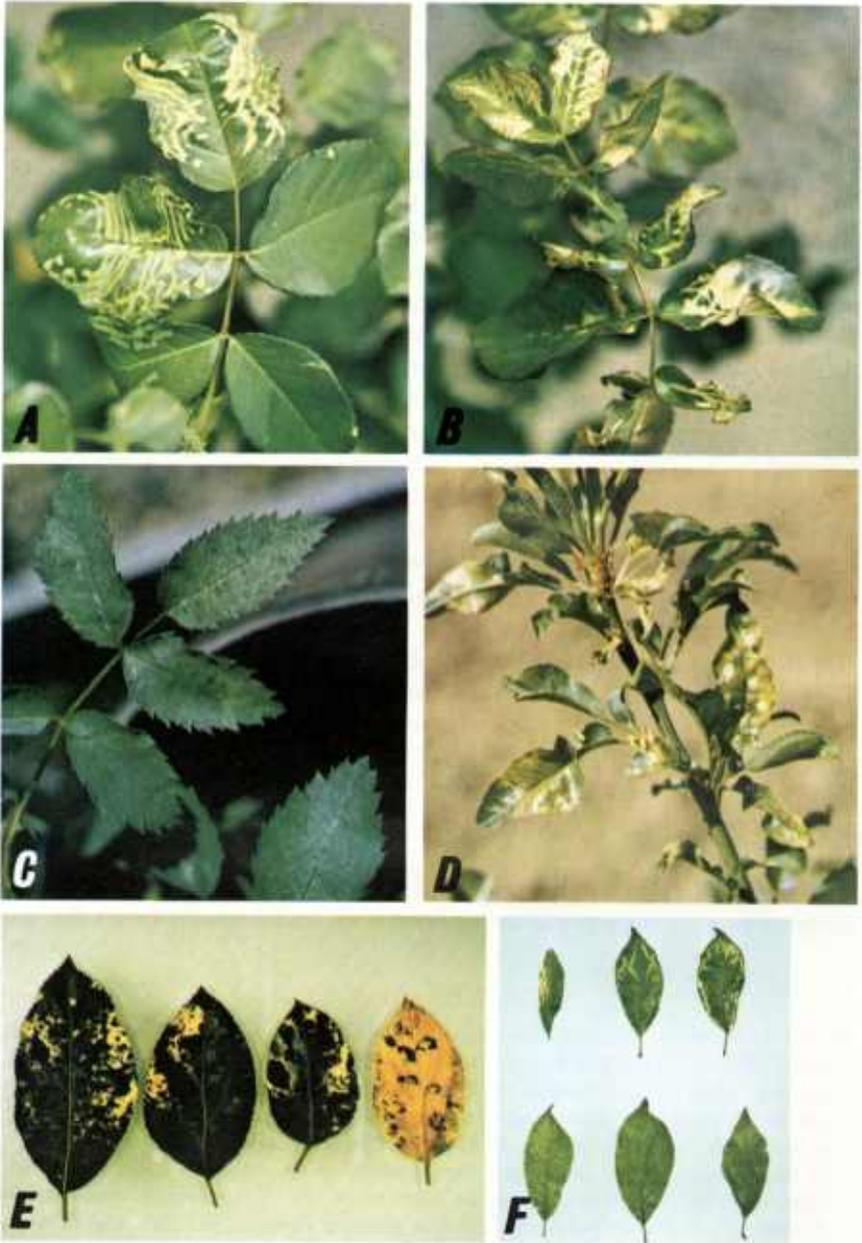


FIGURE 43.—*Prunus* ring spot. *A*, Rose mosaic incited by inoculation with a pure culture of recurrent ring spot strain (RRSs) of *Prunus* ring spot virus (PRSV); *B*, with a pure culture of cherry rugose mosaic strain (CRMs); *C*, with a pure culture of necrotic ring spot strain (NRSs). *D*, Mosaic on almond incited by CRMs. *E*, Mosaic on Gravenstein apple incited by a strain of PRSV. *F*, Line pattern-type symptoms in Shiro plum incited by an isolate of PRSV (spring mosaic, oak leaf patterns, and vein netting).

growing rapidly may develop necrotic spotting as they emerge from the terminal whorl (fig. 46). This phase of the disease, originally described as necrotic spot (122), often follows short periods of excessively high temperature in the Central Valley of California and is an important disease in the nursery (488, 692). Pure cultures of the other strains of the virus also incite necrotic leaf

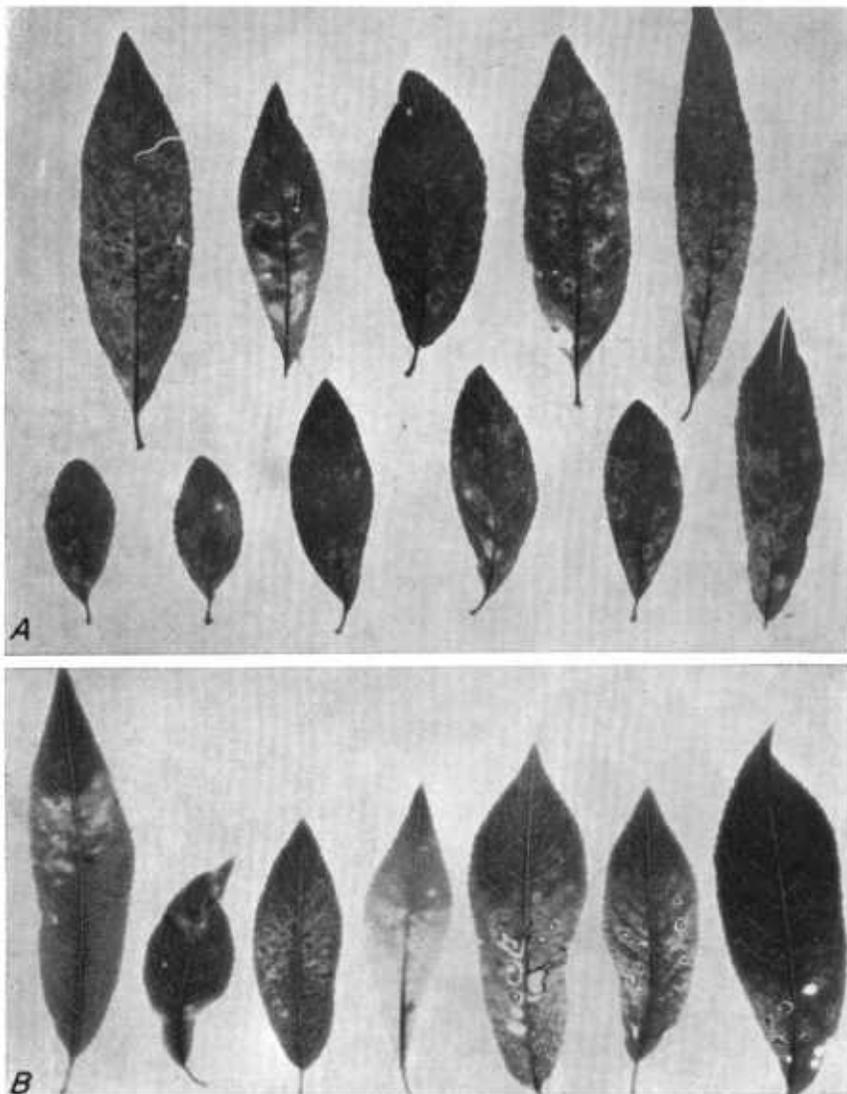


FIGURE 44.—*A*, *Prunus* ring spot affected leaves of Lovell peach, showing rings, spots, and chlorotic patterns. *B*, Affected leaves of July Elberta peach on which the symptoms are predominantly chlorosis but on which rings and shot hole occasionally occur.

spot as summer symptoms in peach (George Nyland, unpublished data). Leaf casting and occasionally necrosis of cortical tissues of the young shoot or death of the shoot tip reduce the growth of peach "June buds" and lower the grade of infected trees.

In addition to necrotic leaf spot symptoms, mature peach trees may show other symptoms associated with necrotic ring spot infection. Color-breaking of the blossoms of Rio Oso Gem and other unspecified cultivars of peach was



FIGURE 45.—Elberta peach in midsummer after showing acute symptoms of *Prunus* ring spot in the spring. Note bare twigs and thin foliage where buds died and were shed.

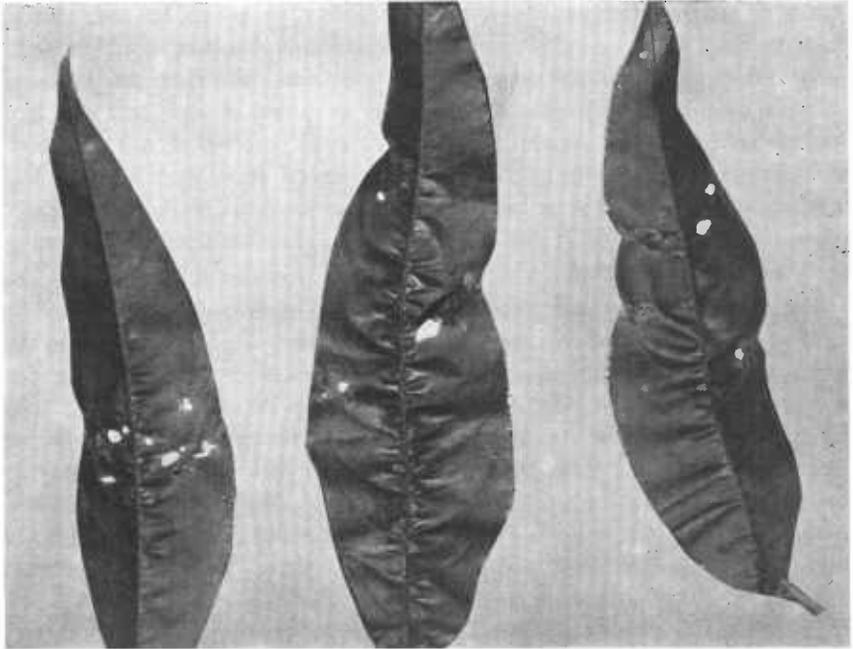


FIGURE 46.—Young leaves of peach showing necrotic spots—the summer symptoms of *Prunus* ring spot in peach.

attributed to ring spot virus in southern California (157). Up to 25 percent of the blossoms on some trees showed symptoms, but no leaf symptoms were seen. Blossom symptoms showed for 3 consecutive years that the trees were observed. Other symptoms that are associated with necrotic ring spot in bearing orchard peach trees are reduced vigor, death of lateral spurs and leaf buds on older fruit-bearing hanging branches, rough bark of the trunk and main scaffold branches, and reduced yield of fruit. Fruit yields of cling peaches may be reduced as much as 5 tons per acre (H. A. Williams, personal communication).

All peach cultivars tested are susceptible to NRSs and show essentially a similar range of symptoms, although the reactions to a particular isolate may vary considerably. Freestone cultivars probably are damaged in an obvious way more than clingstone cultivars. The tolerance of cultivars to individual isolates varies: an isolate that may cause severe necrosis, splitting, and cracking of the bark of Elberta may be relatively mild on Halford or on Lovell seedlings (152).

The NRSs in sweet and sour cherries is coextensive with these hosts. Most trees in bearing orchards of cherries, which were established prior to clean stock programs, are infected, and many show symptoms of some sort each year or intermittently (291, 295). Although the NRSs is essentially symptomless in some trees, careful inspection of trees in sweet and sour cherry orchards in late

spring or early summer will detect some evidence of infection. In sour cherry, scattered older leaves or entire spurs may show necrotic spotting or etched rings early in the season. In sweet cherry, faint to prominent chlorotic rings or banded chlorosis is evident, and often quite obvious symptoms of shot hole and lace leaf are seen. It is not unusual to see one or more branches of a tree, known to have been infected with PRSV for at least several years, develop tatter leaf symptoms. Such trees observed in orchards in San Joaquin County, Calif., sometimes show symptoms of rugose mosaic on those same branches the following year and subsequent years.

Symptoms in healthy cherry trees inoculated artificially with field cultures or with pure cultures of NRSs show symptoms characteristic of the isolate and the host species. In Montmorency sour cherry, the initial effect of necrotic ring spot is a pronounced delayed foliation of individual limbs or entire trees (37) (fig. 47 *A*, *B*, and *C*). Leaves on affected branches are reduced in size, and before they unfold they may show light-green spots and dark rings, which vary in size from 1 mm. or less up to 5 mm. in diameter and have a water-soaked appearance (fig. 48*B* and *C*). Partial rings and sometimes concentric rings are also present. The surface of affected leaves is roughened, and the margins tend to be wavy (38). In the early stages, the ring symptoms are visible by reflected light only. As the season advances, the affected areas may become necrotic, fall out, and give a "shredded leaf" effect (fig. 48*A*). Symptom expression usually is limited to the first leaves that unfold; leaves formed later generally do not show symptoms (fig. 49*A*).

Sometimes, blossom symptoms also may be present on severely affected branches of Montmorency. The pedicels may be shortened until they are almost sessile, and the calyx and corolla may be twisted and distorted. Such flowers usually do not set fruit. In subsequent years, branches that had shock symptoms the previous year are essentially without symptoms, but infected trees may be smaller and may have smaller leaves than healthy trees and show a thinness of foliage as reported from Ontario (37), and confirmed in Oregon and California by inoculation with pure cultures of the strain (J. A. Milbrath and George Nyland, unpublished data). Details of symptoms may vary with the isolate of the strain and with the clone of Montmorency.

The development of symptoms in sweet cherry and Stockton Morello is similar to that in Montmorency in that most isolates of the strain produce initial shock systems followed by recovery (fig. 50) to extreme necrosis involving buds, shoot tips, and cortical tissues. Cankers that exude copious quantities of gum may easily be confused with those incited by fungi or bacteria. Cankers usually are produced in inoculated branches but not in other branches as the virus spreads throughout the tree during the season of initial symptoms or in subsequent years. In some cases, leaf symptoms in subsequent years may be very mild; in other cases, they may be as severe as initially. The lace leaf or tatter leaf symptoms may be evident some years in one or more branches of trees known to



FIGURE 47.—*Prunus* ring spot. *A* and *B*, Branches of Montmorency cherry showing delayed foliation, stunted leaves, and shortened blossom pedicels; *C*, comparable healthy Montmorency branch. *D* and *E*, Italian Prune showing necrotic spotting and shot hole; *F*, comparable healthy Italian Prune branch.

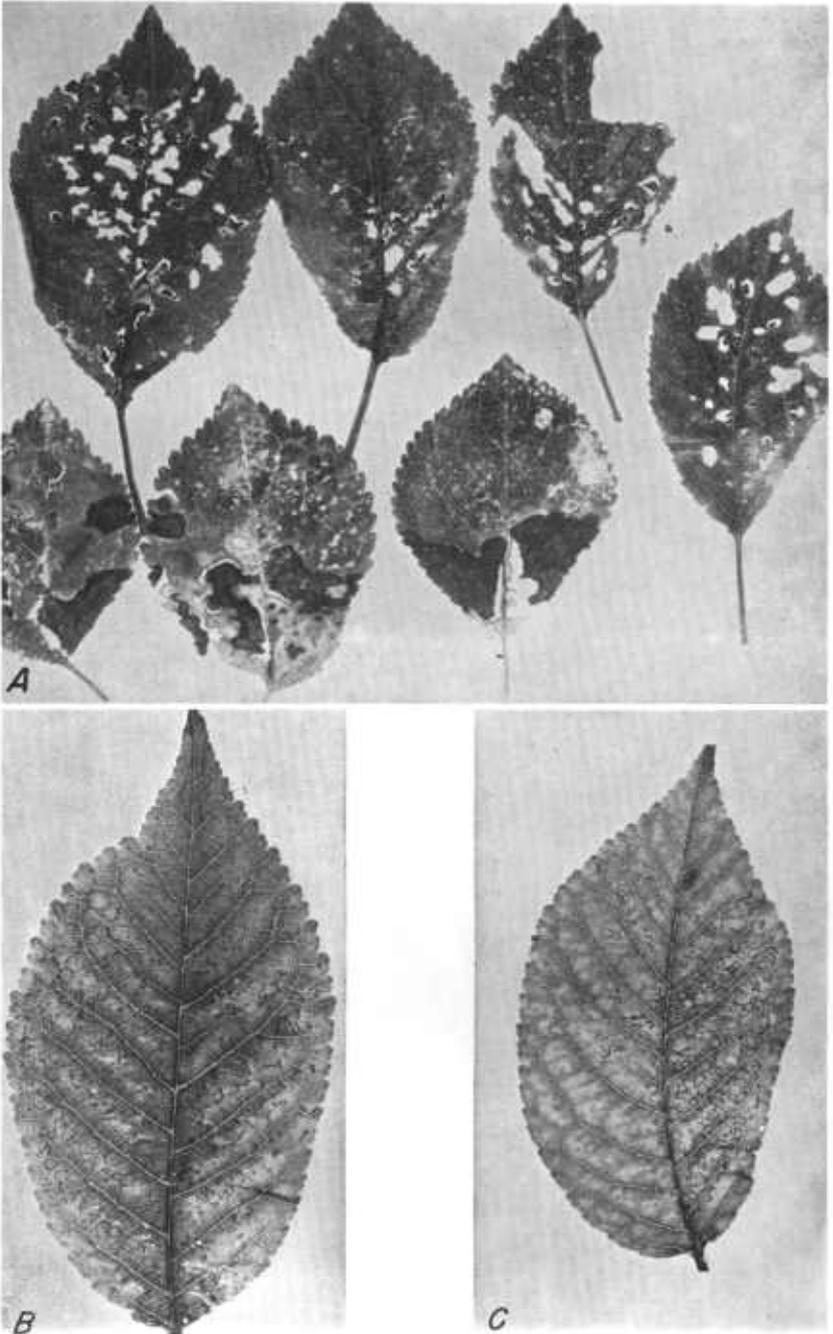


FIGURE 48.—Montmorency cherry leaves with necrotic ring spot, showing *A* severe necrotic shock and leaf shredding and *B* and *C* water-soaked rings and arcs.

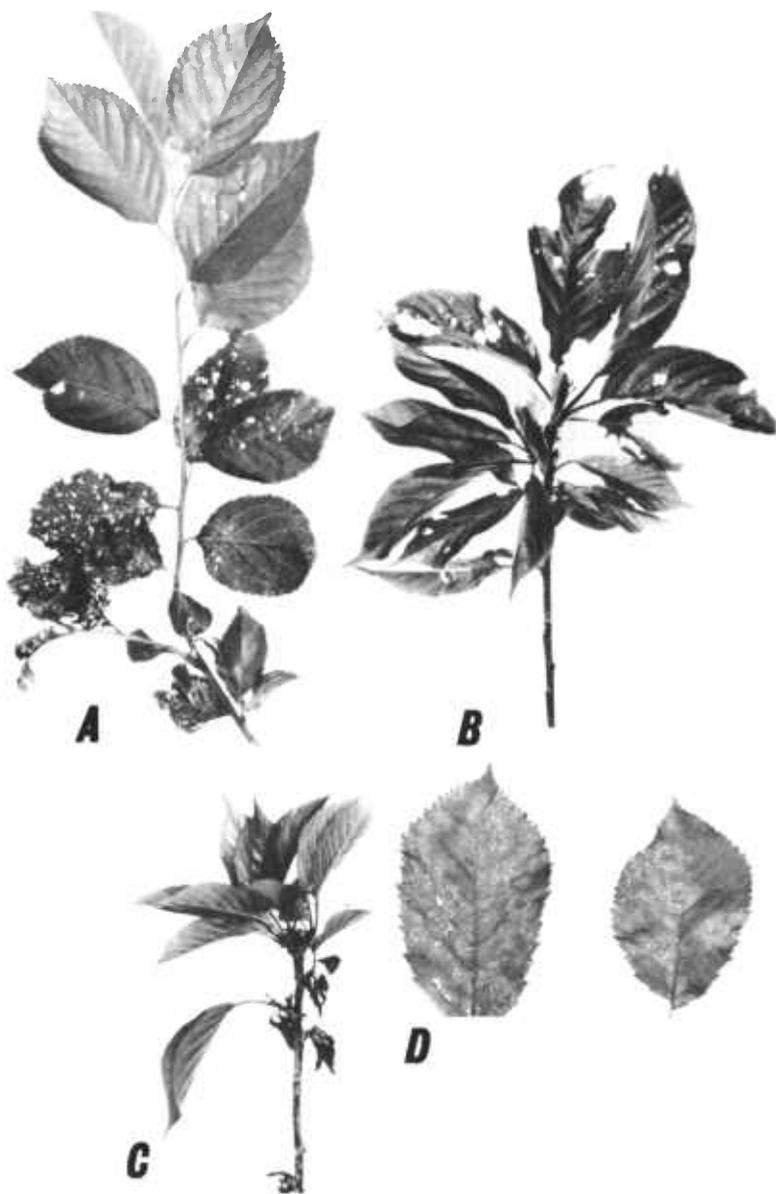


FIGURE 49.—*Prunus* ring spot. *A*, Stockton Morello with shock symptoms of necrotic ring spot on growth produced about 4 weeks after tree was removed from heat-treatment chamber, followed by symptomless leaves. *B*, Chronic spring symptoms of shot hole in Kwanzan cherry. *C*, Spur killing, an initial shock reaction in Kwanzan cherry. *D*, Necrotic and chlorotic spots and rings in sweet cherry.

have been infected with the NRSs for many years. Whether this is the result of infection by another isolate or strain or by PDV or is an environmental effect at the time symptoms are seen is not known. Trees known to have been infected with the rugose mosaic strain for many years also have been observed with lace leaf symptoms in certain other years. Most observers would agree with Helton (295) that annual fluctuations in symptoms in particular trees suggest environmental effects and that consistent variations among trees probably are due to variations in the virus. NRSs may kill the shoot tip of infected nursery cherry

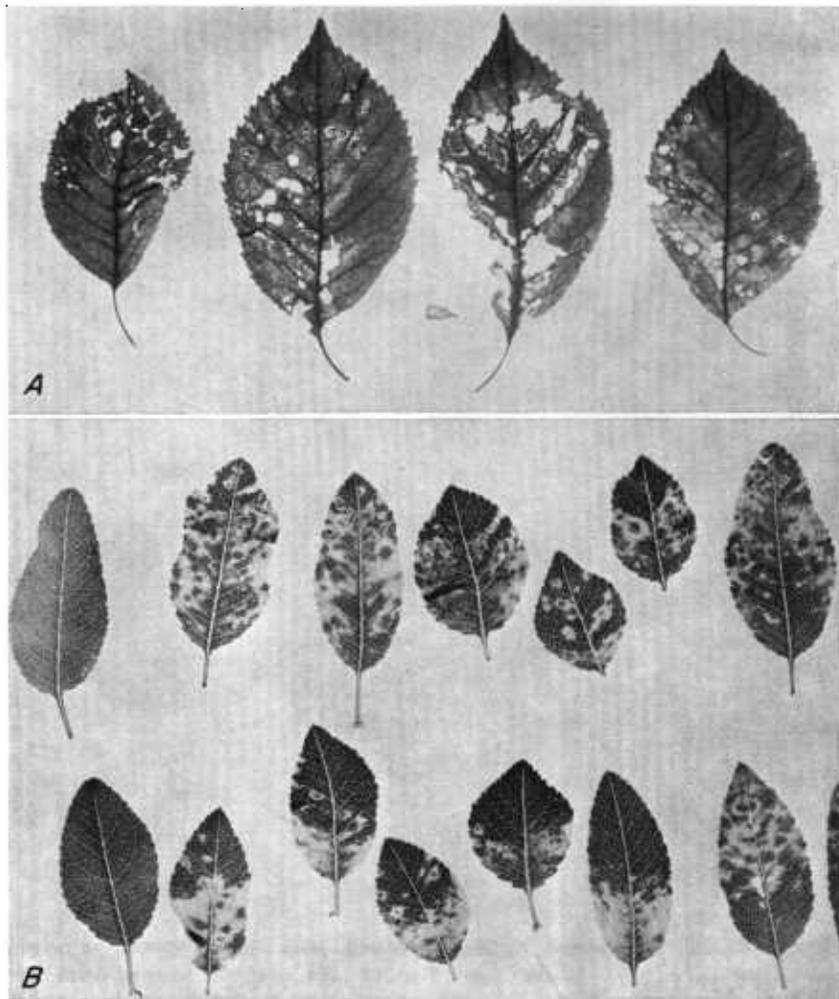


FIGURE 50.—*Prunus* ring spot. *A*, Leaves of mazzard cherry showing rings, shot hole, and chlorotic and necrotic patterns. *B*, Nonpareil almond leaves showing chlorotic rings and spots; unaffected leaves at left.

trees during the summer. This injury resembles insect damage with which it is sometimes confused.

Almond reacts to infection with NRSs much the same as peach. Chlorotic or necrotic rings, bands, and mottle are initial leaf symptoms (fig. 50B), and the amount of bud, stem, or bark necrosis varies with the isolate of the virus. Mild mottle and mosaic in some leaves and occasionally recurrence of shock symptoms in portions of the tree are seen in years subsequent to initial infection. Almond may also show summer necrotic leaf spot similar to that on peach.

Other species of both cultivated and wild *Prunus* show symptoms of varying severity. None has been found that is immune to all isolates of the strain. In general, plums and prunes are affected less severely than peach or cherry in the initial stage of infection and may show few or no symptoms in years subsequent to initial infection. When symptoms are produced, they resemble those in peach and cherry (fig. 47, D-F). *Prunus mahaleb* and *P. tomentosa* develop good symptoms that are diagnostic for some strains (202, 210). Symptoms in *P. mahaleb* in the field may recur annually as chlorotic or necrotic rings, bands, and oak-leaf patterns.

Recurrent ring spot strain (RRSs).—The very obvious chronic symptoms in Montmorency associated with certain isolates of PRSV are consistently distinct and probably justify strain status for these isolates. Fulton's virus A (227) is an isolate of RRSs. The name "recurrent ring spot" implies that symptoms occur on all the branches on a given tree of Montmorency year after year once the virus becomes systemic. Almost all of the leaves formed in the spring and early summer each year show symptoms in contrast with such leaves on a tree infected with NRSs which would show only occasional symptoms in scattered leaves. Initial symptoms are the same under similar conditions for necrotic and recurrent ring spot. The severity will vary with the temperature at which the trees are held and the isolate of the virus. If small potted ring-spot-free Montmorency trees are inoculated at bud-break, 7 to 14 days later the first symptom consists of a downward curvature of the growing shoots. This is followed within a few days by a brownish, watersoaked spotting of leaves, usually evident first on the lower surface. These spots become necrotic to form shotholes, or they may coalesce and form large irregular necrotic areas that result in a tattered or lace leaf effect. The typical dark-green to black watersoaked rings or areas usually appear on later-formed leaves. The symptoms as described are essentially the same up to this point for both necrotic and recurrent strains and occur on the first three to five leaves to unfold. Field trees or potted trees infected the previous season with either strain may show some additional symptoms as described under necrotic ring spot. From here on the two strains react quite differently in Montmorency. With necrotic ring spot, later leaves have few or no symptoms as the "recovery stage" is reached. However, with recurrent ring spot all or almost all later leaves may show the typical water soaked or etched rings or have a pebbly, rough upper surface and smooth areas or scars on the

lower surface (fig. 51; fig. 42, *A*). The specific symptoms may occur over most of the leaf or in restricted areas of the leaf often either near the tip or base. All symptoms except initial necrosis recur annually and with some strain-clone combinations necrosis also may recur.

Other recurring symptoms seen in Montmorency trees infected with pure cultures of RRSs are the presence of bract leaves that do not become yellow uniformly as in healthy Montmorency trees but retain islands of green tissue. These leaves are cast earlier than normal bract leaves. Trees which show bract leaves of this type later develop yellow leaves that retain chlorophyll along the main veins and resemble very closely leaves produced in Montmorency trees that show symptoms of sour cherry yellows and rugose mosaic (486) (fig. 42, *A*). One or more flushes of normal size, yellow leaves are produced each spring in trees infected with recurrent ring spot about the same time that other trees infected with PDV show symptoms of sour cherry yellows. The total leaf fall on trees with recurrent ring spot is usually much less than that on trees with sour cherry yellows.

Prunus tomentosa seedlings inoculated with RRSs show typical shock symptoms as with necrotic ring spot but also show chronic symptoms on both leaves and fruit. The leaves may have calico bands and oak-leaf patterns, and the fruits of some seedlings are deformed and necrotic internally when ripe. Infected trees are stunted (George Nyland, unpublished data).

On peach, sweet cherry, and *P. mahaleb*, the symptoms of recurrent ring spot are similar to those incited by the NRSs of PRSV, including the summer leaf spot symptoms in peach.

Cherry rugose mosaic strain (CRMs).—This is designated a strain of PRSV based on host reactions, cross-protection, and serological tests (495, 496). CRMs can be isolated consistently from naturally and artificially infected trees, from seed and from seedlings, and can be identified readily by symptoms produced in cherry and *Gomphrena globosa* (fig. 58, *C*). Certain isolates differ from each other consistently and constitute mild and severe forms within the strain (36, 486, 490) as is also the case with other strains of PRSV.

Initial shock symptoms of CRMs in a standard host range, free from other strains of PRSV, are essentially the same as those incited by the NRSs and RRSs. If inoculations are made in sweet cherry with CRMs during the summer or fall, shock symptoms of ring spot are produced the following spring. If inoculations are made early in the summer, the portion of the tree near the inoculation site may produce only chronic symptoms the following spring as in the case of an infection 2 years old or older. In sweet and sour cherry, acute shock symptoms of ring spot are followed by rugose mosaic symptoms the same year or the following year. The virus moves through mature trees not infected with other strains of ring spot virus in 2 or 3 years but more slowly if infections are mixed. The most obvious symptom occurs in the leaf blade and consists primarily of chlorotic blotches that cause distortion of the leaves. Leaves so

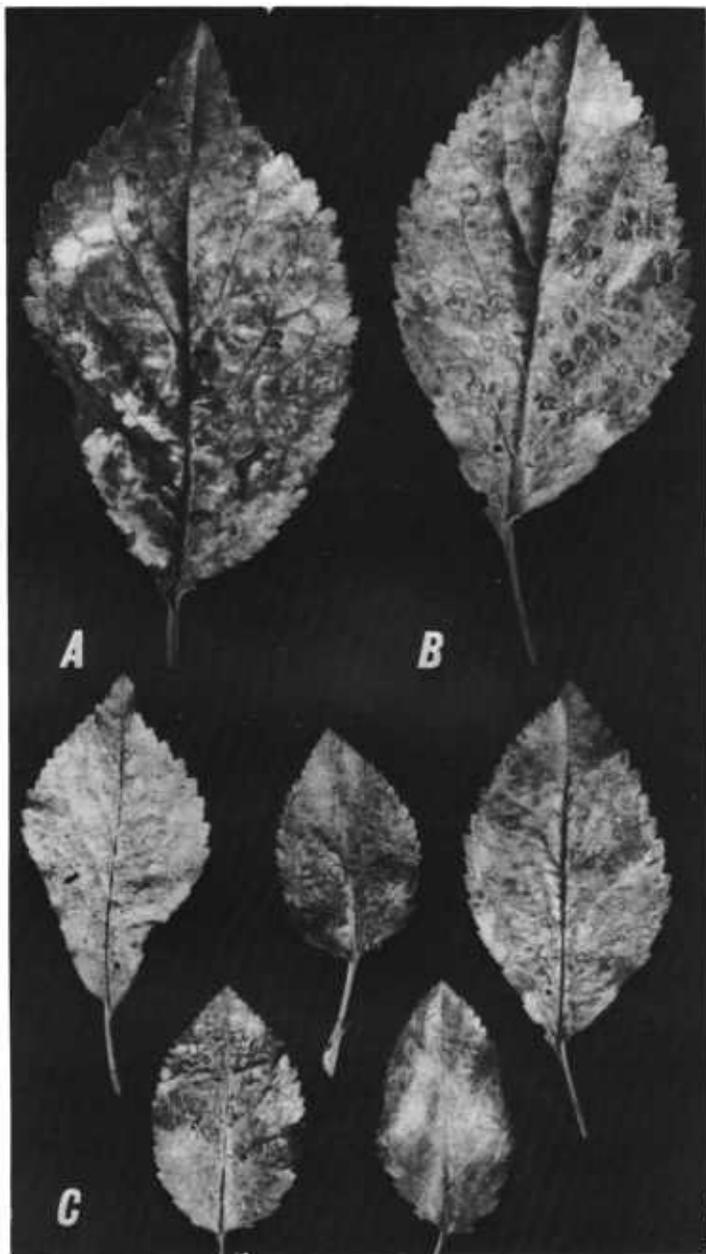


FIGURE 51.—*Prunus* ring spot. Montmorency cherry infected with recurrent ring spot strain (RRSs) of *Prunus* ring spot virus (PRSV): *A*, Initial shock symptoms, showing necrosis and the beginning of shot hole, *B*, recovery symptoms (depressed rings, arcs, and etch) the same season as initial shock; *C*, chronic symptoms (depressed rings and arcs evident by reflected light).

affected often drop by late June or during July, but in some leaves the chlorotic areas become necrotic and drop out, and the leaf remains on the tree. Trees infected for several years with mild forms may show few leaves with symptoms during most of the season. Most isolates produce enations on some leaves. Enations may occur on any part of the abaxial surface of the leaf but are most common immediately adjacent to the midrib near the base of the leaf. Leaves on shoots that arise from latent buds on main scaffold limbs are most likely to have enations (fig. 42, *F*).

Most isolates of CRMs incite yellow-to-white chlorotic speckling or mosaic on scattered leaves of most seedlings and cultivars of sweet cherry. Trees of sweet cherry with rugose mosaic usually bloom later and mature their fruit later than healthy trees. Green, unripe fruits may be seen on scattered branches when most of the unaffected fruits are fully ripe. Delay in ripening may vary from a few days to several weeks. Affected fruits may be mildly to severely deformed; flattened or dimpled at the styler end, or lopsided or partially twisted. Trees infected with severe forms of the strain slowly decline as a result of dieback, death of lateral shoots and spurs, and lack of vigor. Symptoms incited by the CRMs of PRSV are shown in figure 42, *C-F*; figure 43, *B* and *D*; and figures 52, 53, and 54.

In peach, CRMs incites initial shock symptoms like those of necrotic ring spot and also a rosette of some shoots produced from latent buds on the main scaffold limbs. Trees usually recover from the initial severe symptoms but continue to show some necrotic spotting of leaves in succeeding years in the spring and in flushes of growth in summer as new leaves emerge from the terminal whorl as is also the case with other strains of PRSV. Summer symptoms in the Central Valley of California are most obvious a few days after a hot spell of several days duration.

In almond, most isolates of CRMs incite severe leaf distortion; leaves with yellow, white, or light-green mosaic or calico; and bud failure of lateral buds of some shoots of most cultivars inoculated. Trees infected for several years resemble, in gross appearance, those with Drake almond virus bud failure. Isolates obtained from almond trees in California, showing symptoms of "almond mosaic" (fig. 42, *D*), incite extremely severe symptoms of rugose mosaic on cherry and summer leaf spot symptoms in peach.

Sour cherry cultivar Montmorency reacts to inoculation with the CRMs with initial symptoms that are much the same as those induced by isolates of NRSs and RRSs of PRSV. Chronic symptoms of rugose mosaic are a twisting and chlorotic blotch of leaves. Also, yellow leaves are produced 4 to 5 weeks after full bloom, with some green retained along the veins, and very closely resemble leaves of trees with sour cherry yellows. Yellow bract leaves that retain islands of green tissue commonly occur prior to yellows of full-size leaves (fig. 42, *C*; fig. 52, *C*).

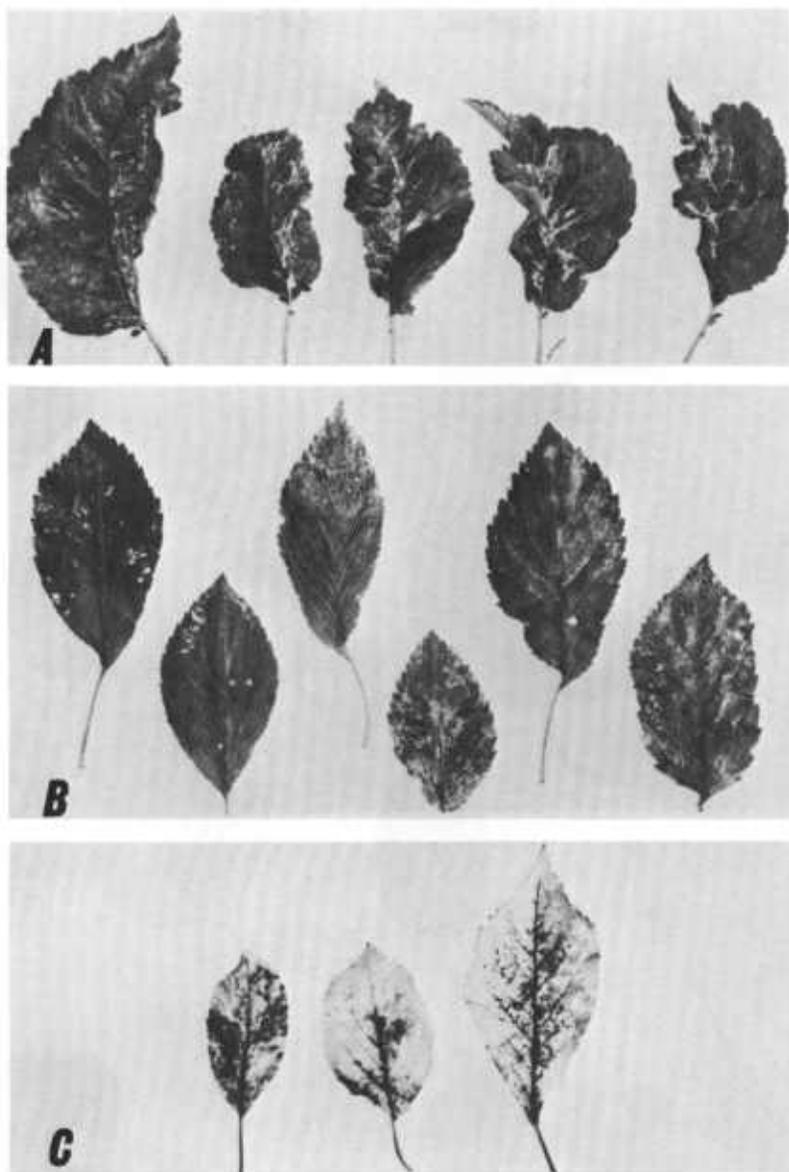


FIGURE 52.—*Prunus* ring spot. Montmorency cherry infected with a pure culture of cherry rugose mosaic strain (CRMs) of *Prunus* ring spot virus (PRSV): *A*, Rugosity and vein clearing, early season symptoms; *B*, calico spots and light-green mosaic, spring and summer symptoms; *C*, yellow leaves with green retained along the veins, similar to sour cherry yellows, midsummer.

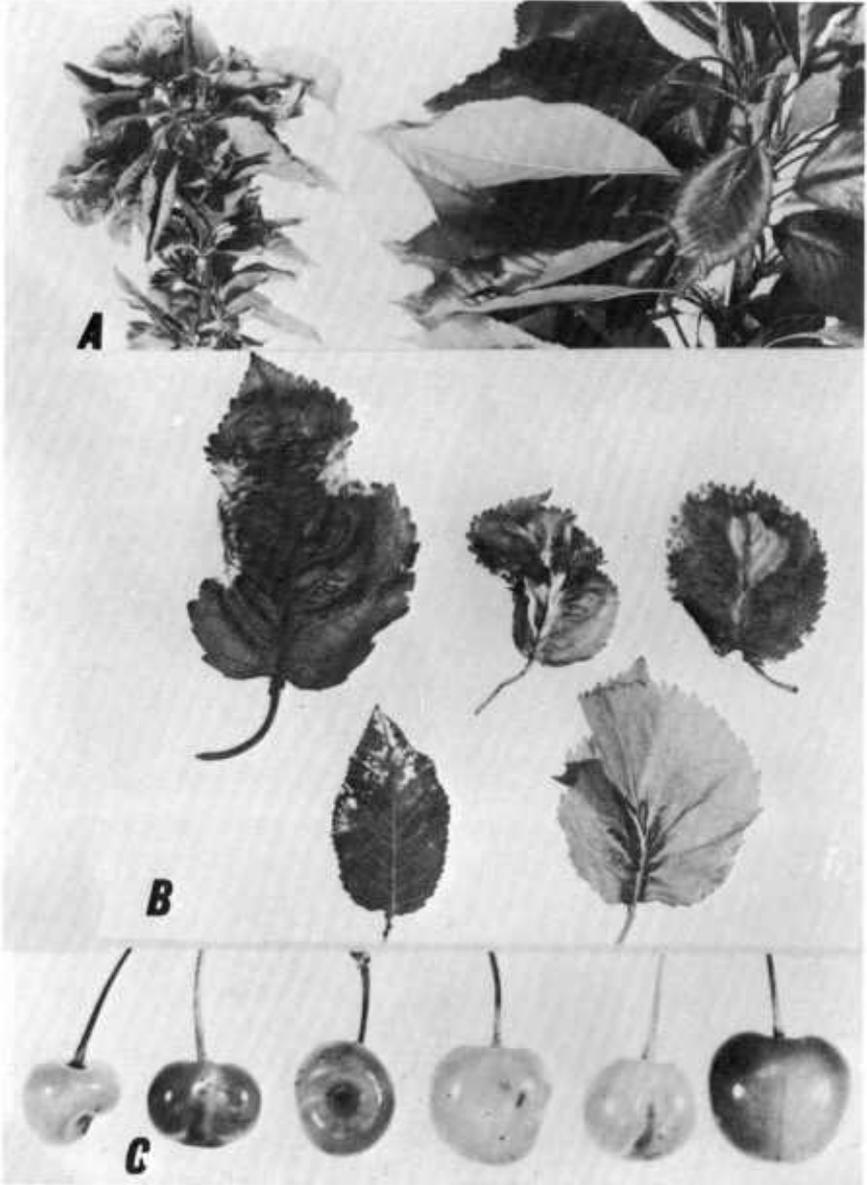


FIGURE 53.—*Prunus* ring spot. Napoleon sweet cherry infected with a pure culture of cherry rugose mosaic strain (CRMs) of *Prunus* ring spot virus (PRSV). *A*, *Left*, severe symptoms second year following inoculation; *right*, normal shoot. *B*, Individual leaves from diseased shoot in *A*. Note the distortion, chlorotic blotches, rugosity, calico mottle, and enations—all chronic symptoms of cherry rugose mosaic. *C*, Deformed fruits on *left* compared with normal fruit at *far right*.

CRMs in Shiro plum incites calico blotches, lines, and oak-leaf patterns that resemble plum line pattern symptoms in this cultivar. However, vein netting or "fish net," a characteristic symptom of plum line pattern, has never been seen associated with infection of CRMs in Shiro plum (fig. 42, *D*).

Almond calico strain (AICs).—Infected trees of almond show variable amounts of foliage with chlorotic spots or blotches (656). The calico spots or blotches on individual leaves may be scattered or aggregated at the tip or base or in a transverse band. The chlorotic areas are predominantly white or light yellow, bleaching to white. Chlorosis in early spring may be quite conspicuous especially on leaves of certain scaffold limbs that also show bud failure symptoms similar to those described for almond virus bud failure. The intensity of the yellow-to-white chlorosis of the foliage varies from year to year and among almond cultivars and seedlings (fig. 55, *A*).

Lateral buds on shoots of affected branches often fail to grow the season after they are produced, and those that do grow usually produce only flowers. The failure of flower and leaf buds to grow is a prominent symptom associated with almond calico in orchard trees of cultivars Peerless, Nonpareil, and Drake. Chlorotic leaf symptoms are usually absent or inconspicuous in trees of Mission

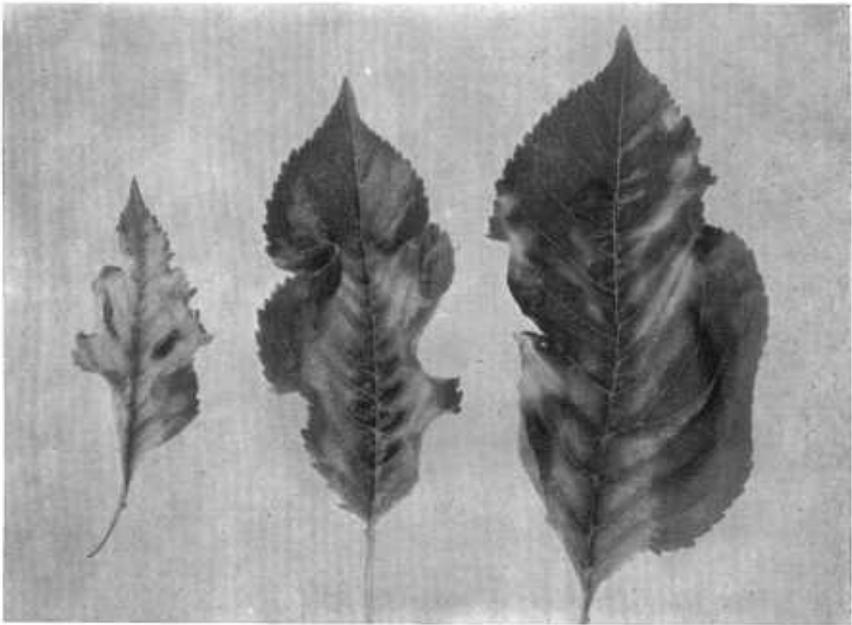


FIGURE 54.—*Prunus* ring spot. Napoleon sweet cherry leaves naturally infected with a moderate isolate of cherry rugose mosaic strain (CRMs) of *Prunus* ring spot virus (PRSV), showing distortion and chlorotic blotching.

and Drake, but the AICs can be recovered from trees with bud failure symptoms (George Nyland, unpublished data). Trees of Nonpareil inoculated with a pure culture of AICs showed typical leaf symptoms of ring spot and calico the

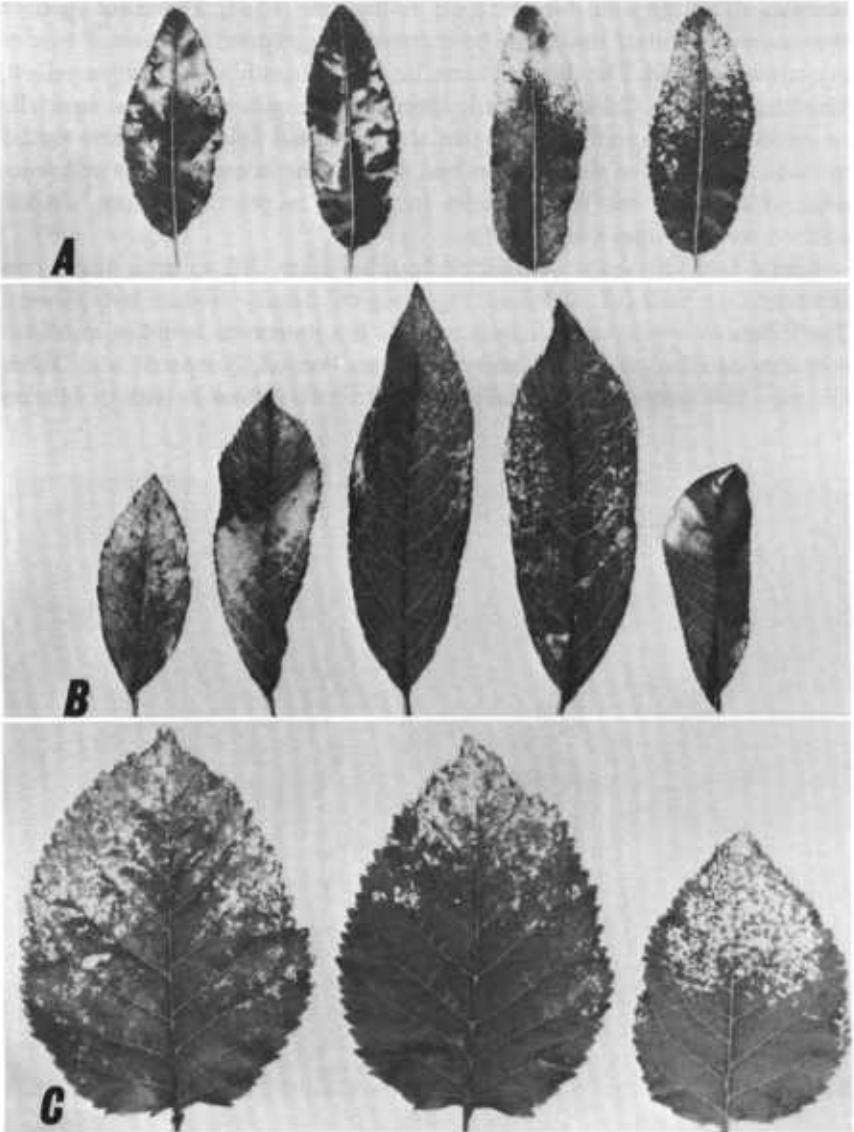


FIGURE 55.—*Prunus* ring spot. *A*, calico on leaves of Jordanola almond; Calico and chlorotic flecks on leaves of Elberta peach *B* and mazzard cherry *C* infected with almond calico strain (AICs) of *Prunus* ring spot virus (PRSV).

following year but only mild bud failure symptoms after 3 years (George Nyland, unpublished data). In time, Nonpareil trees in the field are seriously damaged by AICs. The coidentity of almond virus bud failure (744), almond calico (656), and peach mule's ear (745) is suspected because of their constant association, but research on this point is still in progress.

In sweet cherry, AICs induces initial necrotic ring spot shock symptoms, and in certain cultivars and seedlings AICs produces striking light-yellow to white chlorosis as lines, bands, rings, oak-leaf patterns or speckled mottle the season following inoculation and intermittently in subsequent growing seasons (fig. 42, B; fig. 45, C). Black Tartarian, Chapman, and Saylor show conspicuous calico symptoms. In all sweet cherry cultivars, the calico and ring spot symptoms are less obvious in older infections, but usually a few scattered individual leaves or occasionally all the leaves on individual spurs show symptoms. Symptoms induced by the AICs in sweet cherry resemble those of pinto leaf (367).

In peach, AICs inoculated by tissue grafts induces initial shock symptoms like those incited by the other strains of PRSV. Initial shock symptoms are followed by the development of chronic symptoms of pale-yellow blotches on leaves produced early in the spring. In older infections, these blotches may be difficult to find later in the season or at all (fig. 53, B). In the Central Valley of California, peach inoculated with AICs shows summer leaf spot symptoms like those described for other strains of PRSV.

In Shiro plum, AICs produces a very conspicuous speckled, bright calico spotting and prominent oak-leaf patterns (fig. 42, B). However, almond calico isolates react like those of cherry rugose mosaic and do not produce the "fish net" symptom in Shiro plum characteristic of PRSV isolates associated with plum line pattern. In Shiro-fugen flowering cherry, AICs reacts the same as other strains of PRSV, producing local necrosis around the inserted tissue.

Isolates of the AICs vary considerably. Some incite stronger leaf symptoms in almond and peach than other isolates, and they vary in the symptoms induced on a standard herbaceous host range. A few isolates have incited conspicuous chlorotic blotch symptoms in *Gomphrena globosa* that resemble those of cherry rugose mosaic in this host but without distortion of the leaves (George Nyland, unpublished data).

Other Strains of PRSV.—Several other strains of PRSV may exist, based on published reports (30, 347, 349, 483, 594). Excluded from PRSV would be those isolates that do not infect *Chenopodium* spp., but do incite dwarf symptoms on Italian Prune, yellow mosaic on squash, and tithonia, and local lesions on *Sesbania exaltata* (706). Included as a strain of PRSV would be Stecklenberger virus, based on symptoms in herbaceous and stone fruit hosts and on serological tests (348, 349, 483, 593). The virus described as chlorotic-necrotic ring spot virus (349) shows some properties similar to PRSV and PDV; but, based on host reactions, thermal inactivation point, and serology, it more nearly resembles PDV.

Transmission

PRSV is readily transmitted by tissue grafting. The virus is also transmitted mechanically from sap and pollen and by plant contact (196, 226, 231, 639, 725, 756). The virus is carried in seed from infected plants (123, 145, 147) and, at least in some cases, in seed produced on healthy trees (235, 666, 708). PRSV is carried in pollen of infected cherry (196, 708) and other *Prunus* hosts (725) and was transmitted from plant to plant by pollen of sour cherry (236). Pollen from trees of sour cherry and plum infected with PRSV, when applied to emasculated flowers on healthy trees, resulted in some infected seeds (666, 708). Some infected seeds were produced on open-pollinated field trees of mazzard and mahaleb in Oregon and Washington that presumably tested virus-free (205). In *P. pennsylvanica*, seed transmission occurred only when both the ovule and the pollen parents were infected (227). Open pollinated trees in California's tree registration program have consistently produced seedlings in which little or no virus has been detected so long as the ovule parent tests free of PRSV. Environmental conditions as well as the host plant may influence the incidence of seed transmission of virus.

PRSV and PDV spread from tree to tree of Montmorency grown in cages or in the field in experimental treatments under conditions that strongly suggested pollen as the vector. PRSV did not spread into exposed field trees from which all blossom buds had been removed. Rapid spread occurred in trees that were permitted to flower normally (236, 237). Spread of PRSV can be accounted for by tree-to-tree infection by pollen, by seed transmission, and by transmission through propagation practices. In spite of considerable effort devoted to a search, no insect vector of the virus has been found (640). Some viruses with characteristics similar to those of PRSV have nematode vectors (104).

Buds or bark chips infected with PRSV often die soon after they are inserted into healthy tissue of cherry, peach, and almond. Similar buds inserted into apricot, plum, or some other species of *Prunus* may heal in normally. Whether or not infected buds die depends on several factors such as the sensitivity of the host, virulence of a particular isolate, or the time of year the virus is introduced into the plant. Even if the inoculum tissue dies, transmission usually occurs. The time required for transmission is mostly a function of temperature (212), but to some extent it is also a function of host dormancy (217). At 30° C., transmission of PRSV occurred in 72 hours; at 18° C., 168 hours (212).

The time of infection influences the time of symptom appearance and severity. With sour cherry, inoculation with the RRSs of PRSV in mid-May, June, or July gave etch symptoms after bud-break on most of the trees except the inoculated branch, which had only mild secondary symptoms. Inoculation in late fall resulted in severe shock symptoms the following spring (182). In later experiments (181), trees inoculated at or near full bloom developed initial shock symptoms a year later, except in the inoculated branch, which showed only chronic symptoms. This is the usual condition seen in naturally infected

trees. When inoculations were made prior to full bloom, symptoms usually showed on the inoculated branch the same year and on the remainder of the tree the following year. This latter condition was not found in the orchards. These results add evidence that infection takes place at flowering time.

Characteristics of the Virus

PRSV is a polyhedron of variable size depending on the strain and the particular fraction of the virus measured. Isolates of the NRSs of PRSV had particles averaging 17 $m\mu$ (605) and 23 $m\mu$ (228). Virus particles of CRMs, AICs, and apple mosaic strain measured 21.5 to 28.7 $m\mu$, 20.4 to 23.3 $m\mu$, and 25.9 to 29.2 $m\mu$, respectively. Particles in the upper bands of sucrose gradient columns were consistently smaller and less infectious than those in the lower bands (422, 188). Two bands in a sucrose gradient seem to be characteristic for several strains of PRSV. Sedimentation constants for the two bands in sucrose gradient columns for several strains were 61, 72, 75, 88, and 95s for the upper zone and 87, 99, 105, and 117s for the lower zone (422, 188, 665).

PRSV is inactivated at 54° to 62° C. (227, 706) in vitro and 2 to 3 weeks in vivo at 38° (488). It loses more than one-half its infectivity in dilute cucumber sap (1:80) in 1 hour at 24°. The virus is stabilized in 0.02 M phosphate buffer at pH 8.0 with 0.01 M diethyldithiocarbamate (226, 228) and can be concentrated from buffered sap that is clarified with hydrated calcium phosphate gel. Centrifuging for 2 hours at 78,480 g. produces a clear, highly infectious pellet. Purified PRSV loses its infectivity rapidly in distilled water or when shaken vigorously in buffer (228).

The dilution-infectivity curve of PRSV is steeper than a theoretical one-hit curve. The slope of the curve is markedly influenced by adding infective or noninfective related virus to a dilute PRSV extract. The host inoculated also influences the slope. On *Dolichos biflorus*, each twofold dilution resulted in a three- to fourfold decrease in infectivity; on *Momordica balsamina*, an eightfold to tenfold decrease. Adding related virus to PRSV extracts disproportionately increased infectivity (229).

Strains of PRSV cross-react with each other in gel diffusion or micro-precipitin tests (232, 422, 495). Spur formation between an isolate of the apple mosaic strain of PRSV and PRSV from plum and cherry is reported (188).

Indexing for PRSV

Several methods are available to index efficiently for PRSV. Peach seedlings and Montmorency sour cherry were among the first indicator plants used to detect the virus in plants not showing symptoms (37, 152). Shiro-fugen flowering cherry (407, 445, 461) probably is the most reliable indicator host although peach may be equally as good under the best conditions. The Shiro-fugen test is made as follows (299, 300, 529): One or more buds or bark chips from the plant to be tested are inserted under the bark of a vigorously growing branch

of Shiro-fugen in the manner of budding. Vigorous growth during the test is very important to insure accuracy. The individual buds or chips are spaced about 2 inches apart. Under favorable growing conditions the results can be obtained about 30 days following budding. A necrosis of the tissue of Shiro-fugen around the inserted tissue indicates the presence of virus in the plant from which the tissue came. (fig. 56). The test does not distinguish clearly the strains of the virus or between PRSV and PDV. Indexing can be done as soon as the leaves of Shiro-fugen trees are fully expanded in the spring and can continue until growth ceases in the fall. Tests that are made just prior to cessation of growth in the fall but while the bark is still slipping easily can be read the following spring. So far as is known, only PRSV and PDV cause the local necrotic reaction in Shiro-fugen.

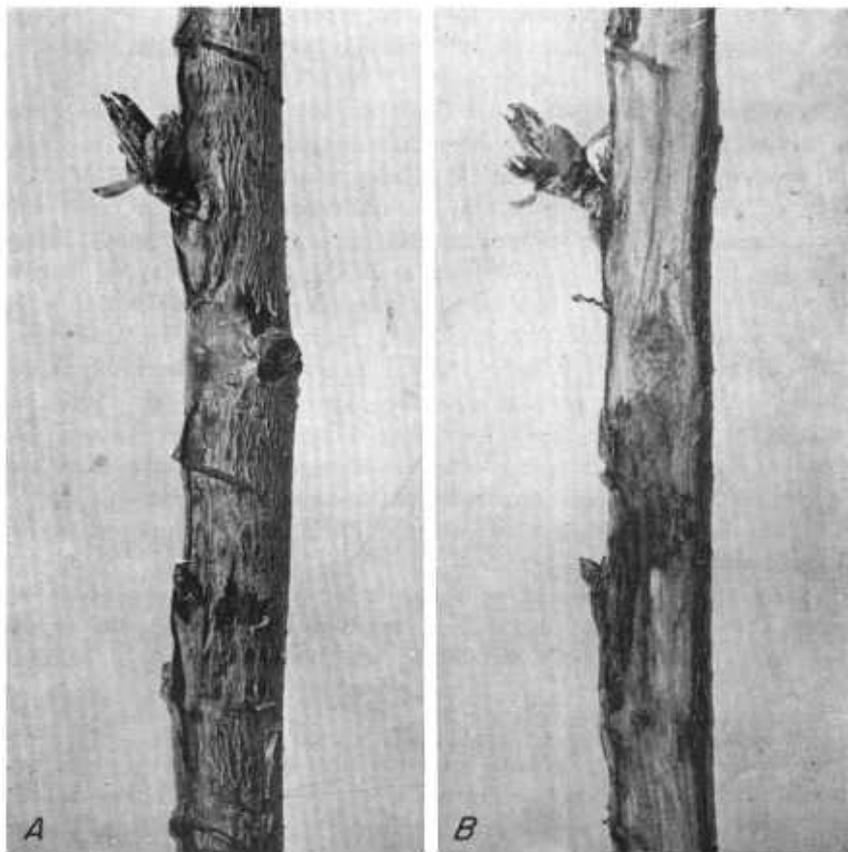


FIGURE 56.—*Prunus* ring spot. A Shiro-fugen cherry stem budded with two sweet cherry buds (*upper* virus-free and *lower* infected with *Prunus* ring spot virus): *A*, Without bark removed; *B*, with bark removed to show necrosis of the tissue adjacent to the infected bud and normal color under virus-free bud.

Prunus tomentosa is another useful indicator plant for PRSV as well as some other viruses (202, 210). Peach and *P. tomentosa* should be inoculated by tissue grafts late in the growing season or just prior to bud-break. Symptoms are best seen soon after bud-break. *P. tomentosa* can be used in the field but may be more sensitive as a greenhouse test plant.

Another useful indexing method for PRSV is sap transmission to cucumber or other herbaceous hosts (figs. 57 and 58). Leaves, roots, pollen, petals, or seed have been used as sources of the virus (93, 235, 246, 446, 447, 668, 725). Mechanical transfer of PRSV from leaves is most successful in early spring and becomes less efficient as the season progresses. Identification of some strains of the virus is possible by using selected herbaceous hosts (225, 495, 706).

Control Measures

Clean planting stock is the most important control measure for diseases caused by PRSV. In special cases, such as trees used for buds and scions or for seeds, roguing diseased orchard trees as soon as possible after they are detected is recommended. Spread of ring spot can be rapid in sour cherry and sometimes in peach. Limited evidence is at hand that spread is more rapid in Alamar and J. H. Hale than in Elberta or the common clingstone peaches. Alamar and J. H. Hale are self-sterile and therefore require cross-pollination. Sweet cherries, almonds, and some plums are also self-sterile; however, in the same areas that spread in self-sterile peaches was very rapid, little or no spread occurred in the other species (George Nyland, unpublished data).

Trees used as budwood sources or for seed production should be isolated as far as possible from infected fruit trees. One-half mile isolation has proved very effective in preventing spread of ring spot. In most cases, greater isolation is difficult to find for commercial orchards. Planting solid blocks of virus-free trees produced under some sort of certification scheme offers the best hope for the future. Selection of self-fertile cultivars that are less apt to be infected under field conditions offers some promise.

Remarks

The presence of PRSV as a contaminant of virus cultures obtained from infected orchard trees and the variability of the virus have caused a great deal of confusion among research workers in the past. The use of pure cultures of isolates alone and in controlled combination is rapidly clarifying the confusion surrounding the *Prunus* ring spot virus complex. As accurate information is obtained, relatedness of isolates that incite different diseases is being established. Thus, isolates inciting such diseases as cherry rugose, almond calico, Stecklenberger disease, plum line pattern, almond mosaic, rose mosaic, an initial symptom in hops, hop nettlehead, and apple mosaic all show relatedness of varying degrees. Questions about nomenclature remain, but these probably will be resolved on the basis of convenience and common usage. In the meantime, we need to learn more about the physical, chemical, and biological characteristics of particular isolates that incite the different diseases.

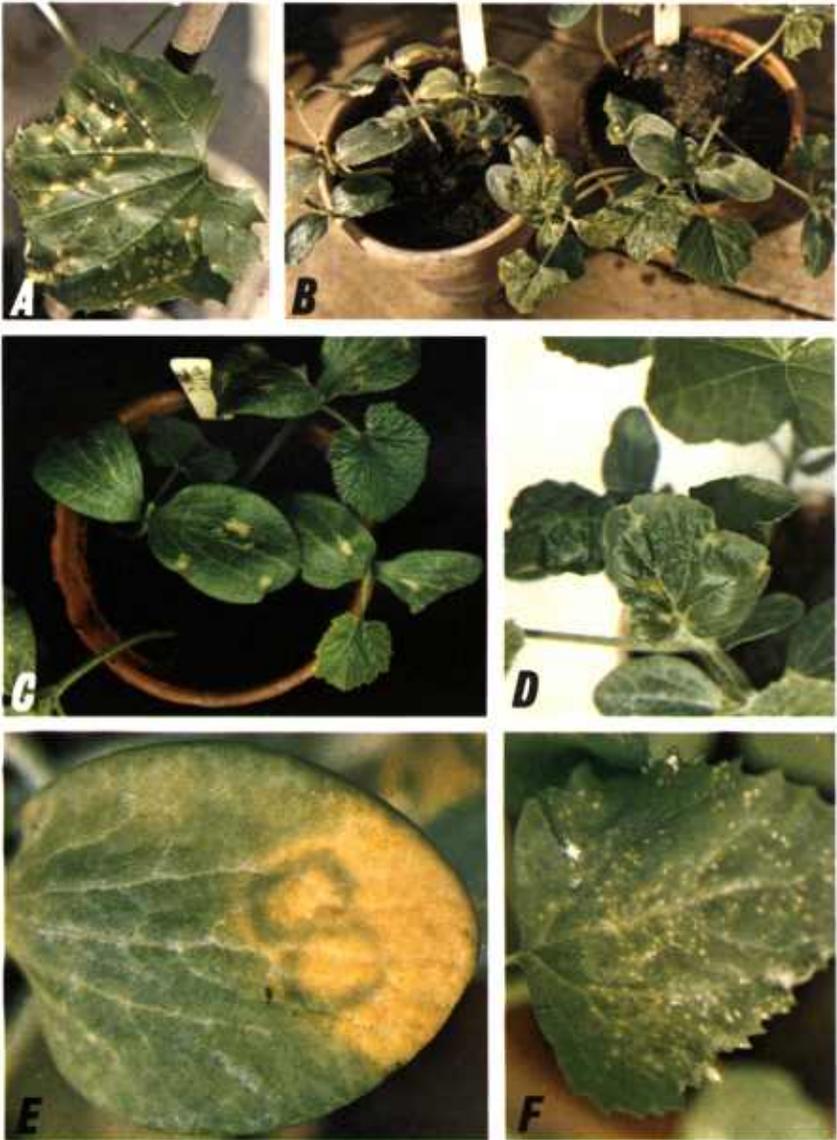


FIGURE 57.—*Prunus* ring spot. Symptoms incited on herbaceous plants by inoculation with *Prunus* ring spot virus (PRSV): *A*, Local lesions on *Momordica balsamina*; *B*, local lesions on cotyledons and systemic mosaic on leaves of cucumber; *C*, necrotic local lesions on cotyledons of Buttercup squash; *D*, limited systemic invasion of Buttercup squash leaves showing chlorotic blotches and deformed leaves; *E*, chlorotic local lesions and green rings on cotyledons of Buttercup squash that became evident 2 to 3 weeks after inoculation; *F*, necrotic local lesions on leaf of *Chenopodium amaranticolor*.

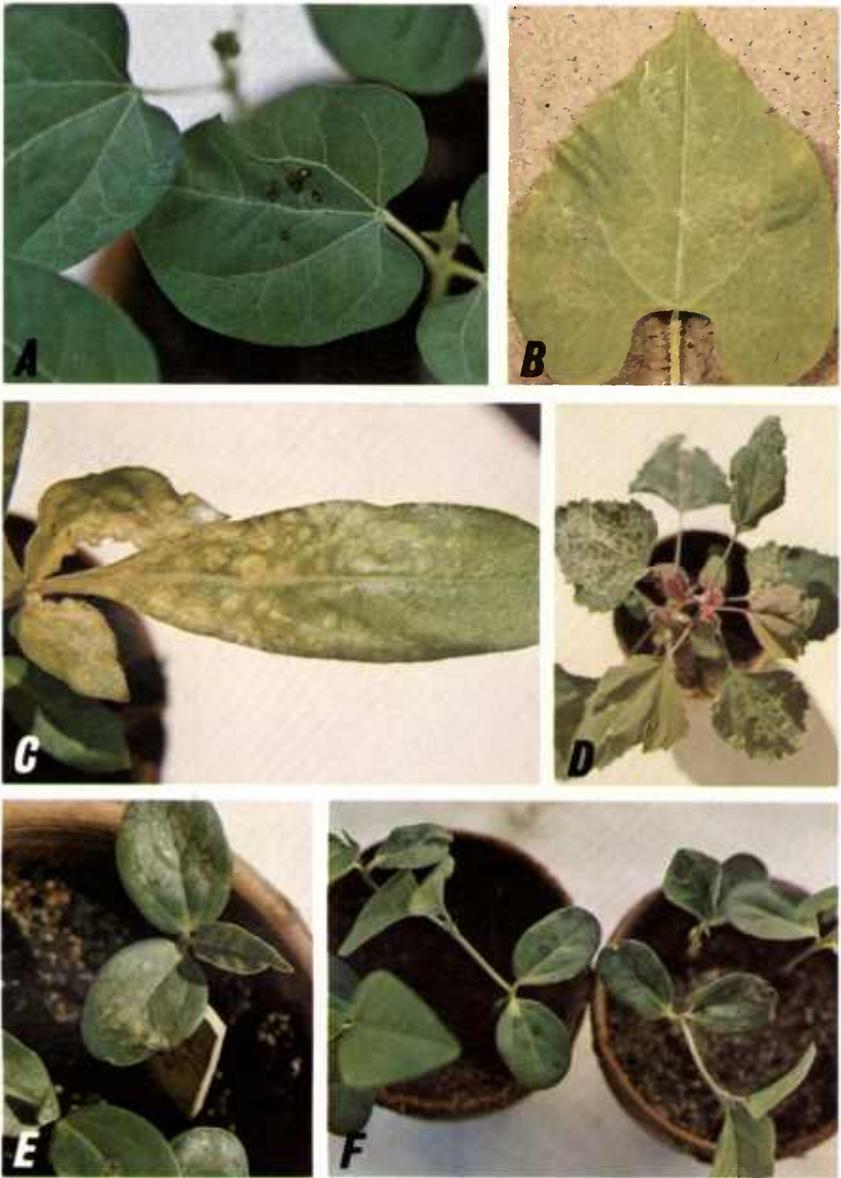


FIGURE 58.—*Prunus* ring spot. Symptoms incited on herbaceous hosts by inoculation with the cherry rugose mosaic strain (CRMs) of *Prunus* ring spot virus (PRSV): *A*, Local lesions on primary leaf of *Dolichus biflorus*; *B*, vein necrosis on leaf of Pinto bean; *C*, rings, blotches, and leaf distortion on leaves of *Gomphrena globosa*; *D*, chlorotic lesions and distortion on systemically infected leaves of *Chenopodium amaranticolor*; *E*, local lesions on cotyledons of *Cyamopsis tetragonaloba*. *F*, Local lesions on cotyledons of *Cyamopsis tetragonaloba* inoculated with recurrent ring spot strain (RRSs), *left*, and almond calico strain (AICs), *right*, of PRSV.

Also, we need to obtain and disseminate information about the advantages of clean stock where these exist and develop practical programs based on biological principles to minimize virus infection of clones during their development, testing, maintenance, and distribution.

RED SUTURE

EDWARD J. KLOS

Red suture occurs in Michigan and Maryland (31, 702); records indicate it was present in Michigan as early as 1911 (642). It was probably introduced into Maryland on infected nursery stock (128).

Red suture has been found on peach and Abundance plum. All peach cultivars tested were susceptible.

Red suture symptoms can be found on fruits and foliage (31, 117). Fruits ripen and soften on the suture side several days prematurely, while the other side is green and hard. Frequently, the suture side is swollen, rough, or bulged (fig. 59). Red suture flesh is coarse, stringy, and watery. The early ripening side of red fruited cultivars is blotched with dark red or purple, which is evident on the apexes of the bumps. Yellow fruited cultivars, such as Gold Drop, have a deeper yellow on the suture side. The fruit flavor is often insipid. The suture side will bruise or break down more rapidly in transit or on the shelf.

Red suture leaves have a yellowish-green to greenish-bronze appearance, which is often overcome by nitrogen fertilization. The color difference of diseased and healthy trees is noted at certain times of the season, particularly a few weeks after petal fall or just before harvest when diseased trees take on early fall coloring. The buds of many 2-year-old branches grow into short, spurlike outgrowths, resulting in a clustering of the leaves along the branch. On normal trees, a few buds remain dormant, others grow long shoots, and only a few develop into spurlike growths. A tree diseased for several years will have an open center. Diseased trees may live for 8 years or more.



FIGURE 59.—Peach fruits showing enlarged, reddened suture characteristic of red suture.

Cation and Bennett (128) noted on Abundance plum that the symptoms of red suture are similar to those caused by peach yellows virus. Diseased trees have lighter colored foliage and fruit ripens prematurely. Red June and Wickson plum (*P. salicina* × *P. simonii*) also exhibit severe symptoms of red suture when infected by peach yellows virus. Red suture virus (RSV) alone had little effect on these cultivars.

Symptoms on mature trees appear in the second year after infection. Nursery trees propagated from diseased buds do not show symptoms during the first year of growth and only sporadically in the second year. Transmission has been successful by grafting with bark tissue with or without buds from diseased trees.

Kunkel (387) found that red suture virus was inactivated when bud sticks were immersed in a water bath at 50° C. for 3 minutes. Peach yellows and little peach viruses have similar inactivation points. On the basis of disease symptoms and virus inactivation temperature, red suture is probably related to peach yellows and little peach. The restricted distribution of red suture would indicate that, if it is a mutant of the peach yellows group, it rarely occurs in nature.

Red suture can be controlled by periodic orchard inspection and eradication of diseased trees. Virus-free budwood and rootstocks are recommended in peach propagation. The disease has been of little economic importance in recent years. Effects of 2, 4-D and 2, 4, 5-T on peach fruits frequently have been mistaken for red suture.

ROSETTE MOSAIC

EDWARD J. KLOS

Causal Virus

Rosette mosaic virus (RMV).

Synonyms

Peach rosette mosaic. The name refers to two of the prominent symptoms on this crop.

History and Distribution

This disease has been present in Berrien County in southwest Michigan since at least 1917 (118, 121). It has been limited to a few scattered orchards in that county. Similar diseases were reported in New York (305) and Italy (265).

Economic Hosts

All peach cultivars tested were susceptible, and the severity of the reaction varied with the cultivar. The following peach cultivars are listed in the order of the severity of effect of the disease: South Haven, Halehaven, J. H. Hale, Elberta, Carmen, Golden Jubilee, Kalhaven, Ambergem, and Gold Drop.

Other Hosts

Italian Prune (*Prunus domestica*) and Wickson plum (*P. salicina* × *P. simonii*) developed symptoms following graft inoculation with RMV. Damson plum, and Burbank, Red June, and Abundance plums (*P. salicina*) appear to be symptomless carriers of the virus (124). Concord grape is now known to be susceptible to infection by RMV (377 and H. Dias, unpublished data).

A number of herbaceous plants have been infected by sap transmission, including *Chenopodium amaranticolor*, *C. quinoa*, *Nicotiana tabacum*, White Burley tobacco, *N. glutinosa*, common bean, cowpea, petunia, and tomato. *Gomphrena globosa* is a symptomless host (H. Dias, unpublished data).

Symptoms

The symptoms in peach may consist of delayed foliation; chlorotic mottling of early formed leaves, generally accompanied by distortion (fig. 60); and a shortening of internodes (fig. 61), which frequently crowds the leaves into the rosettes from which the disease derives its name (fig. 62).

When chlorosis of the leaves occurs, it is evident early in the growing season. Chlorotic areas vary in size and shape and range from yellow or cream color to translucent. Tissue growth in these areas is retarded and distortion results. Less definite retardation gives a wavy or undulating leaf margin. Leaves formed later in the season on affected trees appear more normal in size and shape but actually are about two-thirds as wide as leaves on unaffected trees. Affected trees are darker green than normal trees. A variable number of normal branches may be interspersed with affected ones on an infected tree. Inoculation of healthy

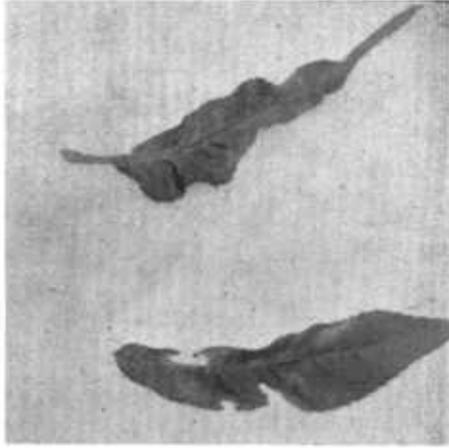


FIGURE 60.—Misshapen leaves with chlorotic areas from a peach tree affected by rosette mosaic.

trees with buds from such normal-appearing branches did not result in transmission of RMV.

Italian Prune showed symptoms similar to those of prune dwarf when inoculated with RMV. Symptoms varied from strap-shaped to slightly dwarfed, roughened, and thickened leaves. Wickson plum did not express symptoms until the third or fourth year after inoculation. The leaves were smaller than normal, and the twigs showed rosetting. Damson plum did not develop definite symptoms.

Concord grape showed delayed foliation with small, distorted, chlorotic leaves and vine decline. A berry-shelling symptom may be present. Many affected vines die in a short time.

Mechanical transmission of RMV by using sap from young infected leaves of Concord grape or peach gave the following symptoms on herbaceous plants: *Chenopodium amaranticolor* and *C. quinoa* showed systemic mottle and leaf deformity with stunting and distortion of the shoot tip 7 to 10 days after inoculation; in *C. quinoa*, these symptoms are followed by severe stem twisting, epinasty and frequently by death of the plant; in White Burley tobacco, systemic mottling with chlorotic ring spots are apparent in 8 to 15 days; bean, cowpea, *Nicotiana glutinosa*, petunia, and tomato are systemic hosts and show only mild symptoms; although *Gomphrena globosa* is symptomless, the virus can be recovered from inoculated and apical leaves.

Transmission

Transmission of RMV has been accomplished by grafting, by transfer of soil from diseased to healthy trees, by planting healthy trees in a location previously

occupied by diseased plants, by the nematode *Xiphenema americanum* (368), and by seed (H. Dias, unpublished data).

Dias has transmitted the virus from peach and grape to various herbaceous hosts by mechanical sap inoculations.

The incubation period of RMV in peach varied from a few weeks under optimum growing conditions in the greenhouse to 9 to 21 months in the field. The length of time required depended on varietal susceptibility and growth stage of the trees at the time of infection. Transmission in contaminated soil took 10 months if transmission was attempted prior to July 15. Trees planted in diseased soil in the spring showed symptoms the next spring, but trees planted in the fall did not show symptoms until the second spring. On seedlings 18 to 24 inches tall, the shortest incubation period was 12 to 16 days when rapid transmission techniques were used (306). With the defoliation technique, 14 to 19 days were required for symptom development, and with the shading technique, 18 to 21 days (311).

Characteristics of the Virus

Dias (unpublished data) found that purified virus preparations of peach rosette mosaic virus and grape decline have identical polyhedral particles of approximately 28 $m\mu$ in diameter. Hildebrand was unable to inactivate RMV by soaking the bud sticks in a hot-water bath at 50° C. for 20 to 22 minutes (124), but Dias has shown that the thermal inactivation point is 10 minutes at 65° to 68°.



FIGURE 61.—Dormant branch of peach tree affected by rosette mosaic showing severely shortened internodes.



FIGURE 62.—Peach branches with severe rosetting symptoms of peach rosette mosaic.

Control Measures

Removal of diseased trees and fumigation of tree sites for nematodes before replanting is recommended (377). Planting of known virus-free stock of peach, plum, and Concord grape is also suggested.

Remarks

Rosette mosaic reduces the production of individual trees. However, this peach disease is found in a limited area in one township so that the total loss is rather small.

The symptoms of peach rosette mosaic are similar in some respects to prune dwarf and peach mosaic. Peach is more severely affected by rosette mosaic than by prune dwarf, whereas Italian Prune is less markedly affected by rosette mosaic and more severely affected by prune dwarf. Rosette mosaic does not cause dieback or leaf spot symptoms in peach as does *Prunus* ring spot.

Infection by RMV has not resulted in blossom break, fruit distortion, shot hole, or the marked and general chlorosis associated with peach mosaic.

STUBBY TWIG

H. KEITH WAGNON, HAROLD E. WILLIAMS, and JACK A. TRAYLOR

Causal Virus

Stubby twig virus (STV).

Synonyms

False yellow leaf roll.

History and Distribution

The disease was first noted in California in 1952 when its symptoms caused confusion in diagnosing of the yellow leaf roll strain of western X-disease (563). In 1958, it was reported (695) to be a virus disease which was widespread in peach and nectarine plantings in California. Certain cultivars had become infected prior to or shortly after their introduction, and the disease had been spread by contaminated propagating stocks. Published reports on the disease have led to the belief that its occurrence is restricted to California, but it has been noted also on trees in Missouri and Oregon and on trees propagated in several other States and shipped to California. Symptoms of the disease also have been observed on peach in both Italy and Turkey. The disease may go undetected because peach and nectarine trees infected with the virus apparently remain symptomless under certain weather conditions.

Economic Hosts

The virus is known to cause disease symptoms only in peach and nectarine.

Other Hosts

Transmission studies have shown that the following hosts could be symptomless carriers of the virus: Blenheim apricot; Jordanolo almond; Wickson, Myrobalan 29C, marianna and damson plum; and desert almond.

Symptoms

The most conspicuous overall feature of an STV-infected tree is the scattered occurrence of shoots or branches with pale-green to yellowish leaves, or both. Three types of leaf symptoms and one type of twig symptom occur on infected trees. Leaves of two of the symptom types, designated primary and secondary, are chlorotic and are most evident in late spring and summer; leaves of a third type are not chlorotic but chimaeralike and are seen only in the spring. Because the chlorotic symptoms can be confused with symptoms due to other causes, the spring leaf symptom, when present, is most reliable for diagnosis. The leaf symptoms vary markedly with weather conditions, with the cultivars involved, and with orchard care. Some symptomatic shoots and branches may be removed by pruning. The three types of symptomatic leaves commonly are retained until time of normal defoliation.

The primary chlorotic leaves which follow initial infection, and which may recur annually, develop on somewhat vigorous, current season shoots (fig. 63).

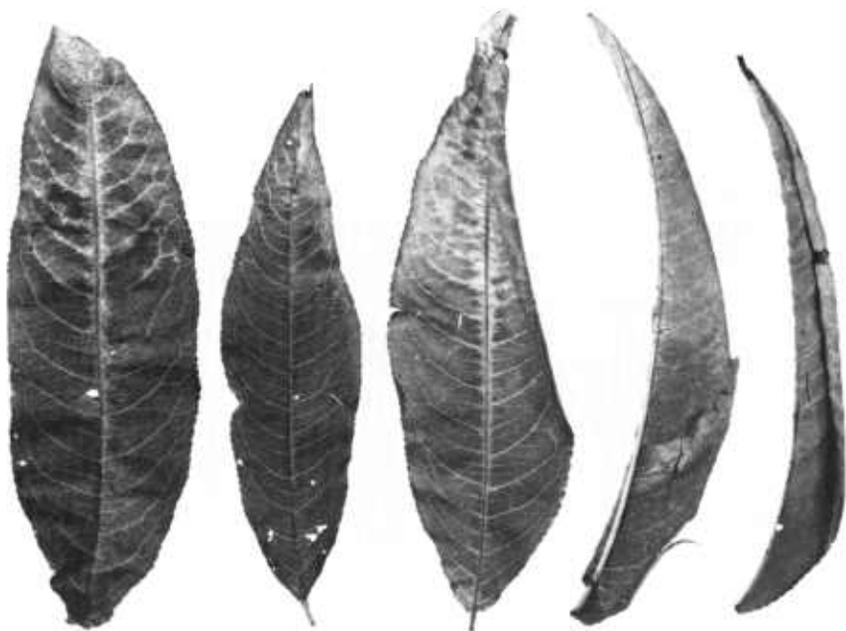


FIGURE 63.—Chlorosis and upward rolling of margins of peach leaves affected by stubby twig.

These leaves, commonly associated with shortened internodes, are pale green to light yellow with margins slightly rolled upward and inward toward the midrib. Affected shoots may have normal-appearing foliage above and below symptomatic leaves. The chlorosis first appears at the leaf apices and extends toward the bases. As the season advances, the veins sometimes become swollen, the yellow usually changes to reddish orange, and the leaf margin and some veins near the apices become necrotic.

The spring chimaeralike chlorotic leaves develop on stems that are 1 year old or older on trees infected 2 or more years. Foliation is retarded on affected twigs and branches. The spring leaf symptoms become distinct in early April; they may be of normal size and shape, but commonly they are subnormal in size, are asymmetric, and have irregular margins (fig. 64). Contrasting pale- and darker-green shades appear side by side in small sectors on angular areas of the leaf blade, giving a marbled effect. This leaf sectoring, although diagnostic for STV, is transitory. As the season progresses, the marbling becomes less distinct; by midsummer it is difficult to recognize. These leaves are seldom produced in abundance and frequently are detected only after considerable searching. Twigs and branches that bear these spring leaf symptoms may or may not exhibit other symptoms of the disease. The secondary chlorotic leaf

symptom is the most common but does not become distinct before June. Twigs and branches which bear the delayed, pale-green, and subnormal-sized leaves are characterized by stubbiness, brittleness, lateral bud failure, and, sometimes, dieback (fig. 65).

With the advance of the growing season, the small or subnormal-sized chlorotic leaves become progressively more chlorotic, and those near the shoot tip may develop red to reddish-brown spots, which may easily be mistaken for symptoms of nitrogen deficiency. By July, affected twigs or small branches with their yellowish foliage become conspicuous, and, at first glance, may be confused with damage resulting from girdling or simple mechanical breakage. When affected twigs and branches are confined to one part, the tree may appear lopsided. Vigorous, normal-appearing shoots often develop below the dead or retarded terminals. The current season vigorous shoots tend to mask the older, affected twigs, but before the end of the season, the primary chlorotic leaves become evident.

Affected twigs bear fewer fruit buds. Mature fruits on these twigs are substandard in size and flavor. The total effect on fruit production depends upon the number of affected twigs in a tree.

Transmission

The virus has been transmitted only by grafting. Attempts to mechanically transmit STV to cucumbers by using pollen from infected trees have been inconclusive.

Characteristics of the Virus

Information on virus morphology and serological relationships is lacking. Stubby twig is caused by a yellows type of virus, which produces symptoms



FIGURE 64.—Chimaeralike spring symptoms in peach leaf affected by stubby twig.

that resemble western X-disease. Young peach trees inoculated with both viruses produced symptoms of both diseases, and the trees showed more decline than did comparable trees inoculated with either virus alone..

Trials indicate that STV is inactivated within 5 but never less than 3 weeks in air at 38°C. (George Nyland, unpublished data).

Control Measures

Observations indicate that there is probably no field spread of the disease in peach and nectarine. The disease is best controlled by using STV-free propagating stock.

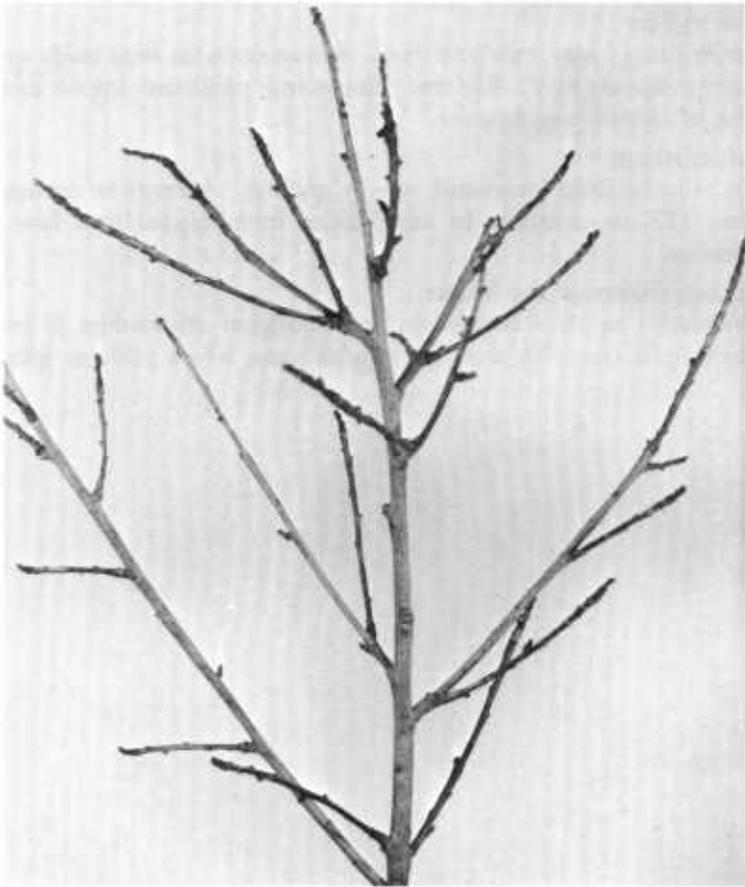


FIGURE 65.—Dormant peach branch affected by stubby twig, showing stubby growth and terminal dieback.

Remarks

STV is not lethal to a tree when present alone, but does, under some conditions in California, cause a debilitation of the tree. In commercial plantings, its effects vary with cultural conditions. The severity of the disease can be increased when STV occurs in combination with some strains of *Prunus* ring spot virus. At least two commercial peach plantings have been removed because of the reduction of salable fruit as a result of a high incidence of the disease. Generally, the overall effect of the disease in the warm interior valleys of California is to depress normal vegetative growth in varying portions of the tree. Fruit production is reduced in proportion to the failure of flower buds and blossoms. Although symptoms are restricted to certain parts of the tree, the virus is completely systemic because progeny trees grown from buds of healthy-appearing shoots have always been infected. The virus is symptomless in growing nursery stock.

Superficially, symptoms of stubby twig resemble those produced by strains of X-disease virus (563). Pale-green to yellowish leaves may be produced by both viruses; however, the development of necrotic spots from water-soaked areas, which is symptomatic of X-disease, has not been observed with stubby twig. The yellowing of affected leaves caused by STV can readily be distinguished from the yellowing which is characteristic of peach leaf roll (500); yellowing in stubby twig begins at the leaf tip and extends progressively toward the base; in yellow leaf roll it develops more or less simultaneously and uniformly throughout the leaf. Vein swelling of affected leaves, a consistent symptom incited by strains of X-disease virus, is much less prominent in stubby twig. Stubby twig is readily distinguished from yellow leaf roll by the location of symptomatic leaves on current season shoot growth. In stubby twig, the symptomatic leaves may have normal-appearing foliage above and below them; in yellow leaf roll the symptomatic leaves are not subtended by normal-appearing foliage. Leaves of stubby twig trees never fall prematurely as with most X-disease. At least three features in the symptomatology of stubby twig are not seen in X-disease—(1) brittleness of infected twigs; (2) red to reddish-brown spots in some leaves, which may be mistaken for nitrogen deficiency; and (3) the diagnostic leaf sectoring that occurs on certain leaves that develop in early spring. The diseases are very different in their reaction to heat therapy.

WEAK PEACH

GLENN KENKNIGHT

At Fort Valley, Ga., 142 weaker-than-average orchard peach trees (*Prunus persica*) were indexed in 1952. One tree of Erli-Red-Fre yielded a virus isolate that, when transmitted by patch grafts or by scions, was able to induce a "weakness" syndrome in trees of several peach cultivars grafted on Lovell peach seedlings (362). The isolate was named weak peach virus (WPV).

Symptoms were induced within 1 to 4 years on trees of Early Hiley, Elberta, Fairhaven, Prairie Rose, and Redcrest; but not on Cardinal, Hiland, Keystone, and Redcap peach. *Prunus mahaleb* can be infected. Symptoms similar to those caused by WPV have been induced in peach by graft transmission from a stunted narrow leaf plum (*P. angustifolia*) and a Concord grape (*Vitis labrusca*). Symptoms have been recognized only in peach. Trees on peach rootstocks show annual delayed onset of growth in spring, progressive stunting, dieback, and summer chlorosis. Trees on mahaleb rootstocks show only delayed onset of growth in the spring.

The disease apparently is not soil borne. Percentage transmission by grafting is very high. Incubation periods appear to vary with age of inoculated tree. WPV-inoculated, first-year Early Hiley trees showed symptoms in 3 to 4 years; 6-year-old trees, in 2 years; and 8-year-old trees, in 8 months. Transmission by bark-patch grafts was not affected by treatment of scions in hot water for as much as 30 minutes at 50° C.

Peach orchards in central Georgia now last half as long as they did early in the history of commercial fruit production in this region. Inadequate investigation has been made of the possible role of debilitating viruses in the short life of peach orchards growing on land that has been replanted to peaches many times. The symptoms induced by WPV closely resemble those caused by the root rot fungus, *Clitocybe tabescens* (Scop. ex Fr.) Bres., which has been detected in as many as 80 percent of stumps in uprooted orchards. When nursery-stock peach trees were inoculated by placing *Clitocybe*-invaded peach stumps in the planting holes, the trees developed symptoms strikingly similar to those caused by WPV, whereas control trees, with sound peach stumps in the planting holes, grew normally. The similarity of symptoms induced by WPV and *C. tabescens* provides uncertainty about the degree to which WPV contributes to the problem of orchard debilitation.

X--DISEASE

R. M. GILMER and E. C. BLODGETT

Causal Virus

X-disease virus (XDV).

Synonyms

XDV is a highly variable virus, and its numerous strains vary considerably in their severity and symptomatology in economic hosts. Because of this variability and the wide range of environments in which the virus occurs, the diseases incited have been described under numerous names: X-disease (628), yellow-red disease or virosis (313, 504), western X-disease (567), western X little cherry (579, 585), western X red leaf (575) wilt and decline (581), red leaf (575), cherry buckskin (548), peach leaf casting yellows (657), peach yellow leaf roll (598), and small bitter cherry (410).

History and Distribution

Detailed accounts of the history and general distribution of the disease have been presented previously (563, 630). X-disease was initially discovered in California in 1931, where it was described as cherry buckskin (548). In 1933, the disease was observed on peach in Connecticut, where it had probably been present for several years before discovery (628, 629). Blodgett (45) observed X-disease of peach in Idaho in 1936. Within a few years, the disease was found in all of the New England States, New York, New Jersey, Pennsylvania, Ohio, Indiana, Illinois, Michigan, Wisconsin, North Dakota, California, Oregon, Washington, Idaho, Colorado, Arizona, Utah, British Columbia, Ontario, and New Brunswick (45, 111, 208, 313, 346, 410, 548, 567, 578, 607). The disease has been observed in chokecherry in western North Carolina and eastern Tennessee (R. M. Gilmer, unpublished data), but does not occur in the peach-growing areas of South Carolina, Georgia, Arkansas, or Texas.

Earlier publications maintained a distinction between the eastern and western forms of X-disease (563, 578, 630). The only direct comparisons of eastern and western X-disease were made by Hildebrand (308), who concluded that the causal viruses were distinct but probably related because of variations in the rates of symptom development and in type of symptoms. This distinction was apparently supported by differences in orchard epidemiology. Spread of X-disease in eastern orchards was closely linked with the presence of diseased chokecherry, but in western orchards spread occurred even in locations where chokecherry did not occur.

Reeves et al. (563), however, pointed out the wide range of symptoms present in different forms of western X-disease; these differences were as marked as those between the western and eastern forms of the disease. These authors also suggested that differences in orchard epidemiology could be

explained most plausibly by differences in vectors in the two areas. These same conclusions were reached independently in Eastern United States (260).

Historically, identification of X-disease in new localities has been based on its appearance in chokecherry. This relationship was noted by Stoddard (629) in the East and by Richards et al. (578, 579) in the West. The disease is widely distributed in chokecherry and occurs in this host even in areas where peaches and cherries are not grown (563). In Santa Barbara County, Calif., X-disease occurs naturally in hollyleaf cherry (H. E. Williams, unpublished data).

Economic Hosts

Peach, nectarine, Japanese plum, sour cherry, and sweet cherry.

Other Hosts

X-disease occurs naturally or has been transmitted experimentally to almond, apricot, mahaleb cherry, Korean cherry, western sand cherry, bitter cherry, hollyleaf cherry, Manchu cherry, wildgoose plum, and several plum hybrids (259, 550, 563, 629, and H. E. Williams, unpublished data). Its important wild reservoir hosts are common and western chokecherry. The disease occurs without evident symptoms in flowering cherry (259, 560), pin cherry, American plum, damson plum, and European plum (259).

Species apparently immune to some strains of the virus are fenzi almond, desert apricot, desert peach, David peach, black cherry, myrobalan plum, Klamath plum, and beach plum (259, 550, 629, 630).

XDV has been transmitted experimentally through dodder to carrot, celery, parsley, periwinkle, strawberry, tobacco, and tomato (98, 260, 332) and has been recovered from naturally infected milkweed (245).

Symptoms

Peach.—In the East, the foliage of infected peach trees appears normal for about 6 weeks after growth commences in the spring. At that time, yellow or red irregular blotches develop in the leaf blades, and affected leaves roll upward longitudinally (figs. 66 and 67). Discolored blotches may occur anywhere on the leaf lamina and are not restricted by the veins; there may be few to several blotches per leaf. Shortly after blotches appear, the remainder of the leaf blade becomes chlorotic and brittle; many of the discolored areas drop out, giving the affected foliage a tattered appearance. At this stage, the diseased trees appear distinctly pale green. Somewhat later, the affected leaves abscise and drop, leaving the terminals bare with only a small persistent rosetted tuft of leaves at the ends.

In the West, the syndrome is generally similar, but the irregular blotches are often pale green or slightly watersoaked and drop from the leaf blade before any red or yellow coloration develops. The affected leaves remain pale green and are cast, the older leaves falling first. Later in the season, newly developed blotches may become necrotic and may or may not fall from the

lamina. In some cultivars, foliage of affected branches or trees may become distinctly red.

The yellow leaf roll strain of XDV incites the same initial symptoms as the common strain, but summer symptoms are more conspicuous. The affected shoots continue to grow throughout the summer with only slight defoliation. Infected trees are conspicuous for their yellow, upward-rolled leaves often with enlarged prominent veins (fig. 68). Trees rarely survive for more than 3 years after infection.

In trees newly infected with the common strain of XDV, only one or two branches commonly develop symptoms, and the foliage and fruits on the remainder of the tree may be normal. In succeeding years, branches with symptoms appear at random throughout the tree, often interspersed with branches with normal foliage and fruits. Branches with symptoms usually show considerable dieback from low temperature injury during the dormant period. Large trees may persist in a generally unthrifty condition for several years following infection, but smaller trees often die within 1 to 3 years after the appearance of the initial symptoms.



FIGURE 66.—Elberta peach leaves showing irregular yellow and red blotches, early season symptoms of X-disease.



FIGURE 67.—Terminal rosetting and leaf rolling in peach infected with X-disease virus in New York.



FIGURE 68.—Peach tree, *foreground*, infected with yellow leaf roll strain of X-disease virus, showing yellowing and upward-rolling of leaves.

The fruits on branches with symptoms commonly abort and drop soon after the foliage symptoms appear. If the fruits persist, they are usually more pointed than normal (fig. 69), with shrivelled nonviable seeds and internal discoloration around the pits. Although such fruits often color prematurely, their flavor is bitter and unpalatable. Large trees with chronic infections set few or no fruits.

Sour cherry.—The initial symptoms of X-disease are apparent in the fruits, which are smaller than normal, often distinctly pointed, and pale red or greenish white. Some normal fruits are often interspersed with fruits with obvious symptoms on the same branches. Affected fruits persist in a half-ripe stage long after normal fruits have matured.

In the early stages of infection, distinctive foliage symptoms are not apparent. In late summer, a few leaves on infected branches may be rusty in color and develop traces of red or orange along the midrib. In the chronic stage of the

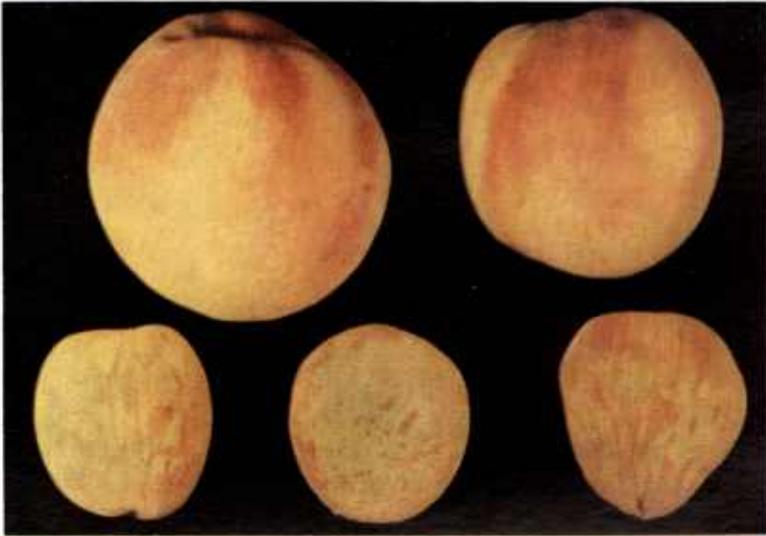


FIGURE 69.—Small and misshapen fruit from Elberta peach tree affected by X-disease, *bottom*; normal Elberta fruit, *top*.

disease, terminals are rosetted, the foliage is a pale green, and considerable dieback of twigs and branches is evident.

As in peach, the initial symptoms of X-disease are often confined to only a few branches, but in succeeding years symptoms gradually appear in the remaining portions of the tree.

Trees on mazzard rootstocks may persist for a number of years with chronic infections of XDV but are economically worthless. Trees on mahaleb rootstocks are generally short-lived after infection. They frequently wilt and die in midsummer under conditions of temperature and moisture stress.

Sweet cherry.—The X-disease syndrome in sweet cherry is quite similar to that described in sour cherry. The initial symptoms are usually evident on the fruits, which are small, pointed, often flat-sided, and pale red to greenish white (fig. 70). Affected fruits are often initially confined to a few branches, while the remainder of the tree bears normal fruits.

Terminals of affected branches are commonly slightly rosetted; in late summer their leaves, which may be slightly undersized, become bronze or rusty green. A few leaves may become red or orange along the midrib. In Utah, the leaf stipules are more broad and elongate than normal (566), but this symptom is less common or entirely absent in other areas. A late flush of growth may develop from normally dormant terminal buds on infected trees.

In California, two well-defined strains of XDV occur in sweet cherry, the Green Valley strain and the Napa Valley strain. The Green Valley strain is

apparently identical with the common strain of XDV present elsewhere in the West. The Napa Valley strain differs in that many of the fruits on infected sweet cherry trees on mazzard roots are cast just before or at the time normal fruits are ripe. These affected fruits are almost normal in shape and are borne on pedicels of normal length.

Leaves on trees infected with the Napa Valley strain roll upward at the margins, and a prominent bronze coloration develops on the exposed abaxial surfaces by early June. Infected limbs are apparent at considerable distances. The Napa Valley strain of XDV is restricted to Napa and Sonoma counties of California.

Trees on mazzard rootstocks may survive for a number of years after they are infected with X-disease virus, but the virus gradually spreads throughout the tree and affects most of its fruits. The infected trees are generally unthrifty and more subject to dieback from winter injury. Trees on mahaleb rootstocks decline rapidly when infected and often wilt and die in midseason.

Chokecherry.—Newly infected chokecherry trees show a definite but transient delay of growth initiation in the early spring but appear almost normal until about 6 to 8 weeks after growth commences. At this time, the foliage gradually becomes bright orange or red (fig. 71). Many of the affected leaves drop but others are retained. A second flush of growth may appear in late July or August

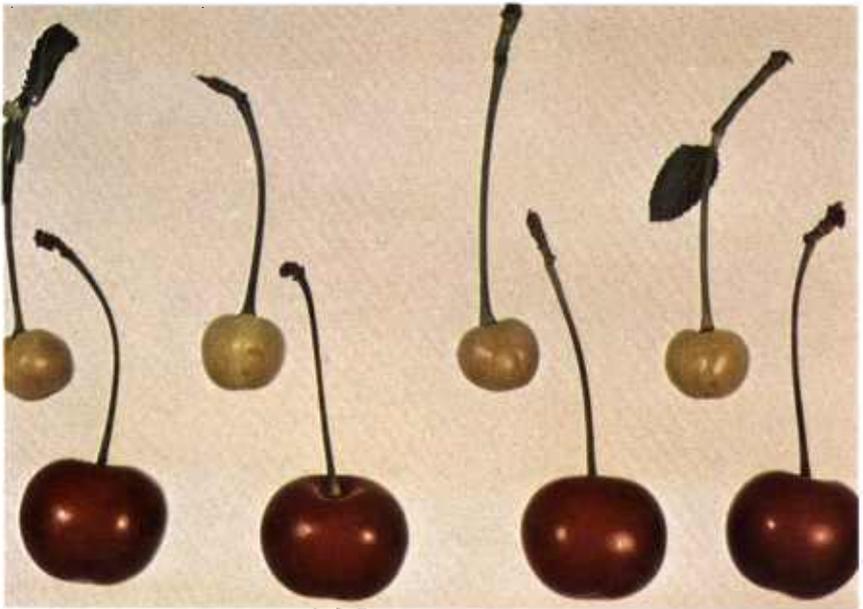


FIGURE 70.—*Top*, immature fruits of May Duke cherry infected with X-disease virus. *Bottom*, healthy fruits of May Duke cherry from an uninvaded portion of the same tree.

from terminal and axillary buds that break dormancy. This growth is initially yellowish-green, but gradually turns red as the season progresses.

Fruits on newly infected trees are pointed and remain pale red without maturing.

In succeeding years, growth of infected trees is very rosetted, and the foliage is duller in color, often yellow instead of red. Leaves are fewer in number and smaller in size. Many twigs and branches die, and few or no fruits are produced. Infected chokecherry trees decline rapidly and commonly die within 3 to 4 years after infection, although they may survive slightly longer in areas with mild winters.

Transmission

XDV usually is readily transmitted by budding or grafting, although a fairly high percentage of buds from peach trees infected with the common strain may not carry the virus, possibly because of irregular distribution of XDV in the infected trees. The yellow leaf roll strain of XDV is very invasive and usually completely systemic in infected peach trees, so that transmission is easily accomplished by budding. Besides differences in the invasiveness of XDV strains in peach, other factors may influence ease of transmission. Successful



FIGURE 71.—Chokecherry tree affected by X-disease, showing characteristic bright orange to red foliage in the second year of symptoms.

transmissions decline markedly if infected peach buds are not used promptly (E. L. Reeves and E. C. Blodgett, unpublished data). Transmissions from buds of trees with longstanding chronic infections is often difficult, probably because of the failure of the debilitated tissue to unite (R. M. Gilmer, unpublished data). The presence of *Prunus* ring spot virus in XDV inoculum may also prevent tissue union and transmission of XDV.

XDV may be transmitted from infected stone fruits to a variety of herbaceous hosts with dodder (*Cuscuta* spp.) (260, 391, and D. A. Slack, unpublished data). Transmission from one herbaceous host to a second is also readily achieved with dodder. But, in spite of repeated attempts by several workers (260, 391), transmissions between stone fruits or from herbaceous plants to stone fruits with dodder has not been achieved.

Although Stoddard (629) and Hildebrand (308) suspected that XDV was transmitted in the field by insects, Wolfe et al. (750, 751) were the first to demonstrate unequivocal transmission of XDV by a leafhopper, *Colladonus geminatus* (Van D.). Jensen et al. (333) confirmed transmission by this insect. Anthon and Wolfe (17) later discovered three additional leafhopper vectors: *Scaphytopius acutus* (Say), *Fieberiella florii* (Stal), and *Keonolla confluens* (Uhl). Several other leafhoppers also may serve as vectors of XDV: *Osbornellus borealis* Del. & M. (332), *Collandonus montanus* (Van D.) (749), *C. clitellarius* (Say) (260, 662), *Gyponana lamina* DeL., *Paraphlepsius irroratus* (Say), and *Norvellina seminuda* (Say) (260).

The most important vector in the West appears to be *C. geminatus*, although Jensen (332) considered *F. florii* to be an important vector in California. In the East, *S. acutus* is probably the most important economic vector, but other species occasionally may be of local importance (260).

In the northeastern United States, serious outbreaks of X-disease are cyclic in occurrence at intervals of about 8 to 10 years. Serious damage occurred during 1942-45 and 1954-1957. A new cycle began in 1968. Although the ecology of the insect vectors has not been worked out thoroughly, these cycles appear to coincide with heavy vector populations.

XDV is not mechanically transmissible so relatively little is known of the characteristics of the virus itself. XDV is heat labile in woody plants (*Prunus*); Hildebrand (308) reported that XDV was inactivated in infected peach buds by immersing them in water at 50° C. for 6 to 7 minutes. Stoddard (629) reported that a range of hot water treatments from 48° for 16 minutes to 53° for 2 minutes inactivated XDV in peach buds. Nichols and Nyland (485) found that the yellow leaf roll strain of XDV was considerably more tolerant of heat than the common strain; in their best treatment the YLR strain was inactivated in only 50 percent of the buds. R. M. Gilmer (unpublished data) found that XDV was inactivated in experimentally infected sweet cherry budlings exposed at 37° for 15 days or more, but many of the trees died as a result of treatment.

The incubation period of X-disease in peach or chokecherry depends largely

upon the stage of growth at which inoculation occurs. Bud-inoculations made in late July or early August result in the appearance of symptoms about 6 weeks after growth begins the following spring. If trees are inoculated just as growth begins, symptoms often develop within 6 to 8 weeks.

The latent (incubation) period of XDV in leafhopper vectors is rather lengthy: 22 to 35 days in *Colladonus geminatus* (751), 45 days in *Scophytopius acutus* (751), and 20 to 40 days in *Fieberiella florii* (260). Transmission by infective leafhoppers is often erratic, i.e., many susceptibles on which infective leafhoppers feed may escape infection (260). Peach is apparently a rather poor host for XDV acquisition by leafhoppers (260, 333), perhaps because the virus is often irregularly distributed in this host. Although periwinkle is easily infected by leafhoppers, it, likewise, is a poor host for XDV acquisition by vectors (260). Celery (333), chokecherry (260), and sour cherry (260) are good virus sources for leafhopper acquisition.

Control Measures

In the East, outbreaks of X-disease in peach or cherry orchards are invariably associated with the presence of diseased chokecherries nearby. Eradication of chokecherries within 500 feet of orchards generally provided acceptable commercial control of the disease in New York (504). Although there is no indication of significant spread of XDV within the orchard from infected orchard trees, the latter are commercially worthless and should be removed. Chokecherry is readily reseeded by birds, so continued vigilance in its removal is required. Distance does not appear to be an effective barrier in the spread of XDV from chokecherry to chokecherry where the rate of spread is much more rapid than in peach or cherry.

In the West, XDV spreads from peach to peach or cherry to cherry, and the presence of infected chokecherry as a reservoir host is not necessary. Chokecherry should be eradicated, and infected orchard trees should be removed as soon as they are identified. In Washington, a survey and eradication program (1947-51) was effective in reducing the threat of X-disease.

In areas where the disease is important and spread is rapid, topworking cherry on a mahaleb framework is recommended (578). Individual cherry limbs may become infected, but the spread of XDV into the remaining portions of the tree is limited by the mahaleb frame. Cherry trees on mahaleb rootstocks appear less likely to be infected than those on mazzard rootstocks. Several sweet cherry cultivars are highly resistant to infection even when propagated on mazzard rootstocks. Among these are Napa Long Stem Bing, Dicki Braune, Blakenburger, and Coop's Special (549 and B. N. Wadley, unpublished data).

Although X-diseased peach or cherry nursery trees grow poorly with obvious symptoms, XDV can occasionally be carried for a short time in a latent form in nursery trees. Scionwood for propagation should be selected from healthy trees, and chokecherry near nursery planting should be completely eradicated. Since seeds from infected trees abort or fail to germinate, there is little danger of XDV transmission in seedling rootstocks.

Remarks

Most stone fruit virologists now consider that the eastern and western forms of X-disease are caused by very similar if not identical viruses. Both forms attack the same economic hosts—peach, sweet cherry, and sour cherry (505, 550, 563, 630). The symptoms of buckskin disease on sweet cherry on mahaleb rootstock in California were very similar to the cherry wilt and decline syndrome induced by western X-disease in Utah (563). Zeller and Milbrath (771) demonstrated that buckskin of sweet cherry and X-disease of peach were induced by the same strain of XDV, and the similarity of cherry buckskin and leaf casting yellows of peach in California to both eastern and western X-disease has been described (549, 657). Considerable strain variation of XDV also occurs in the East (259, 260, 308).

The leafhopper vectors of XDV are members of a single subfamily, Deltocephalinae, with the exception of *Gyponana lamina*, which belongs to subfamily Iassinae. There is a single report of transmission by *Keonolla confluens* (subfamily Tettigellinae) (17), but confirmation would be highly desirable because numerous other members of this subfamily were unable to transmit XDV (260).

Gilmer et al. (260) pointed out that many of the leafhoppers that transmit XDV are also vectors of western aster yellows virus. Although the symptoms of X-disease in herbaceous plants are distinct from those of aster yellows or western aster yellows, they are basically similar. These facts suggest that XDV and aster yellows virus may be related. Yellows-type diseases are associated with mycoplasmas rather than viruses (530), and X-disease may also be induced by a mycoplasma. This hypothesis is supported by recent investigations (George Nyland and T. S. Pine, unpublished data) in which remission of foliage symptoms occurred in peach trees infected with the yellow leaf roll strain of XDV when these were treated with terramycin by trunk injection, foliar sprays, or soil drenches. Individual branches of infected mature peach trees injected with terramycin during dormancy produced normal-appearing fruits the year following treatment, while nontreated branches of the same trees produced no fruits.

Stoddard (629) reported that X-disease could be controlled in infected peach trees by injection or soil drenches with a number of chemicals, including P-aminosulfonamide, P-toluenesulfonamide, maltose, hydroquinone, calcium chloride, and zinc sulfate. A repetition of some of his experiments showed the preinoculation treatments of calcium chloride, zinc sulfate, and hydroquinone were effective, perhaps because these materials caused sufficient injury to prevent union of inoculum (R. M. Gilmer, unpublished data). If inoculum had united, postinoculation treatments with these chemicals were not effective in preventing the disease.

Note: Several reports have appeared (Virology 41: 583, Phytopathology 60: 1534, and 61: 1036) that demonstrate the association of mycoplasmas with X-disease.

YELLOW BUD MOSAIC

ARCHIE SCHLOCKER and JACK A. TRAYLOR

Causal Virus

Yellow bud mosaic (YBMV) strain of tomato ring spot virus (TomRSV).

Synonyms

Winters' disease; Winters' peach mosaic; Eola rasp leaf.

History and Distribution

Yellow bud mosaic was found affecting peach and almond trees in the Winters area of Solano and Yolo Counties, Calif., in 1936 and was described for the first time in 1939 (653). Inspections showed its presence in at least 40 orchards in the two-county area. Between 1950 and 1964, the disease was found in peach in all the peach-growing areas of northern and southern California and in cherry in the cherry-growing areas of San Joaquin and Stanislaus Counties. The disease has not been found in peach or cherry in the southern portion of the San Joaquin Valley. The disease has not been reported in peach outside of California. The disease in cherry in California is the same as that described in Oregon as Eola rasp leaf. All evidence indicates that the disease spreads from native wild plants to fruit trees grown in infested soil (498).

Economic Hosts

Yellow bud mosaic is a serious disease of peach, nectarine, sweet cherry, almond (principally the Texas cultivar), and Wickson plum (636, 653, 698).

Other Hosts

Apricot is a natural host but is not seriously damaged. Infected trees may be symptomless carriers of YBMV and are capable of supplying the virus to the nematode vector (*Xiphinema americanum*), which can carry it to more susceptible hosts (658). Some brambles and a number of weeds have been reported as natural hosts of YBMV in California, including Himalaya blackberry (*Rubus procerus*), red raspberry (*R. idaeus*), red currant (*Ribes sativum*), sand strawberry (*Fragaria chiloensis*), cheeseweed (*Malva parviflora*), seaside daisy (*Erigeron glaucus*), Fuller's teasel (*Dipsacus fullorum*), bristly ox tongue (*Picris echioides*), plantain (*Plantago* spp.) and hedge nettle (*Stachys bullata*) (13, 209, 341 and H. E. Williams and Q. L. Holdeman, unpublished data).

Thomas and Rawlins reported transmission of YBMV to Portugese laurel, Japanese apricot, rose (*Rosa* sp. cv. 'Ragged Robin'), Japanese kerria (*Kerria japonica*), desert peach and myrobalan plum—the last by passage through desert almond (653, 654). YBMV also has been graft-transmitted to Manchu cherry (599).

The virus was transmitted to cowpea, tobacco, bean, and cucumber in expressed juice in 1954 (755, 760). Karle (341) extended the herbaceous host range to include some members of each of the following plant families:

Amaranthaceae, Caryophyllaceae, Chenopodiaceae, Compositae, Cucurbitaceae, Geraniaceae, Leguminosae, Malvaceae, Solanaceae, and Rosaceae. He reported that the best herbaceous indicators for YBMV were Bountiful bean (*Phaseolus vulgaris*), Virginia blackeye cowpea (*Vigna sinensis*), tobacco (*Nicotiana rustica* and *N. tabacum*), and National Pickling cucumber (*Cucumis sativum*).

Symptoms

Peach and nectarine.—Symptoms are most clearly expressed in the spring as soon as the earliest leaves have expanded. Growth from some of the leaf buds is either arrested or severely retarded, producing very short tufts of tiny, pale-yellow leaves. The tufts often turn brown and die before attaining a length of more than 2 to 5 mm., but some may grow slowly to a length of 5 to 10 mm. and appear to remain in an arrested state of development. This severely retarded growth stands out in sharp contrast to normal-appearing leaves on the same branch or in other parts of the tree. However, as the season progresses, many of these retarded, partially developed yellow leaves expand into small rosettes of dwarfed, distorted, yellow-green leaves (figs. 72, 73).

Many tufts die with the onset of hot weather, leaving many bare shoots on the affected limbs. The reduced current-season growth and relatively sparse foliage give the diseased tree an open or thin appearance so that it is possible to see through the lower half of the tree.

The first symptom to appear in the leaves of a newly infected tree are chlorotic blotches or spots in the leaf blades—the “mosaic” phase. These chlorotic blotches and spots are produced on shoots into which YBMV has recently moved. They are irregular in outline, feather-edged, and almost without exception follow along the main veins. The blotches commonly occur toward the base of the leaf and predominantly on one side of the midrib. When the blotches involve smaller veins, they appear to leak out into the green portions of the leaf. They are accompanied by a leaf blade distortion which often takes the form of a pinching, puckering, and corkscrewing, and lateral bending of the leaf blade toward the chlorotic areas. Sometimes the yellow spots consist of small chlorotic flecks approximately 1 mm. in diameter. The chlorotic areas frequently die and drop out, resulting in holes and tattering (fig. 74).

In the second year of infection, yellowish, severely retarded growth from the buds—the “yellow bud” phase—predominates, and the twigs bearing these buds usually die by mid-July. Both types of symptoms may occur on the same twig, but often only yellow buds will be present in a tree. The “mosaic” phase may be observed in leaves in various stages of development. In very mild cases, and on trees which are in the early stages of infection, the only symptoms to be found may be a few mottled leaves which may or may not be distorted. In such cases, painstaking and thorough examination of the tree is necessary to detect these symptoms.

The virus moves slowly upward in infected trees so that, after several years, fruit is produced only on shoots at the branch extremities. The limbs, and

larger branches become sunburned because of sparse foliage and often succumb to wood-boring insects. Infected trees progressively decline into worthless snags (fig. 75).

Symptoms of yellow bud mosaic in infected trees may vary depending on the age of the trees at the time of infection and how long the trees have been



FIGURE 72.—Peach shoot affected by yellow bud mosaic showing retarded buds and two almost normal spurs on *right*; healthy shoot on *left*.

infected. Young, thrifty trees, 3 to 8 years old, that become infected are the best indicators for the presence of YBMV in the soil. They usually show symptoms better than trees that are inoculated at a later age. Any particular infected tree may express strong and abundant diagnostic symptoms one year and mild and sparse symptoms the next year. Symptoms apparently are influenced by environmental conditions, particularly moisture and temperature.

Spread of YBMV within an orchard occurs slowly and mostly to trees adjacent to infected ones, gradually enlarging the original area of infection.



FIGURE 73.—Peach shoots affected by yellow bud mosaic, some dying and others developing rosettes of small leaves.



FIGURE 74.—Chlorotic spots and blotches, vein feathering, and distortion in leaves of Elberta peach affected by yellow bud mosaic.

In some orchards, spread occurs more rapidly in the direction of flowing irrigation water rather than at right angles to it. Frequently, a focus of infection in an orchard occurs at or near the outside row, and the spread to adjacent trees affects areas in a more or less semicircular shape with eventual enlargement of the area in an irregular pattern.

Replants in old infected orchards may become infected by the second or third year after planting. Sometimes, seedlings 2 years of age or older, originating from peach pits under bearing trees, escape destruction from cultivation tools and become infected from soil and develop typical symptoms of yellow bud mosaic. Sometimes symptoms can be found only on these seedlings and not in the old trees above them, although the latter are known to be infected with the virus.

S. H. Smith and J. A. Traylor (unpublished data) have observed stem pitting and bark thickening in yellow bud mosaic infected trees examined in the Sacramento and San Joaquin Valleys. This was the first report associating these symptoms with this disease in peach.

No flower symptoms are known. Flower buds commonly drop from severely affected branches and few, if any, fruit are set. Consequently, fruit production is seriously reduced in severely affected trees.

Almond—Symptoms of yellow bud mosaic in almond consist mainly of sparse foliage, tufting, and lack of terminal growth. Some affected trees show considerable killing of spurs and of the lateral buds and shoots along the branches, giving the trees an open or thin appearance (fig. 76.) Texas almond is most severely affected by YBMV, often showing symptoms extending into the upper-

most portions of the tree. In this cultivar, one or more normal shoots are frequently produced on otherwise completely diseased branches. Sprouts from the trunks of infected trees usually show rosette-type growth. Unusually wrinkled, rough fruits with hulls abnormally thickened have been observed on diseased branches of Texas almond (fig. 77). Reduction in yield is directly proportional to severity of symptoms (342). In trees of Nonpareil and Ne Plus Ultra, yellow bud mosaic symptoms seldom show above the main trunks or bases of the scaffold limbs. Occasionally, leaf symptoms resembling those in peach have been seen on almond leaves.

Sweet cherry.—Affected trees are characterized by a bare-limb appearance which starts in the lower portion of the tree and advances slowly upward. The disease kills spurs, twigs, and small branches as it moves. Leaves on affected spurs are undersized with prominent whitish secondary veins that branch off from the midrib almost at right angles, resembling those of elm leaves. Initial shock symptoms from YBMV inoculation may appear in some cherry leaves in the form of chlorotic blotches associated with the veins, resembling the leaf blotches observed in peach leaves infected with the virus. Enations develop on the underside of affected leaves, most commonly adjacent to the midrib. They are smaller, fewer, and accompanied by less leaf distortion than the enations caused by cherry rasp leaf virus.

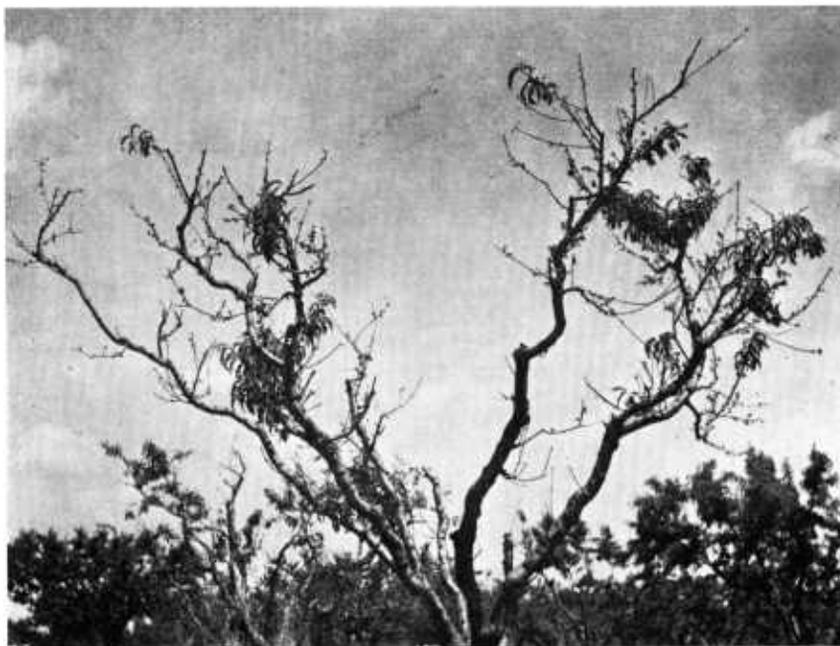


FIGURE 75.—Lovell peach tree in advanced stage of yellow bud mosaic.

Transmission

Xiphinema americanum Cobb, the dagger nematode, is the natural vector of YBMV (645, 646). The virus can be transmitted through grafts and mechanically in expressed sap (340, 341, 755, 757, 760). Peach, almond, mahaleb, and



FIGURE 76.—Yellow bud mosaic in Texas almond: *A*, healthy branch; *B*, affected branch with dead and sparsely foliated spurs.

mazzard rootstocks become infected when grown in infested soil. In limited trials in the orchard, Myrobalan 29C, myrobalan seedlings, and Marianna 2624 did not become infected for 3 years when planted in infested soil (George Nyland, personal communication). Teliz et al. (646) succeeded in transmitting YBMV to the roots of Royal apricot and damson plum seedlings by *X. americanum*.





FIGURE 77.—Yellow bud mosaic in Texas almond: healthy fruit on *left*; diseased fruit on *right*. Note roughness and larger size of diseased fruit due to thicker hulls.

Characteristics of the Virus

The YBMV particle is spherical with a diameter close to $25 \text{ m}\mu$ (107, 108, 711). Cadman and Lister (107) reported that the virus lost its infectivity when infective plant sap, diluted one-fifth with distilled water, was heated for 10 minutes at 60° C . but not at 58° , or when diluted more than 10^{-4} with distilled water. Sap lost little infectivity when kept at -10° for 24 hours but lost 80 to 90 percent in 5 days at this temperature. They considered YBMV to be less stable than other viruses of the ring spot type, such as arabis mosaic and tomato black ring, because its infectivity was destroyed by most of the customary means of clarifying sap. The virus was inactivated *in vivo* in buds of infected peach trees exposed to 38° for 21 days (George Nyland and A. C. Goheen, unpublished data).

Control Measures

Strict adherence to quarantine regulations, which restrict the movement of susceptible host material and soil from infested properties and areas, and the use of propagating stock free from all known viruses are the primary preventative measures for controlling yellow bud mosaic.

To suppress or eradicate the disease in infested orchards, the following steps are recommended: (1) If the affected area involves only a small part of the orchard, the infected trees and adjacent trees in at least two rows beyond should be removed. If several areas of diseased trees are present in a contiguous

planting, complete removal of the block may be advisable. Trees to be removed should be pretreated with a herbicide applied into cuts made with an ax into the bark and well into the wood around the tree trunks near the ground to aid in killing the roots. As many roots as possible should be removed with the trees. (2) The infested soil should be fallowed for at least two growing seasons, preferably three, after tree removal. The fallow period must be long enough for all tree roots to die, plus an additional year for the nematodes feeding on the roots to die. The land should be kept free of weeds during the entire fallow period because many weeds are hosts for both the virus and the nematode vector. (3) Fumigate soil with a nematocide. Specific dosages will depend on soil type and material. (4) Replant with nonhosts where fumigation is not to be done. These include most plums and prunes on plum rootstocks, and probably walnuts, although specific tests have not been made.

Remarks

Gooding et al. (267) showed that the grape yellow vein disease found in California vineyards was caused by a soil-borne virus serologically related but distinct from TomRSV. Teliz et al. (645) demonstrated that *Xiphinema americanum* was the natural vector of grape yellow vein virus. Cadman and Lister (107, 108) compared isolates of YBMV, TomRSV, and tobacco ring spot virus (TRSV). Based on plant protection and serological tests, they concluded that YBMV and TomRSV were closely related and should be regarded as synonymous. They found no relationship between YBMV-TomRSV and TRSV.

Eola rasp leaf of cherry, a soil-borne virus disease described from the Willamette Valley, Oreg., is caused by TomRSV (457), with symptoms very similar to those found in YBMV-infected cherry trees in the San Joaquin Valley of California (George Nyland, personal communication). A Lovell peach seedling bud-inoculated with Eola rasp leaf in Oregon developed typical yellow buds in the second season after inoculation (457).

VIRUS DISEASES OF PLUM AND PRUNE

PLUM LINE PATTERN

H. C. KIRKPATRICK and R. W. FULTON

Causal Virus

Plum line pattern virus (PLV).

Synonyms

Shiro line pattern, plum mosaic, peach line pattern, and banded chlorosis of oriental flowering cherry.

History and Distribution

Line pattern has been reported on plum, plum nursery stock, peach and oriental flowering cherry in Canada, and throughout the United States (35, 120, 130, 679, 729); on plum, peach, and apricot in New Zealand (131); and on plum in Great Britain (541), Italy (116), India (19), and Germany (28). Line pattern probably is widely distributed in most of the plum-growing areas of the world.

Economic Hosts

Line pattern occurs in nature frequently on Red June and Abundance Japanese plum; on Amanogawa, Shirotae (Mount Fuji), Naden, Kwanzan, and Temari oriental flowering cherry; and, to a lesser extent, on peach seedlings (130). It occurs on myrobalan plum rootstocks in nurseries, on a number of European plum and greengage cultivars, and on damson plum and mahaleb cherry in New Zealand (131).

Other Hosts

Prunus spp. and cultivars that have been experimentally infected include: Elberta, J. H. Hale, Rochester, and Carman peach; David peach; Becky Smith Japanese plum; Shiro, First, Ember, LaCrescent, Climax, Duarte, and Marianna 2624 hybrid plum; Italian Prune, Early Golden Drop, German, Imperial Gage, Reine Claude, Lombard, Grand Duke, and Richards Italian European plum; Klamath plum; sweet cherry seedlings and cultivars Black Tartarian, Napoleon, Gil Peck, and Seneca; Montmorency sour cherry; Amur cherry; pin cherry; Bessey cherry; Yoshino flowering cherry; common chokecherry; desert peach; sloe plum; flowering almond; and Drake, Nonpareil, and Mission almond (28, 130, 211, and P. W. Cheney and E. L. Reeves, unpublished data). Niagara apricot appeared to be a symptomless host. The virus has been transmitted mechanically to 86 herbaceous species representing eight families (372, 512).

Symptoms

Symptoms are invariably confined to leaves emerging in the spring when the daily mean temperatures are below 13° to 15° C., and they persist in these leaves

throughout the season. Therefore, in the summer, most of the leaves on infected trees are symptomless.

Plum.—Commercial plum cultivars are variable in disease expression. Symptoms in leaves consist of lines, bands, and oak-leaf patterns. Vein-banding and small chlorotic rings also may be present. In Shiro plum (fig. 78, *A*) and myrobalan plum (fig. 79, *D*), the patterns are conspicuously yellow or yellow-green in the spring and become a vivid creamy white early in the summer. In Abundance plum, the patterns resemble those in Shiro but are less conspicuous and more transient. The symptoms consist of chlorotic rings, spots, and line patterns bordered with a white to bright golden band. In Italian Prune, Reine Claude, and First plums, very faint oak-leaf patterns may appear although in Italian Prune they may be absent in some seasons. In German and Grand Duke fine, irregular yellowish lines predominate.

Willison and Berkeley (130) found that Imperial Gage and Early Golden Drop plum and some myrobalan plum seedlings carried the virus without developing symptoms. Other myrobalan seedlings showed a golden net pattern bordering the veins and veinlets (768). In Lombard plum only a few leaves are affected. In these, faint markings of the Shiro type may be noted, but the more characteristic symptom is a regular, chlorotic, and eventually necrotic, line outlining the marginal triangular section of the lamina and usually distorting the leaf.

Peach.—No differential reaction of peach cultivars to infection by PLV was detected by Willison (130). The characteristic symptom is a fine, irregular, wavy band on each half of the lamina, usually forming a symmetrical pattern (fig. 78, *B*). Some leaves are marked with a network of fine lines, or a golden net pattern, fine confluent rings, vein-banding, or an oak leaf pattern, or both of the last two. Markings on peach leaves are generally pale green or more rarely yellow-green, and usually disappear during the summer. The number of affected leaves and the predominant type of symptom varies from year to year in response to the temperature conditions in the spring (130).

Sour cherry.—In Montmorency sour cherry, the symptoms in the early stages of infection are inconspicuous pale-green bands, spots, and coarse rings; a few necrotic spots also may be present. In the following year, sharply defined lines do not appear until early June (fig. 79, *B*). These lines are sometimes chlorotic, but more commonly they appear as watermarks seen best by transmitted light (130). Symptoms observed by P. W. Cheney and E. L. Reeves (unpublished data) in Washington were vein-banding, mosaic mottle (light-green mottle developing to a yellow or orange mottle with large necrotic areas resembling those of rusty mottle), and some leaf cast.

Mahaleb cherry.—Symptoms in mahaleb consist of oak-leaf patterns, coarse lines, and rings sometimes darker and sometimes paler than the rest of the leaf (fig. 79, *C*). Short, narrow, yellow bands along the larger veins are of diagnostic value. In these areas the veins, and frequently part of the yellow-band

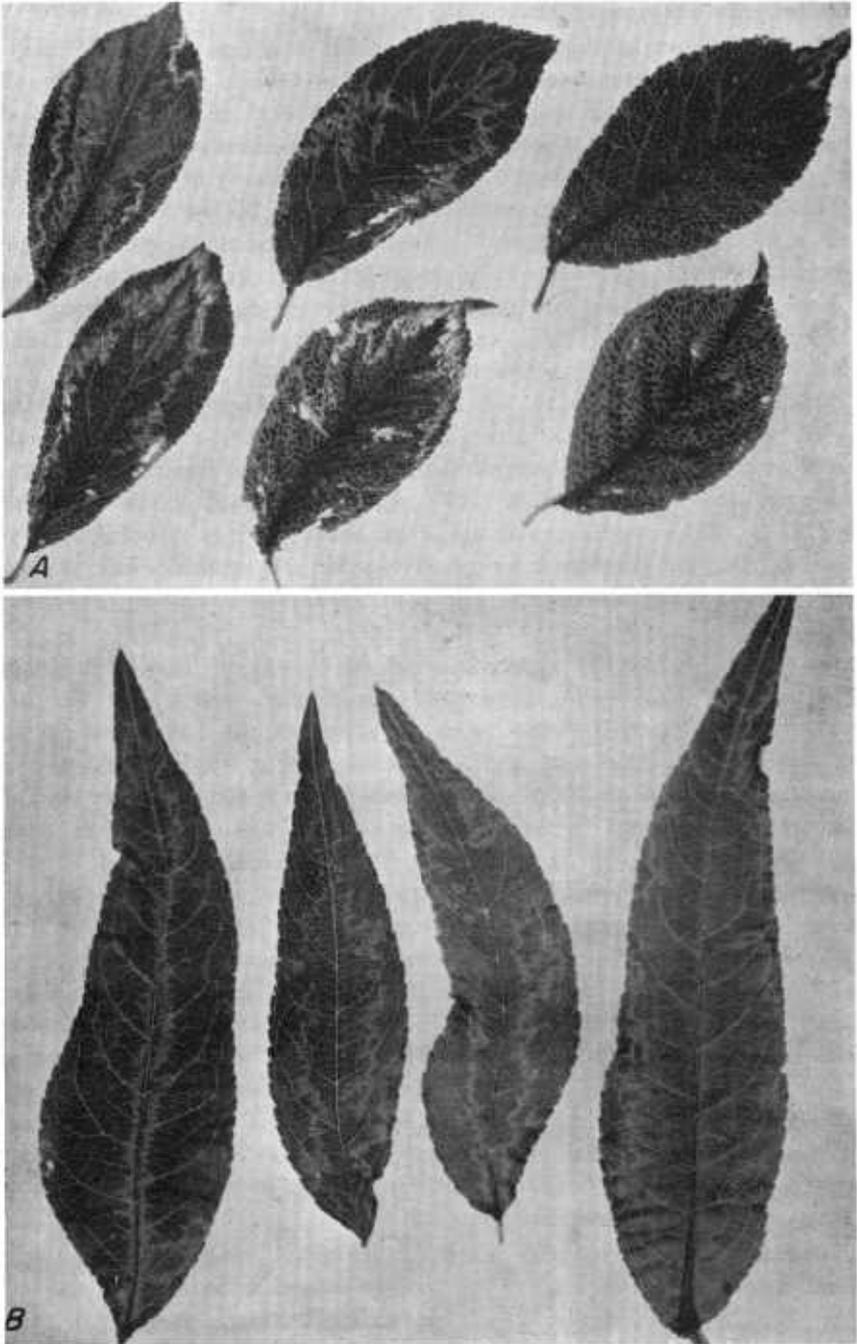


FIGURE 78.—Plum line pattern. *A*, Chlorotic lines and patterns and vein netting on Shiro plum leaves; *B*, chlorotic lines and patterns on peach leaves.

tissue, eventually become necrotic and either split or drop out. The severity of symptoms varies with individual trees. One strain of PLV from Michigan produces a golden blotch mottle with some ring spots, whereas a strain from Oregon, from Amanogawa flowering cherry, produces a banded oak-leaf pat-

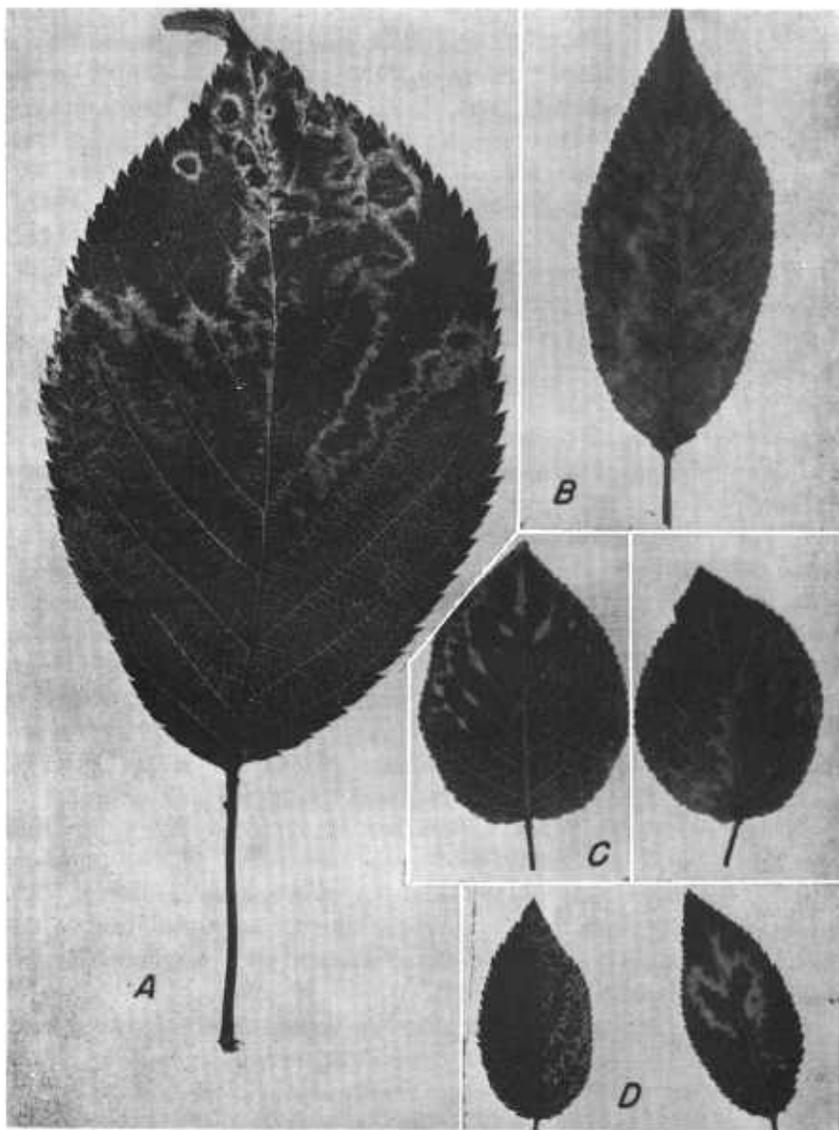


FIGURE 79.—Plum line pattern. Chlorotic rings, patterns, or vein netting in *A*, Amanogawa oriental flowering cherry; *B*, Montmorency cherry; *C*, mahaleb cherry; and *D*, myrobalan plum.

tern in some leaves and banded circular spots or irregular line patterns in others. The majority of leaves show no symptoms (130).

Sweet cherry.—Symptoms in Napoleon and Black Tartarian sweet cherry are divided into two groups. The first is made up of tiny rings, larger coarser rings, and faint oak-leaf patterns which are more or less transient. The second group consists of persistent watermark lines and more conspicuous yellow or creamy white lines, up to 1 mm. in width, which form striking irregular patterns (130). Black Tartarian is considered resistant to PLV in Washington (P. W. Cheney and E. L. Reeves, unpublished data). In the young foliage of Bing and Black Republican, PLV causes faint line pattern symptoms, which later become pronounced. Chlorotic mottle develops as the trees become older. Zeller and Milbrath (768) found that mazzard seedlings vary greatly in disease expression. Some seedlings may show a well-formed, banded oak-leaf pattern while others show an irregular chlorotic or red mottle. In some seedlings, affected tissue becomes necrotic and falls out (130).

Oriental flowering cherry.—Symptoms are similar in all affected cultivars of Oriental flowering cherry. Leaf borders are faintly chlorotic to pronounced golden or white (fig. 79, A). The patterns are sometimes made up of large rings but more often are an oak-leaf type (130).

Almond.—Drake, Mission, and Nonpareil all have line pattern symptoms similar to those in peach. A fine light-green, vein-banding network is a prominent symptom, with some irregular oak-leaf or chlorotic areas in a few leaves.

Herbaceous hosts.—Plum line pattern virus, originally from Myrobalan 29C, was mechanically transmitted from Shiro plum to Chicago Pickling cucumber and other herbaceous plants (372). The herbaceous host range for PLV was extended by using another source of the virus, which was separated from other *Prunus* viruses by passage through Abundance plum (474, 512). Some differences were noted in the herbaceous host ranges of these two isolates. However, evidence based on serology indicates the strains react to the same antiserum (A. Q. Paulsen and R. W. Fulton, unpublished data). The originally described PLV culture, while not a lineal propagation, was related in host range and symptom data with the PLV culture used by Paulsen and Fulton and was closely related serologically. The symptoms on four herbaceous hosts, which can be used for an index or for greenhouse culture of PLV, are presented rather than the entire herbaceous host range.

Physalis floridana. Symptoms of plum line pattern develop in new growth following juice inoculation. A light-green or chlorotic interveinal blotching is noticed first, followed by leaves which develop chlorotic vein-banding or ring spots. Some leaves show a chlorotic or light-green, oak-leaf pattern (fig. 80, A). No other stone fruit virus has been transmitted to *P. floridana*, and this host is considered part of a selective host range to isolate PLV from cultures of mixed content (374).

Nicotiana megalosiphon. Chlorotic or necrotic local lesions and ring spots occur as primary symptoms in inoculated leaves. Systemic chlorotic or light-green mottle with necrosis develops in the new growth (fig. 80, B). This host is commonly used for propagating PLV (512).

Vigna cylindrica. Inoculated leaves develop necrotic local lesions or rings followed by a systemic mottle and necrosis of new growth (fig. 80, C). The primary symptom is effective for local lesion assay studies (512).

Cucumis sativus. Chicago Pickling cucumber cotyledons, inoculated as the first primary leaf is expanding, will develop some chlorotic lesions. When the cotyledons are cleared of chlorophyll and stained in iodine-potassium iodide solution, starch rings or broken rings are revealed (fig. 80, D). PLV does not become systemic in cucumber (372).

Transmission

The virus is easily transmitted in tree hosts by budding and bark-patch grafting. No evidence of an insect vector has yet been found.

Mechanical transmission from young Shiro or mahaleb leaves with line pattern symptoms to herbaceous hosts has been accomplished by homogenizing infected tissue at 1:10 (w/v) dilution in 0.001 M neutral phosphate buffer containing 3 percent nicotine sulfate (40 percent solution), the slurry centrifuged at 6,000 g. for 15 minutes to remove cell debris, and the supernatant centrifuged at 74,000 g. for 1 hour to sediment the virus from the nicotine sulfate solution (372). Alternatively, inoculum may be prepared in 0.03 M phosphate buffer, pH 8.0, containing 2-mercaptoethanol, 0.01 to 0.02 M. Sodium diethyldithiocarbamate, 0.01 M, is added when inoculum is prepared from rosaceous species (A. Q. Paulsen and R. W. Fulton, unpublished data).

Mechanical transmission from PLV-infected *P. floridana* to mahaleb cherry seedlings was positive when the inoculum had been partially purified and concentrated by homogenizing at 1:10 (w/v) in 0.001 M neutral phosphate buffer including 0.001 M cysteine HCl, centrifuging at 6,000 g. for 15 minutes, and centrifuging the supernatant at 85,000 g. for 1 hour. The pellet was suspended in the buffer at a concentration of 1:2 (original w/v). The inoculum was applied with an airbrush to the leaves of vigorously growing mahaleb seedlings in the 8- to 12-leaf stage. Symptoms of plum line pattern in the new leaves developed within 16 to 20 days after inoculation (372).

Cuscuta campestris and *C. subinclusa* failed to transmit PLV from infected to healthy *Nicotiana megalosiphon*. *C. campestris* did transmit PLV from *N. megalosiphon* to petunia but *C. subinclusa* did not. Attempts to transmit PLV from petunia to mahaleb or from mahaleb to Italian Prune through *C. campestris* were negative. PLV could not be recovered in mechanical inoculations from dodder tissue (R. W. Fulton, unpublished data).

Attempts to transmit PLV with apterous adults of *Myzus persicae* from infected petunia or *Vigna cylindrica* were negative (512).

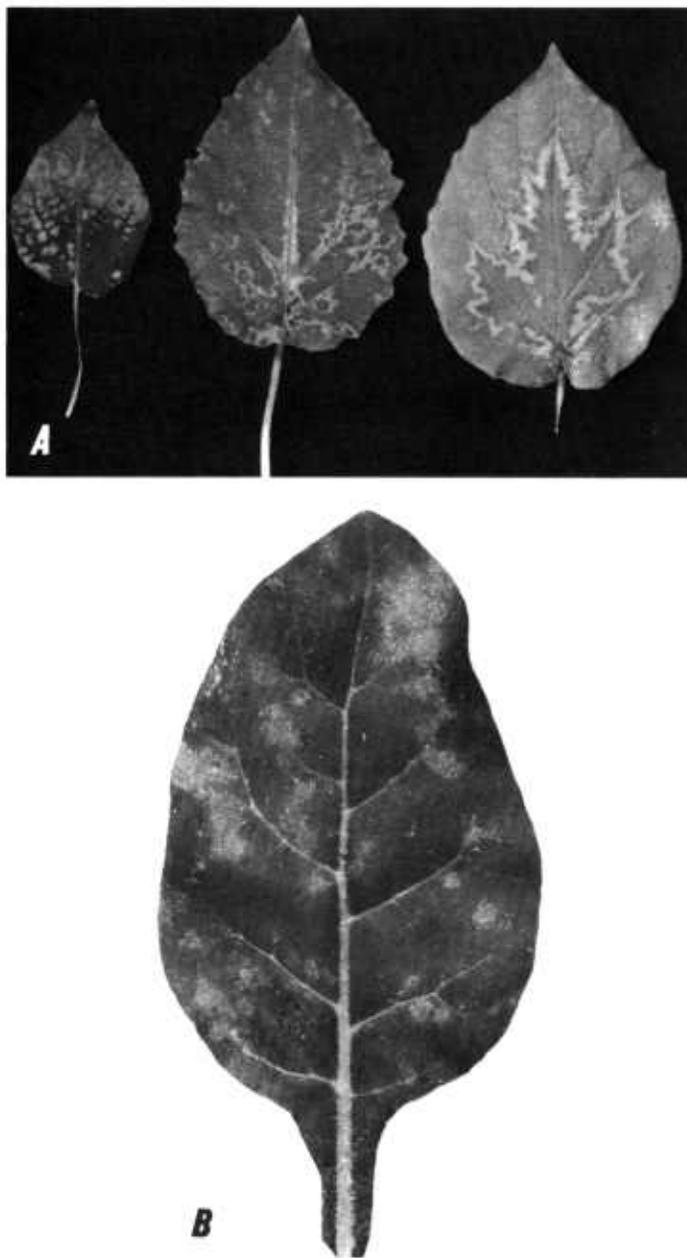
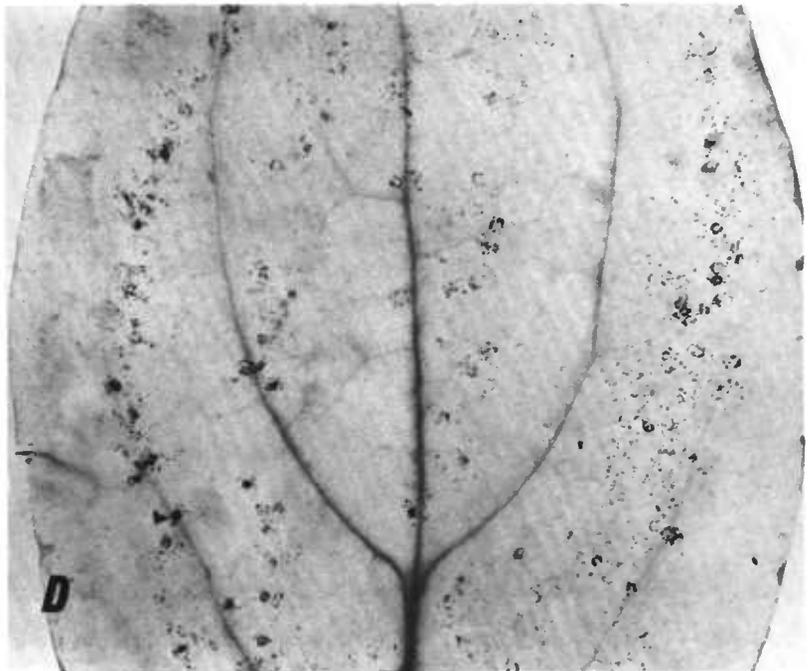
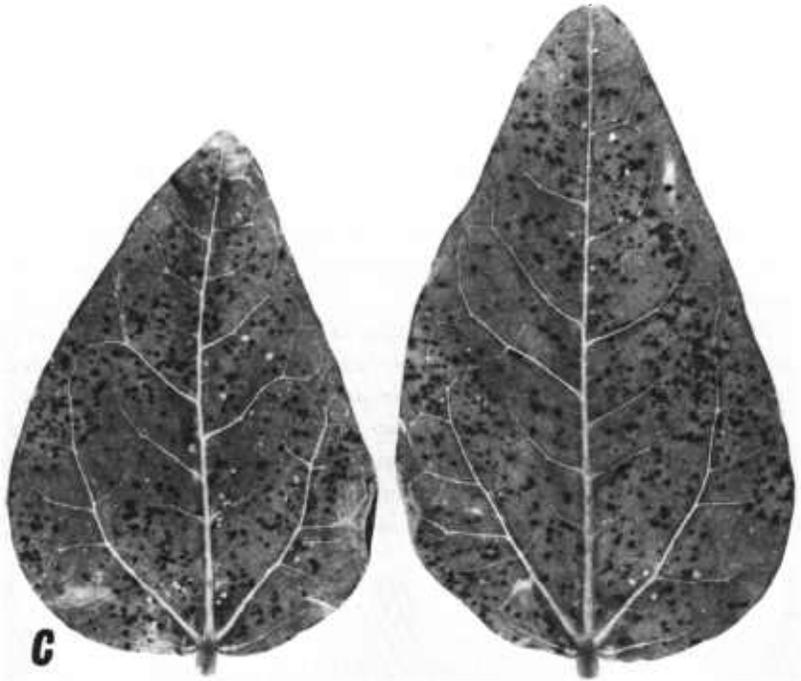


FIGURE 80.—Plum line pattern. Herbaceous hosts infected with plum line pattern virus (PLV): Leaves of *A*, *Physalis floridana* showing chlorotic spots, rings and patterns; *B*, *Nicotiana megalosiphon* showing chlorotic mottle and necrosis; *C*, *Vigna cylindrica* showing necrotic local lesions; and *D*, Chicago Pickling cucumber cotyledon, cleared of chlorophyll and stained with iodine-potassium iodide solution to show starch lesions.



Characteristics of the Virus

A. Q. Paulsen and R. W. Fulton (unpublished data) have purified PLV with two cycles of high- and low-speed centrifugation. Inoculated leaves of *Nicotiana megalosiphon* were homogenized in 0.02 M phosphate buffer, pH 8.0 (1.5 ml./g. leaf), containing 0.02 M 2-mercaptoethanol. Clarification of the first supernatant was made by adjusting the pH to 4.8 with 0.1 M citric acid. The green coagulum was removed by centrifugation and the extract adjusted to pH 6.5. After concentrating the virus by centrifugation, the remaining host protein was precipitated with the gamma-globulin fraction of antiserum to *N. megalosiphon* protein.

Purified PLV had an A_{280}/A_{280} ratio of ca. 1.7 and formed two zones in density gradients at pH 6.0-7.0. The virus was about 30 $m\mu$ in diameter in negatively stained preparations, and the particles are easily disrupted (fig. 81).

PLV is quite unstable in crude sap but infectivity can be maintained if infected tissue is ground in buffer with 0.02 M 2-mercaptoethanol. Under optimum conditions, sap of some infected species is still infectious when diluted 1:6400. Infectivity is greater in diluted than in undiluted sap of most

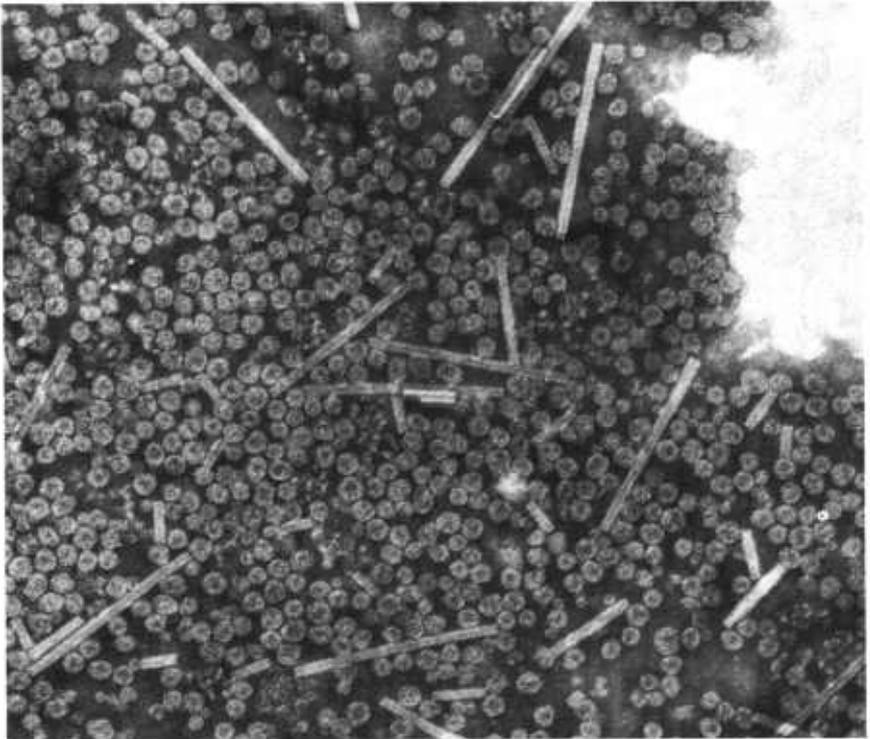


FIGURE 81.—Negatively stained plum line pattern virus, $\times 94,000$ (tobacco mosaic virus was added as a standard of size).

species. The thermal inactivation point of PLV in diluted sap is 66° C. for a 10-minute exposure.

Control Measures

It is doubtful whether roguing diseased trees should be recommended (130). Use of disease-free stock and scion material for nursery propagation is suggested.

Remarks

The combination of PLV with prune dwarf virus increased the virulence of prune dwarf in some hosts and of line pattern in others (130).

The similarity of symptoms in peach and plum caused by PLV and apple mosaic virus (AMV) has been a confusing issue. AMV was transmitted from apple to peach and from peach to apple (370). The symptoms in peach were similar to those induced by PLV. Apple mosaic symptoms were induced in apples by PLV, and plum line pattern symptoms were induced in Shiro plum by AMV (243). Two isolates from plum showing line pattern, and AMV isolates from apple, produced similar symptoms in peach and apple (541). A. F. Posnette (unpublished data) has now shown one of the line pattern isolates in plum to be serologically identical to AMV, whereas the second isolate is serologically *Prunus* ring spot virus. A vein-banding strain of AMV causes what appears to be line pattern in plum (481). Fulton (232) presented serological evidence that a line pattern of plum (DLPV) reacted only weakly with AMV antiserum. Host range and symptom data indicate Cation's originally described PLV culture to be similar to the PLV culture purified by Paulsen and Fulton, but both differ somewhat from the PLV culture of Kirkpatrick. All three PLV cultures, but not DLPV, reacted with antiserum to PLV made after the development of methods of the purification of PLV were determined (A. Q. Paulsen and R. W. Fulton, unpublished data). This indicates strain variation of PLV. AMV cultures were not serologically related to PLV. It is clear that line pattern symptoms in plum and peach can be induced by viruses other than **PLV**.

PRUNE DIAMOND CANKER

H. KEITH WAGNON and HAROLD E. WILLIAMS

Only an occasional tree with prune diamond canker was noted prior to 1915 (619), but during the following 20 years its increased occurrence in newer plantings attracted considerable attention. Extensive investigations on the disease were started about 1927 by R. E. Smith and culminated in a report in 1941 with evidence of its virus nature (620). Although the disease has been observed during the years following this report (691, 701), there has been very little investigative work on it. The disease is known only in California and affects only the French (Agen) prune cultivar (*Prunus domestica*.) It was of particular concern in Napa and Santa Clara Counties (622) and was rare in the Sacramento and San Joaquin Valleys. When it was found to be associated with the use of infected propagating budwood, nurseries became more selective in their choice of budwood (620, 701), and as a result the incidence of the disease has decreased in newer plantings in Napa and Santa Clara Counties. The disease has not been observed during the last 25 years in the Sacramento and San Joaquin Valleys, where higher temperatures are believed to suppress the development of symptoms. Many new, large plantings of French prune are being made in California, particularly in the Sacramento Valley, with stock propagated from selected budwood sources free of Prune diamond canker virus (PDCV).

Recognition of prune diamond canker depends on the occurrence of bark cankers on the trunk and older branches of a tree. No fruit or leaf symptoms are recognized. The cankers become apparent as the bark becomes thickened and roughened in a somewhat diamond-shaped area frequently observed around such wounds as pruning cuts (fig. 82). The wood may or may not be exposed. In some infected trees, the cankers may not be distinct, but the symptoms may consist of roughened bark on older parts of the tree. Slight roughening of bark is sometimes seen on bark only a year or so old.

PDCV has been transmitted only by grafting (620). Symptoms may develop in 2 years, but generally do not do so until several years later. Young trees propagated from infected buds may not develop symptoms in the orchard until 5 to 8 years of age. When diseased prune scions are grown on apricot, almond, peach, myrobalan plum, and Marianna 2624, these understocks do not develop symptoms. In at least one of these (myrobalan plum), the disease was demonstrated to move through the rootstock from an infected scion on one side to a healthy scion on the other side of the tree.

Information on virus morphology, properties, and serological relationships is lacking. Graft-inoculation tests on Shiro-fugen flowering cherry made with buds from different infected French prune orchard trees indicate that there is no

relationship between the occurrence of PDCV and strains of *Prunus* ring spot virus complex.

Prune diamond canker is of economic importance only in French prune. Affected trees at first may show stimulated growth, but they will decline in vigor as they grow older. Secondary causes may contribute to a more rapid decline of the tree. Fruitfulness also declines, although the individual fruits may



FIGURE 82.—French (Agen) prune tree affected by prune diamond canker showing discrete cankers *above* and general rough bark *below*.

be large. The disease is best controlled by using PDCV-free planting stock. If tested budwood source clones are not available, young trees should be avoided as budwood sources, because they may be infected but have not yet developed symptoms.

PRUNE DWARF

R. M. GILMER, GEORGE NYLAND and J. DUAIN MOORE

Causal Virus

Prune dwarf virus (PDV).

Synonyms

Diseases incited by PDV in economically important stone fruits have been described under a variety of names. In plums and prunes, the disease has been termed prune dwarf, prune mosaic, willows, and shoestring (312). In peach, Muir peach dwarf (329) and peach stunt (452) are incited by PDV, and some strains incite gummosis of apricot (214 and P. R. Fridlund, unpublished data). In sweet cherry, PDV may induce symptoms indistinguishable from tatterleaf (733) and some other ring spotlike symptoms, or it may be present without symptoms (312).

In sour cherry, the names sour cherry yellows (353) yellow leaf, physiological yellow leaf (626), physiological leaf drop (264), and boarder tree (354) have been applied to the disease. The initial acute symptoms in sour cherry have been called chlorotic mottle and chlorotic ring spot (255). These symptoms were undoubtedly often described as *Prunus* ring spot, an entirely different disease, in much of the literature before 1960.

The virus itself also has several synonyms (249); sour cherry yellows virus (353), "yellows" strain of ring spot virus (447, 452), peach stunt virus (452), Muir peach dwarf virus (329), and virus B (225, 226, 227). A proposal to conserve the name sour cherry yellows virus against the earlier name prune dwarf virus (249) has not been generally accepted, although the former term is preferred by some authors (182, 184, 246, 248, 249, 709).

History and Distribution

Chronic effects of PDV infection on the tree habit and fruit production of sour cherry were perhaps observed in France in 1768 and in England in 1839 (248). The virus may have been introduced into North America early in the 19th century in the cherry cultivar Large Montmorency (248).

Yellows of sour cherry was observed in New York in 1919 by Stewart (626) and again in 1928 by Gloyer and Glasgow (264), who suggested that the symptoms resulted from winter injury.

In 1936, Thomas and Hildebrand (651) described prune dwarf in Italian Prune in New York and Ontario and demonstrated the virus etiology of the disease. Keitt and Clayton (352, 353) soon afterwards showed that sour cherry yellows was a virus disease. Concurrently, Hutchins et al. (329) demonstrated that Muir peach dwarf, observed in California as early as 1920, was also caused by a virus. As will be shown later, each of these diseases is induced by PDV.

PDV was perhaps the first of the tree fruit viruses to be transmitted

mechanically to herbaceous plants (93, 473, 680), a fact that has greatly facilitated research on it and many other viruses of tree fruits. Although PDV was demonstrated to be graft transmitted quite early (352, 651), its method of spread in the orchard was not discovered until 1963 (236, 237, 250, 261).

PDV is prevalent and widely distributed in sweet and sour cherries throughout North America and appears equally common in Europe (540, 762) and Australia (507). It undoubtedly occurs in all areas where these fruits are grown. In plums and prunes, where the virus was first discovered, prune dwarf is a rare disease, although a few infected trees have been found in New York, Ontario, and British Columbia (651).

Natural infections in peach occur in California (329) but have not been reported elsewhere.

Economic Hosts

PDV is very prevalent in sour cherry and sweet cherry, and natural infections occur in peach and occasionally in European plum. Damson plum is a symptomless carrier of PDV (728 and R. M. Gilmer, unpublished data). Mazzard and mahaleb, the common cherry rootstocks, are frequently infected without evident symptoms (123, 125, 256).

Thomas and Hilderbrand (651) transmitted PDV to Japanese plum, apricot, and myrobalan plum with few or no evident symptoms. Fridlund (214) found that some apricot cultivars developed severe gummosis when infected with PDV, but others were symptomless carriers. Moore and Keitt (In 354) considered that Burbank and Abundance plums (*Prunus salicina*) were immune, but these cultivars were reported susceptible by Gilmer (242).

All isolates of PDV tested induce a severe localized bark necrosis and gummosis in Shiro-fugen flowering cherry (*P. serrulata*); this host is widely used as an indicator of PDV and of *Prunus* ring spot virus (PRSV). Similar symptoms developed in Krassa Severa, a duke cherry, with each of eight PDV isolates but not with isolates of PRSV (275).

Other Hosts

Moore and Keitt (In 354) inoculated wild American plum, wild black cherry, common chokeberry, and pin cherry with PDV and recovered PDV from each of them. Fulton (227) has shown that PDV is readily transmitted to pin cherry mechanically. Nearly all of 122 *Prunus* spp. inoculated with virus isolates containing PRSV and PDV (as sour cherry yellows virus) showed symptoms (242). It was not clear that the symptoms on all the hosts were the result of PDV infection, but PDV was recovered from a number of species, including: *P. davidiana*, *P. fasciculata*, *P. fruticosa*, *P. glandulosa*, *P. lyoni*, *P. maackii*, *P. munsoniana*, *P. mume*, *P. pensylvanica*, *P. ssiori*, *P. tenella*, *P. tomentosa*, *P. triloba*, and *P. virginiana* (R. M. Gilmer, unpublished data).

PDV infects a number of herbaceous plant species; Fulton (225) recorded infection of 86 of 227 species by virus B (PDV), including a number of species not infected by PRSV. *Momordica balsamina* and *Cyamopsis tetragonaloba* were

differential hosts for PDV (706). Several squash cultivars—Butternut (246), Buttercup (447), and Cocozelle (244)—are good diagnostic hosts of PDV (fig. 83). *Tithonia speciosa*, a composite, is commonly used as a diagnostic indicator for PDV (246, 589), but is not infected by some isolates (706).

Symptoms

As Keitt et al. (354) suggest, many of the early descriptions of the symptoms of PDV infections were complicated by the presence of such other contaminating viruses as PRSV or green ring mottle virus (GRMV). For the most part, the syndromes described below in sour and sweet cherry are based on inoculations with PDV isolates purified by single lesion selection in cucurbits or inoculations by graft transmission from trees in which PRSV and GRMV were absent.

Sour cherry.—Davidson and George (182) have demonstrated that the severity of acute or "shock" symptoms in Montmorency is governed by the stage of tree growth at which the inoculations are made. Inoculations in the early stages of growth result in severe symptoms, and those made late in the season induce severe symptoms the following year, but those made in midseason just after terminal growth has ceased, may result in the introduction of the virus without symptoms.

In newly infected Montmorency trees, chlorotic mottle or chlorotic rings and flecks develop in the foliage (227, 255). Tissues in portions of the chlorotic areas may become necrotic and fall from the lamina, resulting in a tattered appearance (227, 255). As additional leaves develop, the amount of chlorotic mottle becomes progressively less, and it is often entirely absent in the last leaves formed. In succeeding years, these acute symptoms are absent or affect only an occasional leaf. This phase of the prune dwarf syndrome is much like

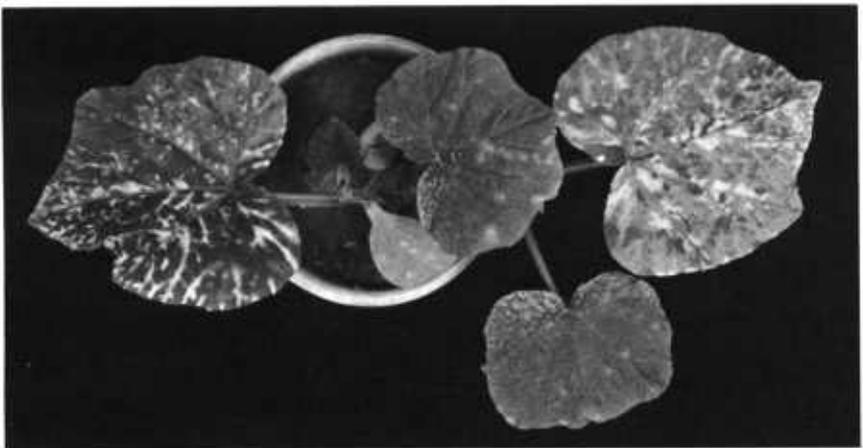


FIGURE 83.—Bright yellow mottle and vein-banding of Butternut squash induced by prune dwarf virus.

that of ring spot (227), but the etched appearance of ring spot lesions is absent, and the degree of necrosis is commonly much less (227 and R. M. Gilmer and K. G. Parker, unpublished data).

Chronic symptoms—leaf yellowing and leaf casting—may appear in the same year as the acute symptoms, but more commonly do not develop until 1 to 3 years later. Moore and Keitt (476, 477) and Mills (466) have shown that temperature is a critical factor in the development and severity of leaf casting yellows symptoms; symptoms are favored by temperatures of about 16° C. during the postbloom period and are limited or absent at higher temperatures. In the West, low night temperatures (10° to 16°) alternating with relatively high day temperatures (30° to 35°) seem particularly favorable for leaf casting symptoms. With favorable temperatures, leaf casting yellows may recur each year.

A striking yellow and green mottle appears in the foliage about 3 to 4 weeks after petal fall. The mottle pattern may be irregular, but more commonly green is retained along the midrib and larger veins, with the interveinal areas becoming yellow (fig. 84, *A* and *B*). Leaves with these symptoms are soon cast; many leaves may drop even before any significant degree of chlorosis is evident.

Older leaves are usually the first to develop mottle, which is often initially evident on the scale leaves. If temperature conditions are favorable, younger leaves may develop symptoms in the order of their age. Successive waves of yellowing and leaf casting may occur in varying severity throughout the early portion of the growing season, but these gradually decrease in severity and finally cease as temperatures increase. Under favorable conditions for symptom development, 30 to 50 percent of the leaves may drop.

On fruiting trees with infections of some duration, the leaves are fewer in number and somewhat larger in size than on healthy trees. The number of fruiting spurs is much reduced; those which persist tend to grow into shoots, so that infected trees eventually develop a willowy growth habit with long bare spaces on the twigs (fig. 84, *C* and *D*). Fruit yields may be reduced by 50 percent or more (184, 378). Although the fruits are fewer in number, they are large and of excellent quality. If young trees are infected, their growth is markedly checked (184).

Symptoms in Early Richmond cherry are identical with those just described. In English Morello sour cherry, the yellow color of leaves is less pronounced and often is restricted to the leaf margins, and the amount of defoliation is considerably less than in Montmorency, but the absence of fruiting spurs,

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FIGURE 84.—*A-C*, Sour cherry yellows in Montmorency cherry; *A*, Leaf showing irregular pale-green or yellowish mottling; *B*, leaf showing retention of green along main veins; *C*, affected branch showing willowy type of growth. *D*, Comparable normal branch with fruit spurs.



willowy growth habit, stunting of the trees, and reduction in fruiting are as pronounced.

Sweet cherry.—The initial symptoms of infection are the appearance of large chlorotic rings or mottle and varying degrees of necrosis and shot-holing of the foliage (fig. 85), symptoms identical with those described as "tatterleaf" (733). In some cultivars (Yellow Glass, Giant, Ebony, and Van), chlorotic rings and mottle, usually with little or no necrosis, recur each year; these symptoms are generally more prominent in the early leaves and absent in later leaves. In other cultivars (Emperor Francis, Windsor, Noble, and Hedelfingen), the recurrence of chlorotic mottle is rare or completely absent, and infected trees cannot be identified by visual inspection. Yields may be seriously reduced (540, 709), but some varieties are more tolerant of infection than others. A strain of PDV, recently described in California (493), induces narrow rough-textured leaves in sweet cherry and reduction in tree growth. Similar symptoms have been observed in New York (K. G. Parker, unpublished data).

Plum and prune.—In trees of Italian Prune infected with PDV, the leaves are narrow and straplike in shape, rugose, and somewhat thickened (fig. 86). Early in the season, the terminals are rosetted with shortened internodes and narrow leaves, but as temperatures increase, terminal growth may be almost normal, with normal leaves. In large trees, symptoms may be restricted to a



FIGURE 85.—Chlorotic ring spot and mottle on Yellow Glass sweet cherry leaves induced by experimental pollination with prune dwarf virus—infected pollen.



FIGURE 86.—Strap-shaped rugose leaves of Italian Prune affected by prune dwarf.

portion of the tree, or occasional branches with normal foliage are interspersed among those with severe symptoms. Hildebrand et al. (312) indicated that the flower petals were elongated and pistils frequently aborted, but that the fruits that matured were large and of excellent quality. In Lombard plum (312), symptoms were similar to those of Italian Prune but considerably milder, and yield reduction was slight.

In addition to Italian Prune and Lombard, experimental inoculation has produced prune dwarf symptoms on the following cultivars: Giant, Standard, Emilie, President, Tragedy, Green Gage, and Albion (150 and R. M. Gilmer, unpublished data). No symptoms are produced on cultivars of *Prunus salicina* except by the cherry narrow leaf strain (493).

In controlled temperature experiments in the greenhouse and growth chambers, a constant temperature of 22° C., beginning at budbreak and continuing until new growth ceased, completely masked prune dwarf symptoms in Italian Prune. Exposures of infected potted trees to a constant temperature of 22° for 48 to 72 hours reduced symptom severity or masked symptoms completely on certain leaves, whereas exposure to 12° for at least 10 hours every 48 hours resulted in development of typical prune dwarf symptoms. It was immaterial if the low temperature exposure was during the dark period or the light period (J. Duain Moore, unpublished data).

Peach.—In peach seedlings inoculated in New York, varying degrees of budkill and terminal dieback appeared in the spring after inoculation the

previous fall. Foliation was delayed, and chlorotic rings, mottle, or oak-leaf patterns appeared in the early foliage. These symptoms were probably caused by a PRSV contaminant. The seedlings were dwarfed, and their foliage was an intense deep green. Partial recovery occurred by midseason.

Inoculations of 2-year-old peach trees in the field in California with pure cultures of PDV produced only mild symptoms of stunting in inoculated branches and rootstock suckers in the spring following fall inoculation, and mild stunting persisted during the 4 years the trees were observed. Italian Prune growing on the inoculated peach trees showed severe symptoms of prune dwarf. However, when PDV was introduced into trees already infected with a pure culture of PRSV, moderate to severe symptoms of peach stunt were produced in succeeding years. In all cases, symptoms became less obvious as the season progressed but recurred each spring (George Nyland and J. A. Milbrath, unpublished data). The severity of symptoms on mature trees in the orchard varies among infected trees and is influenced by the pruning system used. The internodes of spring shoot growth are short, and the leaves on these shoots are more erect and darker green than those on normal shoots. In older infections, failure of many leaf buds to grow results in thinly foliated trees and bare branches with few lateral shoots. The shortened internodes and erect leaves are obvious until late May or early June after which shoot growth is nearly normal. Infected trees are smaller than healthy trees and yield less fruit. The disease is very conspicuous in the Muir peach (329).

Flowering cherry.—Inoculation by T-budding Shiro-fugen flowering cherry with tissue infected with PDV results in the development of an initially localized bark necrosis and gumming at the point where the tissue was inserted (fig. 87). In inoculations at midseason (July), these symptoms are evident in 4 to 6 weeks. If the infected shoots are not removed, the virus moves down the inoculated shoot and kills large branches and finally the entire tree. Hampton (273) indicated that the rate of movement averaged about 2 to 5 cm. per day.

Transmission

PDV is readily transmitted by budding or grafting. Gilmer and Brase (254) reported that the virus was irregularly distributed in sweet cherry and that some buds from infected trees were free of virus. Hampton (274) recorded some "false" negatives in indexing buds of infected sweet cherry on Shiro-fugen but concluded that the virus was present in too low titre for detection rather than being irregularly distributed.

PDV is readily transmitted through seeds of several stone fruits: mahaleb (123, 256), sweet cherry (256), and sour cherry (261). One almost certain instance of transmission through myrobalan seeds has been observed (R. M. Gilmer, unpublished data). In sour cherry, the percentage of infected seeds may range from 20 to 70 percent (261), and incidences of PDV infections of 15 to 30 percent have been found in commercial lots of mahaleb and mazzard seedlings (256). The infection of rootstocks through seed transmission un-



FIGURE 87.—Gumming and necrosis of Shiro-fugen oriental flowering cherry about 6 weeks after insertion of a sweet cherry bud infected with prune dwarf virus (PDV).

doubtedly accounts for much of the prevalence of PDV in sweet and sour cherries.

PDV also spreads naturally in the orchard. The spread in sour cherry is much more rapid than in sweet cherry (247). Little or no natural spread occurs in plum (651).

Typically, the rate of spread in sour cherry orchards is low for the first 2 to 4 years after planting but increases rapidly in trees 5 to 15 years of age until all or nearly all of the trees are infected. The initial spread is often concentrated around individual infected trees, but occasional trees some distance from an infection locus may become diseased. There is a rather pronounced tendency for new infections to occur downwind from sources of inoculum, and the same tendency has also been observed in the spread of peach stunt and Muir peach dwarf (329).

In 1961, Gilmer and Way (262) demonstrated that PDV was transmitted by pollen to seeds of sour cherry. Das and Milbrath (180) demonstrated plant-to-plant transmission of PDV in squash by pollen. George and Davidson (236, 237) soon afterwards showed that PDV was transmitted from tree to tree in sour cherry by pollen. Tree-to-tree transmission in sweet cherry and *Prunus fruticosa* also occurs via pollen (250). Deblossomed sour cherry trees remained noninfected in the field under conditions where rapid spread of PDV occurred in controls (236, 237, and J. A. Milbrath, unpublished data). These data suggest that pollen transmission is responsible for the spread of PDV in the orchard and that bees and other pollinating insects are indirect vectors.

The reasons for the very slow rate of spread of PDV in sweet cherry (247) and the virtual absence of spread in plum remain obscure, particularly since sweet cherry cultivars are obligately cross-pollinated. Some limited and inconclusive data (R. M. Gilmer, unpublished data) suggest that in the sweet cherry cultivars, Van and Hedelfingen, most of the ovules fertilized with infected pollen abort quickly, perhaps before the virus can be translocated from the flower. In the cultivars Windsor and Yellow Glass, such ovule abortion is limited or absent, and these cultivars are rather easily infected experimentally by pollen. Field trees of cherry, peach, and plum that index negative for PDV or PRSV may produce some infected seed (205). Ehlers and Moore (196) and Williams et al. (726) showed that pollen from infected trees contains virus. The latter workers successfully transmitted PDV to peach by inserting infected pollen beneath the bark (726).

Characteristics of the Virus

Fulton and his coworkers (226, 227, 271, 706) have determined the properties and morphology of PDV. PDV particles are isodiametric "spheres" about 22 m μ in diameter; it is thus one of the smaller plant viruses (228). The UV absorption spectra are typical of nucleoproteins (228).

In cucumber sap, the thermal inactivation point was 44° C. (226), but in squash extracts stabilized with DIECA and thioglycolate, some isolates were inactivated at 44°, whereas others remained infective at 54° (706).

At 1:80 dilution, PDV retained about half of the original infectivity after 3 hours at 24° C.; there was essentially no loss of infectivity after 3 hours at 0° (226). Polyphenol oxidase completely inactivated PDV, and oxidized catechol partially inactivated it (271).

The dilution end point is 10^{-2} — 10^{-3} (R. M. Gilmer, unpublished data).

PDV is rather easily inactivated in infected stone fruits by thermotherapy at 36° to 37° C. for 15 days or longer (488 and R. M. Gilmer and R. D. Way, unpublished data; P. R. Fridlund, unpublished data).

Control Measures

The first essential step in control is to provide nursery trees free of the virus. Both scion and rootstock must be free of PDV, and the seed-source trees

for seedling rootstocks must be maintained free of PDV and protected from infected pollen.

In New York, the incidence of PDV in certified cherry nursery trees was reduced from about 40 percent to a level of 1 to 2 percent by the use of indexed scionwood, procurement of clean rootstocks, and roguing (255). Elimination of PDV from the nursery will only be possible when virus-free rootstocks are available.

Orchards should be planted with certified nursery trees and well isolated from older infected trees. In the early years of the orchard (age 1 to 5 years), the trees should be visually inspected when leaf casting occurs (3 to 4 weeks after petal fall) and any infected trees should be removed. Vacant sites may be replanted safely. At tree ages of 6 to 10 years, continued removal of infected trees is probably advisable, even though replanting of vacant sites may not be economical. In orchards over 10 years of age, roguing of infected trees is probably useless, and the entire orchard may be left undisturbed until it becomes uneconomic and is removed entirely.

Replanting vacant sites in old orchards with virus-free trees is not recommended because the young trees will be infected as soon as they reach fruiting age.

Remarks

Because of its prevalence and the serious yield reductions that accompany the disease, sour cherry yellows is the most economically serious virus disease of sour cherries in midwestern and eastern North America. In orchards 12 to 15 years of age, which should be near peak production, the incidence of sour cherry yellows commonly approaches 100 percent, and the expected yields are reduced by half or more (184, 378).

In sweet cherries, where the presence of PDV is more difficult to determine, the effects of the disease in reducing the crop are not as well known. Yield losses are undoubtedly considerable. Way and Gilmer (709) showed that pollination with infected pollen may reduce fruit sets in sweet cherry by 25 to 90 percent. Posnette et al. (540) have estimated that the complex of virus diseases in England, of which PDV is an important component, reduces cherry yields by an estimated 30 percent or more. PDV in peach, especially when mixed with PRSV, is of considerable economic importance. Tree size and yields are markedly reduced.

Earlier publications (353, 354) on sour cherry yellows suggested that the disease resulted from infection by two viruses, PRSV and PDV. Dual infections are common in orchard trees (247, 589, 706), but the absence of PRSV from many trees with yellows (247, 706) and the development of sour cherry yellows following experimental inoculations with pure PDV isolates (225, 227, 228, 452, and R. M. Gilmer, unpublished data) indicate that PDV alone is capable of causing the disease. The added presence of PRSV, however, probably increases the severity of yellows symptoms (175, 225). Both the recurrent

necrotic ring spot and rugose mosaic strains of PRSV can duplicate the "yellows" symptoms in Montmorency induced by PDV (496). This suggests caution in identifying the disease solely on the basis of yellow leaves.

PDV exists naturally in a wide spectrum of strains that differ in properties (226, 706), herbaceous host range (225, 706), rate of movement (254, 453, 728), and symptomatology in *Prunus* (493, 706). Milbrath (452) has suggested that PRSV and "yellows" strains of ring spot virus (= PDV) may be strains of a single virus. This hypothesis is not supported by available information on some of the properties of the viruses or serological evidence (226, 227, 233, 669). Although PDV and PRSV are morphologically similar in size and shape (228) and the symptoms each induces in many *Prunus* spp. are similar, these viruses differ in herbaceous host range (225) and in some properties (226, 706). Some evidence for the relationship is that there is interference in Montmorency trees between the recurrent ring spot strain of PRSV and PDV. When healthy scions of Montmorency were grafted on mahaleb rootstocks previously infected with the recurrent ring spot strain, symptoms showed in the tops the following year. If Montmorency scions infected with PDV were grafted on similar mahaleb rootstocks, symptoms of recurrent ring spot did not appear fully distributed in the tops even after 3 years (J. A. Milbrath and George Nyland, unpublished data).

PRSV and PDV have many characteristics in common with the NEPO viruses, the soil-borne polyhedral viruses vectored by nematodes. The individual viruses of the NEPO group do not share common antigens, but there is clear evidence of natural affinity among members of the group (104). There is no evidence that PRSV or PDV are soil-borne, but it is conceivable that the viruses have become separated from a vector that may exist in the area of origin.

VIRUS DISEASES OF SOUR CHERRY BARK SPLITTING IN MONTMORENCY CHERRY

H. R. CAMERON

The bark splitting virus (BSV) was first reported in Montmorency sour cherry in 1954 (112). There are no known synonyms, but the symptoms incited in apricot by this virus are similar to those reported by Blodgett and Twomey (73) for apricot gummosis. The BSV was discovered in Montmorency trees budded with Napoleon sweet cherry from Oregon.

Symptoms incited by the virus have been described on Montmorency sour cherry and apricot. In Montmorency, narrow, brown stripes appear on the new shoots in midsummer. The bark usually splits for several inches or more along these stripes and gum may be exuded (fig. 88). Symptoms recur in succeeding years but usually not on all shoots and only on the current season's growth. Affected shoots tend to bend away from the trunk. In subsequent years, the areas of original splitting are somewhat flattened, and the limbs are weakened at these points. Symptoms on apricot are more striking and consist of gum pockets breaking through the bark. The gum flow is excessive, and all of the new growth dies back. A new flush of growth starts at about the time the tree appears to be dead. The gumming and dieback appear again, and the process is repeated until the tree dies. The virus has not incited symptoms on sweet cherry, Italian Prune (*Prunus domestica*), Shiro-fugen and Kwanzan flowering cherry (*P. serrulata*), or Abundance and Burbank plum (*P. salicina*). The virus was recovered from symptomless peach seedlings 13 years after they had been inoculated (J. Duain Moore, unpublished data).

The virus is easily transmitted by grafting, and symptoms are usually observed on the new growth the year following inoculation. Even though the bark splitting virus is widespread in Oregon sweet cherry orchards, it has not been known to spread to adjacent Montmorency trees.



FIGURE 88.—Splitting and necrosis of the bark of current-season growth on Montmorency cherry inoculated with bark splitting virus (BSV).

GREEN RING MOTTLE

K. G. PARKER, P. R. FRIDLUND, and R. M. GILMER

Causal Virus

Green ring mottle virus (GRMV).

Synonyms

Green ring yellows; constricting chlorosis; rough bark of oriental flowering cherry; epinasty of oriental flowering cherry.

History and Distribution

Green ring mottle of sour cherry was first reported in Michigan in 1937; by 1942, the disease was also observed in New York, Wisconsin, and Ontario (547). In 1945, GRMV was transmitted from symptomless sweet cherry trees in Oregon (461) and has since been recovered from symptomless sweet cherries in Washington (223), New York (247), and Switzerland (600). Leaf symptoms resembling those in sour cherry were reported in duke cherry in Idaho in 1955 (291).

A rough bark of oriental flowering cherry was observed in New York in 1937 (309) and in Oregon in 1942 (460). In 1959, a virus-induced epinasty of oriental flowering cherry was reported in New Zealand, and the same virus was recovered from symptomless sweet cherry and mazzard clonal rootstocks (1). Subsequent investigations (21, 223, 451) demonstrated that rough bark and epinasty were induced by GRMV and not by *Prunus* ring spot virus (PRSV) as previously supposed. This point should be kept in mind when referring to the early literature.

Economic Hosts

Natural infections of GRMV occur in Montmorency and English Morello cultivars of sour cherry (*Prunus cerasus*) and in oriental flowering cherry (*P. serrulata*) (1, 309, 460).

Other Hosts

Infections without symptoms are common in sweet cherry (*P. avium*) (213, 215, 223, 247, 445, 467, 600); the cultivars Bing, Lambert, Black Republican, Napoleon, and Deacon are often infected with GRMV. Foliage symptoms like those in sour cherry have been observed in duke cherry (*P. cerasus* × *P. avium*) cultivars Late Duke (291), May Duke, and Reine Hortense (R. M. Gilmer, unpublished data).

Peach, nectarine, and apricot are symptomless carriers of GRMV (213). The peach cultivars Halehaven, Sunhaven, Richaven, Glohaven, Suncling, and Rio Oso Gem appear to be universally infected (127 and P. R. Fridlund, unpublished data).

Preliminary results (P. R. Fridlund, unpublished data) indicate that *P. tomentosa* is susceptible to infection and *P. cistena* is immune. GRMV was

transmitted experimentally to *P. mahaleb* (547) and has been recovered from a symptomless mahaleb clone of German origin (R. M. Gilmer, unpublished data). Milkweed (*Asclepias syriaca*) has been suggested as a possible host of GRMV and other cherry viruses (336), without later confirmation.

Symptoms

Sour cherry.—In mature leaves of Montmorency, a yellow and green mottle (persistent green islands or ringlike bands) appears about 4 to 6 weeks after petal fall (fig. 89). Some GRMV strains may also induce irregular necrotic spots of varying size in Montmorency leaves (P. R. Fridlund, unpublished data).

The yellow and green mottle develops predominantly over a 2 to 3 week period, and leaves with these symptoms soon drop. Mottle may continue to appear in an occasional leaf as late as September in some areas. Mottle symptoms do not necessarily occur each year, and some GRMV strains may incite no symptoms in infected Montmorency trees (P. R. Fridlund, unpublished data).

An additional foliage symptom, constricting chlorosis (247), appears to result from the failure of sections of the leaf lamina to develop normally in areas along the midribs or major lateral veins. Linear areas of chlorotic and distorted tissue appear along these veins (fig. 89). Constricting chlorosis appears during the period of leaf expansion, and the affected leaves persist indefinitely.



FIGURE 89.—Green ring mottle in Montmorency cherry. *Right*, a yellowing leaf with persistent green rings and islands; *center*, constricting chlorosis along a lateral vein; *left*, a normal leaf.

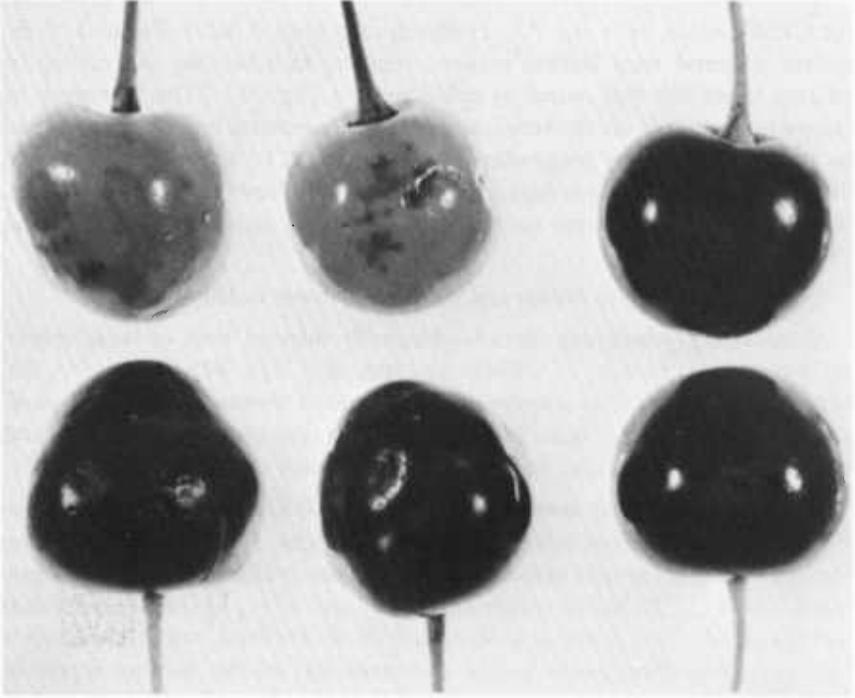


FIGURE 90.—*Left*, pitting and necrosis of Montmorency cherry fruit affected by green ring mottle; *right*, normal Montmorency fruit.

In fruits on infected trees, some GRMV strains induce necrotic pits or rings in the epidermis with ring-shaped areas of necrosis deep in the flesh (fig. 90). The affected fruits are bitter and off-flavor (454, 510). Other GRMV strains may induce subepidermal netlike patterns of necrosis in fruits, visible only by transmitted light (454 and P. R. Fridlund, unpublished data).

Leaves with the yellow and green mottle are most abundant in the interior, shaded portions of infected trees, but fruit symptoms and constricting chlorosis are randomly distributed throughout the tree (21).

In infected English Morello trees, the green and yellow mottle of leaves and necrosis of fruits are lacking. In English Morello trees inoculated with a mixture of GRMV and PRSV, scattered symptoms of constricting chlorosis were observed, suggesting that some GRMV strains may incite symptoms in this cultivar (20).

Duke cherry.—The characteristic green and yellow leaf mottle occurs in at least three cultivars: Late Duke, May Duke, and Reine Hortense.

Oriental flowering cherry.—Infected trees of Shirofugen and Kwanzan show epinasty of the foliage that may range from very mild to severe, depending on

the GRMV strain (454 and P.R. Fridlund, unpublished data). Portions of the midrib or lateral veins become necrotic, resulting in a twisting and curling of affected leaves like that caused by aphid feeding (fig. 91). The internodes of elongating terminals are shortened, and as the bark matures it is often roughened by the development of longitudinal fissures (fig. 91). Considerable terminal dieback usually occurs in infected Kwanzan trees in New York, but at Prosser, Wash., dieback is apparent only when GRMV is contaminated with certain PRSV strains.

Symptoms are mild in Naden and completely absent in Shirotae (460).

Sweet cherry, peach, and apricot.—Naturally infected trees of these species are symptomless carriers of GRMV (1, 208, 213, 247, 445, 454, 467, 589, 600). No differences in symptoms were observed between peach and sweet cherry trees inoculated with PRSV and others inoculated with PRSV and GRMV; the symptoms that developed were attributed to PRSV (20, 21, 518).

The development and severity of symptoms of GRMV infections in various *Prunus* hosts is influenced by environmental conditions. In the oriental flowering cherries, leaf epinasty and veinal necrosis are most pronounced at cool temperatures (18° C.). At higher temperatures (22° and 26°), a mild, limited veinal necrosis was the only symptom in Kwanzan (P. R. Fridlund, unpublished data). Conversely, the characteristic yellow and green leaf mottle does not appear in the foliage of infected Montmorency sour cherry until about midseason, when ambient temperatures are relatively high.

In experimentally shaded Montmorency trees in the field, Barksdale (21) found that low light intensity favored the development of leaf mottle and that low light intensity at a temperature regime of 21° C. or a diurnal regime of 24° to 16° resulted in pronounced leaf symptoms. In other experiments (P. R. Fridlund, unpublished data), symptoms were very pronounced in infected Montmorency trees at 26° to 30°; mottle symptoms were absent in infected trees at 18°. Large necrotic spots appeared in leaves at 22°; these spots were considerably smaller at 30°.

Transmission

GRMV is readily carried in scionwood used in propagation, particularly in symptomless carriers such as sweet cherry, peach, and apricot. GRMV appears to be completely systemic in infected peach and sweet cherry. However, there is preliminary evidence that GRMV may not be completely systemic in some infected Montmorency trees (P. R. Fridlund, unpublished data).

Natural spread of GRMV in sour cherry orchards has been observed repeatedly (21, 454). The grouping of infected sour cherry trees is often in a pattern suggesting spread from a central locus. Because the rate of spread is relatively slow, some of the local spread may result from root grafts between adjacent trees.

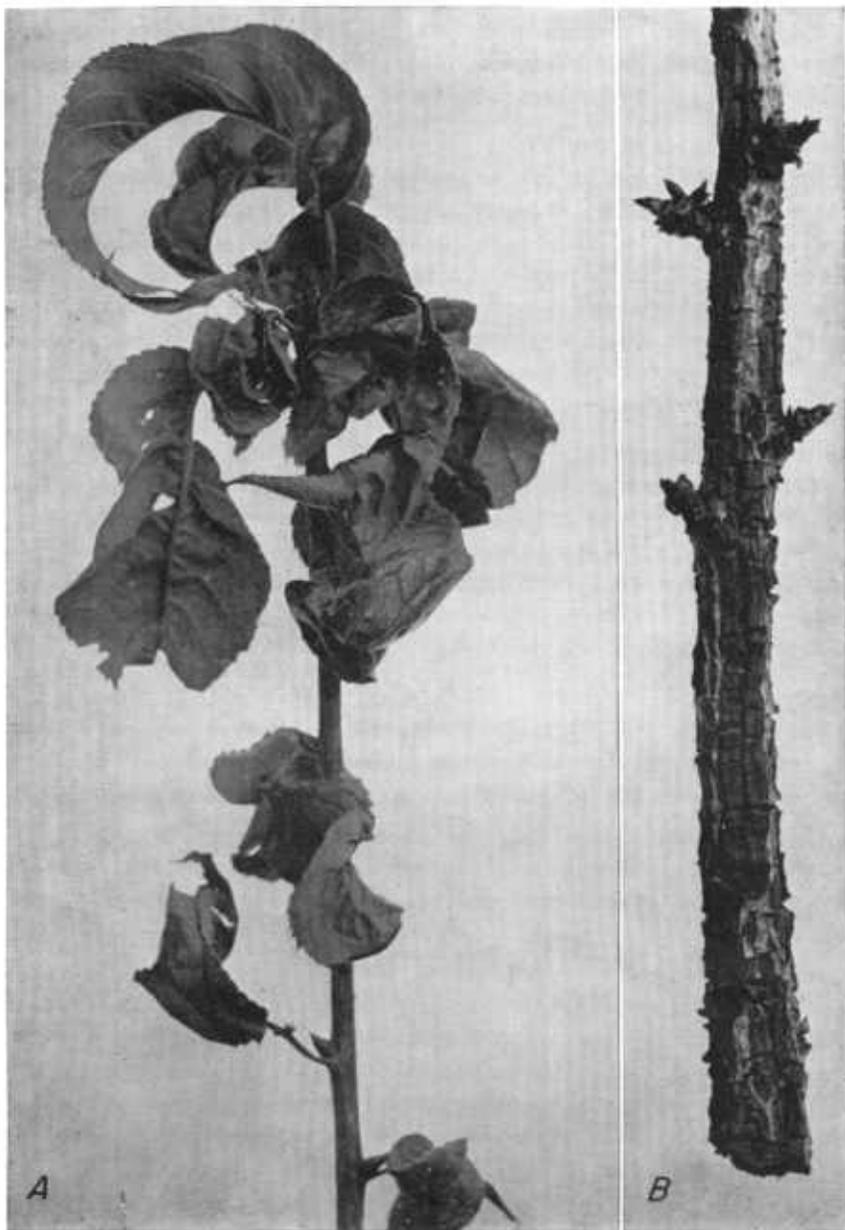


FIGURE 91.—*A*, Current-season growth of Kwanzan oriental flowering cherry infected with green ring mottle virus (GRMV), showing characteristic twisting and curling of leaves; *B*, older infected branch showing severe splitting and rough bark symptoms.

Evidence against transmission of GRMV through cherry seeds appears conclusive (127, 219, 253). Attempts to transmit GRMV mechanically to woody or herbaceous plants have been unsuccessful. No vectors are known.

Characteristics of the Virus

Because GRMV has not been transmitted mechanically, very little is known of its properties. GRMV is highly resistant to heat therapy. An exposure at 36° to 37° C. for 13 days or more inactivated PRSV but not GRMV in sweet cherry (451). P. R. Fridlund (unpublished data) did not eliminate GRMV from infected sweet cherry or peach buds exposed to 38° C. for 21 days or from sweet cherry buds after hot-water treatment at 50° C. for 20 minutes. Nyland (491), however, reported eliminating GRMV from Stockton Morello cherry buds with thermo-therapy (38° C. for 6 weeks).

Control Measures

The sole method of controlling GRMV is to propagate and plant trees free of the virus. Sources of propagating materials may be indexed on Kwanzan flowering cherry, which appears superior to Montmorency cherry as an indicator for GRMV. Because GRMV is not seed transmitted, seedling rootstocks may be safely used for propagation. The removal of infected trees may prevent spread in sour cherry orchards.

Remarks

Green ring mottle is potentially an important disease of sour cherries because the fruits of infected Montmorency trees are frequently unmarketable. Its effects on fruit production and tree growth have not been studied adequately.

GRMV is common in several commercially important sweet cherry cultivars, but the amount of economic injury that it causes, if any, is not known. Limited data suggest that infected sweet cherry nursery trees are slightly smaller than healthy ones (445). In peach, latent GRMV does not appear to reduce either yields or growth of infected trees (518).

The prevalence of GRMV in some sweet cherry cultivars is probably a result of their having been topworked on diseased rootstocks (215, 467), but its widespread occurrence in other sweet cherry cultivars (see "Other Hosts") suggests that the original cultivars sources may have been infected. Extensive indexing at IR-2 Repository indicates that some peach cultivars are universally infected, again suggesting that the original sources were infected.

The virtual restriction of GRMV to clones of North American origin suggests that the virus is indigenous to North America perhaps to the West because the virus is more common in western sweet cherry cultivars than those originating in the East (213, 215, 467, 589).

An earlier suggestion that GRMV might be a strain of sour cherry yellows virus (prune dwarf virus) (547) has not been supported by additional data. Green ring mottle disease spreads in Montmorency sour cherry orchards inde-

pendently of the other viruses (21). Its rate of spread is generally much slower than that of sour cherry yellows or *Prunus* ring spot, and the pattern of spread, which tends to concentrate around a central locus differs from the spread patterns of the latter diseases. Moreover, GRMV has not been transmitted to any of several herbaceous plants that are readily infected by PRSV or PDV. GRMV passes across graft unions at a slower rate than PRSV or PDV (222).

PINK FRUIT

P. W. CHENEY, C. L. PARISH, and FOLKE JOHNSON

Pink fruit was reported first in Washington in 1934 (287) and was shown to be caused by a transmissible virus (PFV) in 1940 (554). Orchard surveys conducted in 1939 and 1942 revealed from 2 to 9 percent of the Montmorency sour cherry trees in northwestern and central western Washington were infected (166, 565). In terms of reduced sour cherry yield, pink fruit was considered second in importance only to brown rot (*Monilinia laxa* Aderh. and Ruhl.) (568). Pink fruit occurs in the sour cherry-growing areas of western Washington from the Canadian border to the Columbia River. A similar-appearing disorder, reported in Idaho (47), may have been caused by the western X-disease virus (E. C. Blodgett, personal communication).

Montmorency sour cherry is the only cultivar known to be naturally infected by PFV. Inoculated trees of Early Richmond sour cherry and Ronald duke cherry show typical pink fruit. Only questionable symptoms occur on Royal and Bicentennial duke cherry. Bing, Lambert, and Star sweet cherry are symptomless carriers. Italian Prune (*Prunus domestica*), several peach cultivars, and seedlings of *P. emarginata* and *P. mahaleb* are immune.

Diagnostic symptoms of pink fruit occur in the fruit (fig. 92), first appearing about 2 weeks prior to harvest. Affected fruit are smaller than normal, pinkish yellow to pinkish brown, and have brown, necrotic areas in the flesh. Affected fruit lack flavor and tend to drop prematurely. Normal and affected fruit may occur on the same branch or even on the same spur of a tree. The ratio of pink to normal fruit varies with the length of time elapsed since inoculation and also the size and vigor of the tree when infected.

Leaves of affected trees may fold upward along the midvein, presenting the appearance of drought. Diseased trees bloom later than normal, and tree growth is suppressed.

Rootstocks have a marked effect on symptom expression. Although Montmorency trees on mahaleb stock may show symptoms the second year after inoculation, these are meager, and the trees usually appear to recover. Conversely, Montmorency on mazzard roots become more severely diseased until all the fruit are affected. Montmorency on its own roots is more severely affected than when on mazzard.

Transmission has been effected only by some form of grafting, using shoot or root material. Symptoms appear from 15 to 27 months following inoculation.

Some diseased trees are still to be found scattered throughout the former fruit-growing areas of western Washington. Most of these are in backyard orchards. Replacement of diseased trees with virus-free Montmorency on mahaleb roots could, in time, eliminate loss from pink fruit.



FIGURE 92.—Montmorency cherry fruit affected by pink fruit, showing small pinkish yellow to pinkish brown color and necrotic areas in the flesh.

VIRUS GUMMOSIS OF MONTMORENCY

EARLE C. BLODGETT and MURIT D. AICHELE

An undescribed virus was discovered (69) as a result of indexing mahaleb seed source trees in the Pacific Northwest (1954-59) for sour cherry yellows virus (SCYV) and *Prunus* ring spot virus (PRSV).

Trees of Montmorency on mahaleb rootstock were established at Puyallup and Bellingham, Wash., to test for SCYV. The trees of Montmorency were bud-inoculated in the early fall with tissue from the seed source trees and observed for symptoms the following summer. Most symptoms were typical, but Montmorency trees inoculated with tissue from seven mahaleb trees gave a new and severe reaction. Previous indexing of these same trees on Shiro-fugen at Prosser, Wash., and Corvallis, Oreg., had failed to detect PRSV.

The original seven mahaleb trees carrying virus gummosis virus (VGV) showed no symptoms or apparent effect on their growth. Healthy Montmorency trees bud-inoculated in the fall with infected mahaleb budwood showed symptoms the following growing season. Growth commenced normally in the spring. When the new Montmorency shoots were several inches long (up to 12 inches), the terminals died and curled downward (fig 93). New growth often took place from buds below the dead tip, and this top killing usually was repeated one or more times during the season. Some of the current year's shoots developed necrotic areas even before the tips died. Gum pockets formed on the new shoots and beads of masses of gum exuded. The same condition also occurred under the bark of the scaffolds and trunk until, by the end of the season, the whole tree became a mass of puffy blisters and gum. Affected trees are dwarfed, with numerous, short, side branches and weak growth (fig. 94).

This type of gumming was first observed on Montmorency trees inoculated in 1949 at Corvallis. The inoculum had been taken from a Napoleon sweet cherry tree on mahaleb root. Natural spread of the disease was observed in a block of 100 virus-free trees of Montmorency at The Dalles, Oreg., in 1952. Five trees developed gumming and tip dieback symptoms in 1959.

VGV is not seed transmitted and does not incite symptoms on Bing and Lambert cherry.

Montmorency trees propagated on mazzard rootstock with virus gummosis-infected budwood were affected less than those on mahaleb.

Although a great many mahaleb as well as other seed and scion source trees have been indexed over the years in Oregon and Washington, only the original seven trees infected by this virus have been discovered.



FIGURE 93.—Current year's growth of Montmorency cherry affected by virus gummosis, showing typical dieback of the growing shoots.



FIGURE 94.—*Right*, three healthy Montmorency cherry trees; *left*, three Montmorency cherry trees inoculated with buds from mahaleb seed trees affected by virus gummosis of Montmorency. All trees are 5 years old.

VIRUS DISEASES OF SWEET CHERRY

ALBINO

H. R. CAMERON

Albino virus was discovered in the Rogue River Valley of southern Oregon in 1937 (773). The virus moved rapidly within the valley and eliminated all the commercial sweet cherry acreage. With the loss of the orchards, the virus virtually disappeared and now is only found rarely. Most sweet cherry cultivars, on either mazzard or mahaleb rootstocks, are severely affected. Lambert, Black Tartarian, Macmar, and Montmorency sour cherry exhibit milder symptoms and are not killed as quickly. Peach is not susceptible to albino virus.

Symptoms are usually quite striking, consisting of limb dieback and leaf casting. The leaves are rolled inward and highly colored. Color varies from orange to bright yellow to golden bronze through olive-brown. Leaves produced in late summer come from terminal buds and are rosetted and small. Young green fruits remain small, become light colored, and fail to mature (fig. 95). A



FIGURE 95.—Normal Bing cherry, *left*; Bing cherry affected by albino, *right*, showing small, light-colored fruit that failed to ripen.

heavy fruit drop may occur prior to harvest, but some small white fruit remain attached well into the fall. Symptoms become more severe with time and result in tree death in 1 to 4 years.

The virus is easily transmitted by budding or grafting, but a vector has not been found. Inoculated sweet cherry trees died faster on mahaleb than on mazzard rootstocks (765). Inoculated Montmorency sour cherry reacted more severely on mazzard than on mahaleb rootstocks. Inoculated peach trees did not transmit the virus when indexed on susceptible selections of sweet cherry.

Diseased tree removal was not effective during the period of rapid virus spread. Recently, Lambert and Macmar have been planted in the Rogue River Valley. The only commercial orchard is now 9 years old and has not shown any symptoms of the albino disease. Inasmuch as this orchard has escaped infection, it appears that either the virus is not present in other hosts or the vector is no longer active. Provided the virus is not spread from occasional infected backyard trees, it may be possible to reestablish plantings of sweet cherry in the Rogue River Valley.

APPLE CHLOROTIC LEAF SPOT AND OTHER FILAMENTOUS VIRUS INFECTIONS IN *PRUNUS*

A. JUERGEN HANSEN and R. M. GILMER

Causal Viruses

Until the viruses of this rather heterogenous group have been classified more precisely, they may be tentatively referred to as filamentous *Prunus* viruses. Some isolates of this group have been designated as raspberry bushy dwarf virus (105) or apple chlorotic leaf spot virus (CLSV) (25, 251). These viruses should not be confused with the cherry chlorotic leaf spot virus of some European authors (25, 533), which is distinct.

History and Distribution

Filamentous viruses have been found repeatedly in *Prunus* during the last 10 years in the United Kingdom (105, 194, 195) as well as in Germany (29), Poland (763), and Switzerland (90). Similar viruses were recovered from sweet cherry trees in the United States in 1967 (251 and R. M. Gilmer, unpublished data) and in Canada in 1968 and 1969 (276 and A. J. Hansen, unpublished data). Although no field surveys have been made, these reports suggest that filamentous viruses may be fairly common in commercial and ornamental *Prunus* hosts (536).

Hosts

In Europe, filamentous viruses have been isolated from apricot, flowering plum, peach, European plum; Pandy, and Black Spanish sour cherry; Volynska; sweet cherry; and the species *P. colina*, *P. fruticosa*, and Krassa Severa duke cherry (90, 106, 194, 195, 763). The North American isolates were obtained from trees of the European sweet cherry cultivars Emperor Francis, Noir de Chaournes (251), Frueheste der Mark, Pimento, and Yellow Spanish (R. M. Gilmer, unpublished data) in a New York collection, and from cultivars Lambert (276) and Stella (A. J. Hansen, unpublished data) in British Columbia. The filamentous viruses were completely latent in the above seven infected sweet cherry cultivars. One cultivar, Emperor Francis, had previously been indexed on a range of woody stone fruit virus indicators and had failed to induce symptoms in any of them.

Symptoms

In plum, symptoms much like those of plum pox (sarka) were observed in England in a Wardwickshire Drooper tree from which CLSV was isolated (195). In peach, infection with cherry CLSV resulted in the development of narrow, slightly depressed meandering lines, darker green than the remainder of the leaf lamina (195 and R. M. Gilmer, unpublished data). This dark-green, sunken mottle was often restricted to one or two leaves of an infected seedling. Similar symptoms occurred in inoculated apricot seedlings, but the mottle pattern

was generally more ringlike (R. M. Gilmer, unpublished data). The Canadian isolates did not induce any symptoms in either apricot or peach (A. J. Hansen, unpublished data).

In France, a decline of apricot and Japanese plum is associated with scion-rootstock incompatibility induced by CLSV (R. Bernhard and J. Dunez, unpublished data). CLSV also incites fruit deformities (pseudo plum pox) in certain European and Japanese plum cultivars (R. Bernhard and J. Dunez, unpublished data).

Among herbaceous plants, several members of the Chenopodiaceae are susceptible, and *Chenopodium amaranticolor* and *C. quinoa* are useful indicators (fig. 96).

Transmission

Graft transmission from infected trees to susceptible woody hosts is readily achieved, although complete invasion of the inoculated tree may take 12 months or longer. The natural occurrence of filamentous viruses in a number of *Prunus* cultivars suggests that transmission may occur in the field, but how transmission is effected is not known. Numerous attempts to transmit the New York isolates with dodder from woody or herbaceous hosts have been unsuccessful (R. M. Gilmer, unpublished data). Attempts to detect the Canadian isolates in seeds or seedlings of infected sweet cherry and *C. quinoa* gave similarly negative results (276 and A. J. Hansen, unpublished data).

Characteristics of the Viruses

The characteristics of the filamentous viruses have been only partially determined: the thermal inactivation point (10 minutes) is between 42° and 50° C. (251, 276), dilution end point is between 10⁻³ and 10⁻⁴ (276), longevity in crude sap at 20° is several hours, and particle length as measured by electron microscope is around 700 mμ.

Control Measures

The economic justification for control measures is doubtful because filamentous viruses are not associated with any particular *Prunus* disease, and because their mode of field transmission is unknown. Their presence, however, might synergistically increase injury from other viruses, and they may confuse indexing results. Healthy scionwood could presumably be selected by indexing petals of candidate material on *C. quinoa*.

Remarks

The discovery of filamentous viruses in stone fruits is relatively recent. It is therefore impossible to even roughly estimate their prevalence in orchards or their economic importance. These viruses were probably previously overlooked simply because they do not induce symptoms on the commonly used woody indicators, or because they infect the same herbaceous hosts as *Prunus* ring spot and prune dwarf viruses.

The relationships of the cherry isolates to other filamentous fruit tree viruses

are still being investigated. The New York cherry isolates correspond in most characteristics to those of apple CLSV (Lister's type 1 apple virus) (251, 405, 406). The British Columbia isolates have been separated into two types on the basis of host range, symptom expression in herbaceous hosts, and serological relationships (A. J. Hansen, unpublished data).



FIGURE 96.—Necrotic lesions surrounded by green rings on a leaf of *Chenopodium quinoa* inoculated with chlorotic leaf spot virus (CLSV) from a Lambert cherry tree.

BLACK CANKER

P. W. CHENEY, EARLE C. BLODGETT, and C. L. PARISH

Black canker was first described in Oregon (774) and has been reported in Washington and the Okanagan Valley, British Columbia (767). Sweet cherry (*Prunus avium*) is the only known host. Napoleon is the most severely affected cultivar, but severe symptoms have also been noted on Bing, Republican, and Deacon.

The most distinctive symptoms of this disease are the rough black cankers occurring on the scaffold limbs and branches of affected trees. Cankers start as swollen areas on branches and twigs; and the bark splits lengthwise along these swollen areas forming rough, thickened ulcerlike wounds (fig. 97). In some



FIGURE 97.—Rough, black cankers on limbs of sweet cherry tree affected by black canker.

instances, cankers may completely girdle a twig or branch, causing dieback to occur. Affected trees are generally debilitated. No fruit or leaf symptoms have been observed.

Transmission has been effected by grafting shoot tissue from an affected tree into a susceptible host. Symptoms occur 2 to 3 years following inoculation.

The nursery certification program, increased grower awareness of virus problems, and the apparent absence of vectors have combined to make a rarity of this potentially economic important disease.

BLOSSOM ANOMALY OF CHERRY

C. L. PARISH, P. W. CHENEY, and E. W. ANTHON

This disease was noticed first in 1964 on a Bing sweet cherry tree (*Prunus avium*) near Wenatchee, Wash. (137). Spurs from this tree were grafted to healthy Bing trees. In one instance, 3 years elapsed between inoculation and the appearance of symptoms; in another instance, symptoms appeared 1 year after inoculation. In the first case, the trees were quite old and there was an appreciable distance between the inoculation site and the first flower bud. In the second case, the trees were young and there was only 1 inch between the inoculation site and the first flower bud. As symptoms began to appear in the inoculated trees, varying amounts of green developed along the midribs of the normally white petals. The amount of coloration in some blossoms ranged from a narrow green streak, not much wider than the veins, to entire green petals (fig. 98). Some petals, as well as being entirely green, were leaflike in appearance. Sometimes entire flower buds looked like leaf buds, except for the yellow



FIGURE 98.—Bing cherry blossoms affected by blossom anomaly of cherry, showing green areas in petals.

anthers in the center of the bud. In some flowers, a few of the anthers differentiated into wide, petallike structures with green centers; in other flowers, the entire stamen resembled a narrow petal. In a few instances, stigmalike and stylelike structures arose from the top of anthers.

Fruits developing from the affected blossoms were more pointed than normal, had roughened skin, and usually had a persistent style (fig. 99). The disease spreads very slowly in affected trees. Although the original test trees are old, it has been 3 years since symptoms were first seen, and they have advanced only about 5 feet from the inoculum sites. The disease cannot be considered economically important at the present time.

The symptoms described above resemble those induced by aster yellows virus in other plants. However, numerous attempts to transmit the causal agent of blossom anomaly to aster by the six-spotted leafhopper have been negative.

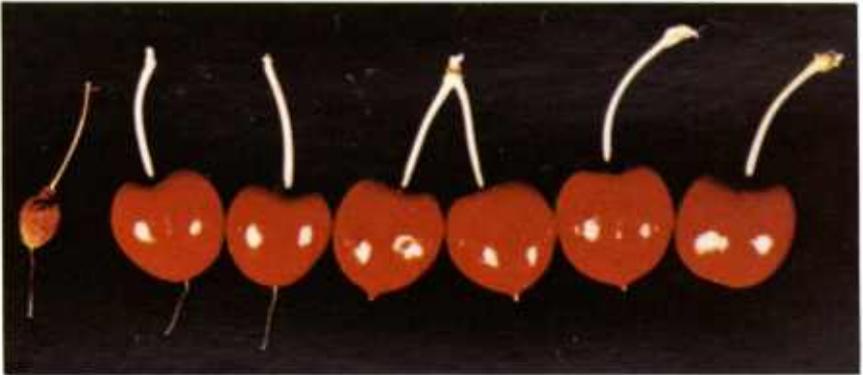


FIGURE 99.—Fruits from a Bing cherry tree affected by blossom anomaly of cherry. The cherry on the *left* is unfertilized; the five cherries in the *middle* are more pointed than normal, and at least part of the style has persisted on each. The fruit on the *right* is a normal Bing fruit.

Causal Virus

This disease may be caused by the interaction of a virus with its host to cause boron deficiency. The rosette symptoms have been transmitted by grafting (455) but have been corrected by application of boron (456). A strain of cucumber mosaic virus isolated from infected trees is the suspected etiological agent, but conclusive evidence for this is still lacking.

History and Distribution

Similar symptoms in cherry were first observed in 1952, but did not reoccur the following year. In 1962, trees with symptoms were observed near Salem and the Mosier area of Oregon (J. A. Milbrath, N. D. Dobie, and H. R. Cameron, unpublished data). In the following years, additional trees with symptoms appeared in both areas.

Economic Hosts

The virus has not been isolated from woody hosts other than sweet cherry. Mahaleb cherry (*Prunus mahaleb*) and peach (*P. persica*) have been infected by tissue grafts but have shown symptoms only occasionally.

Other Hosts

A virus was transmitted by sap from rosetted cherry trees through National Pickling cucumber to bean (*Phaseolus vulgaris*) and directly to bean from peach infected by tissue grafts from cherry.

Symptoms

The most striking early symptom in cherry is the complete loss of crop on the affected areas of the tree (fig. 100). The flowers open late and then abscise prior to fruit set. Leaf symptoms are similar to those for boron deficiency. Leaves are elongated and narrow and curve in toward the midrib. This may be accompanied by an interveinal chlorosis. In older infections, the leaves originate from the terminal buds, giving a rosette of leaves at the end of bare branches (fig. 101). The boron content of diseased tissue is usually lower than that of normal tissue. Symptoms on bean consisted of epinasty similar to that caused by cucumber mosaic virus (88). No symptoms were reported on squash (*Cucurbita pepo*), tobacco (*Nicotiana tabacum*), petunia (*Petunia hybrida*), *Chenopodium amaranticolor*, *C. quinoa*, *Gomphrena globosa*, *Vinca rosea*, or zinnia (*Zinnia elegans*).

Transmission

Six graft-inoculated sweet cherries developed rosette symptoms within 3 years. The two noninoculated check trees remained healthy. All trees were in the field.



FIGURE 100.—Loss of fruit on sweet cherry branch affected by cherry boron rosette (*arrow*); normal branch, *below*.

Transmission to herbaceous hosts has been irregular, and symptoms have not been observed on trees inoculated from herbaceous hosts.

Control Measures

Because a deficiency of boron was associated with diseased tissue, spray and soil applications of boron were tried. The vigor of the trees improved whenever boron was applied. In some cases, two spray applications eliminated symptoms during the following year. Application of boron has now become an acceptable control for this disease.

Remarks

The data suggest an interaction in the tree between a virus and either the movement or metabolism of boron.

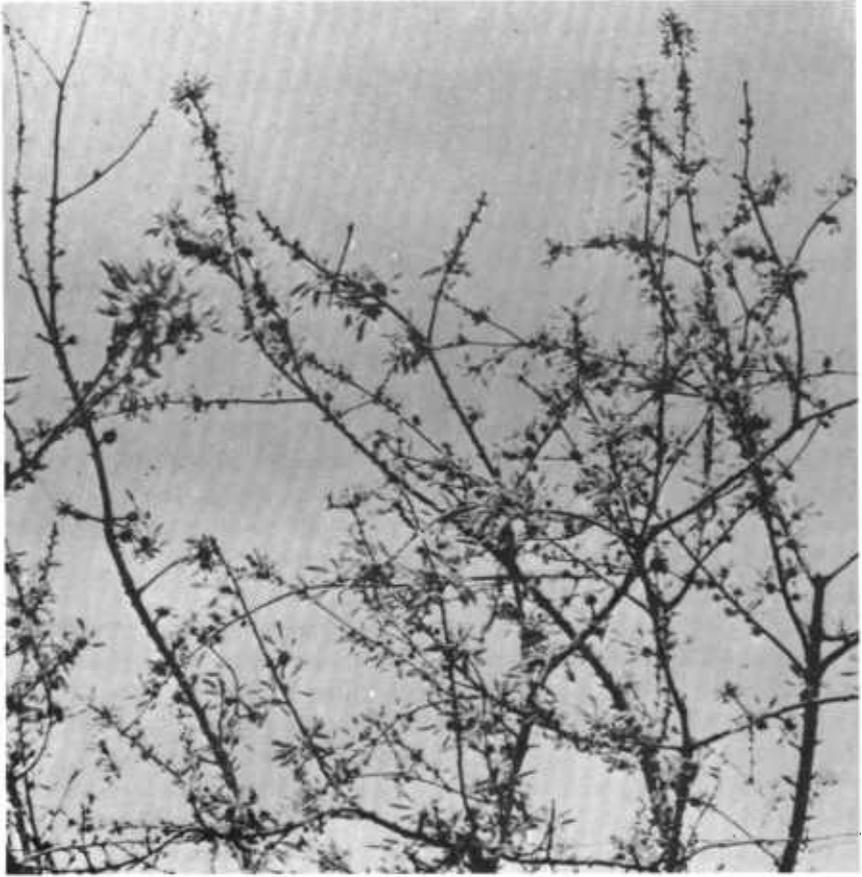


FIGURE 101.—Rosettes of narrow leaves on sweet cherry tree affected by cherry boron rosette (second year symptom).

CHERRY MOTTLE LEAF

P. W. CHENEY and C. L. PARISH

Causal Virus

Cherry mottle leaf virus (CMLV).

Synonyms

Severe mottle leaf (rugose type); mild mottle leaf (smooth type) (430, 766).

History and Distribution

Mottle leaf was first observed in Oregon in 1920 (764). The infectious nature of the disease was established in 1935 (429, 551). Cherry mottle leaf occurs generally in the sweet cherry-growing districts of Washington, Oregon, Idaho, and British Columbia, and has been seen in California, Utah, and Montana (430). Cherry mottle leaf is most prevalent in foothill or canyon orchards where wild bitter cherry (*Prunus emarginata*) is growing.

Economic Hosts

CMLV occurs naturally in sweet cherry and peach. Peach is symptomless when infected by most strains of the virus. Transmission tests show the following cherry cultivars are severely affected by cherry mottle leaf: Bing, Heldefingen, Lamida, Larian, Sodus, Velvet, and Vic. Napoleon (Royal Ann) is intermediately affected; Republican and Van are mildly affected.

Thirty-seven sweet cherry cultivars are symptomless or nearly symptomless. Among these are Bada, Berryessa, Black Tartarian, Burbank, Chapman, Chinook, Corum, Deacon, Ebony, Gil Peck, Jubilee, Lambert, Mona, Rainier, Sam, Schmidt, Seneca, Spalding, Sparkle, Star, Stark Gold, Sue, Victor, and Windsor. Sour cherry and duke cherry (*Prunus avium* x *P. cerasus*) are also practically symptomless carriers of CMLV.

Other Hosts

Mottle leaf has been found naturally occurring in apricot and ornamental flowering cherries (*Prunus serrulata* cv. Kwanzan (417) and Amanogawa and *P. yedoensis* cv. Yoshino).

Transmission tests show that the following are essentially symptomless carriers: Burbank plum (*P. salicina*), myrobalan plum, Drake and Nonpareil almond, and seedlings of David peach, Amur cherry, mahaleb cherry, black cherry, and Manchu cherry.

Apple and pear cultivars are resistant to the virus as are Italian Prune (*P. domestica*) and seedlings of desert peach, Bessey cherry, and Klamath plum.

Symptoms

The most distinctive symptom of mottle leaf is the irregular chlorotic mottling and distortion of the foliage, especially terminal leaves (fig. 102). High

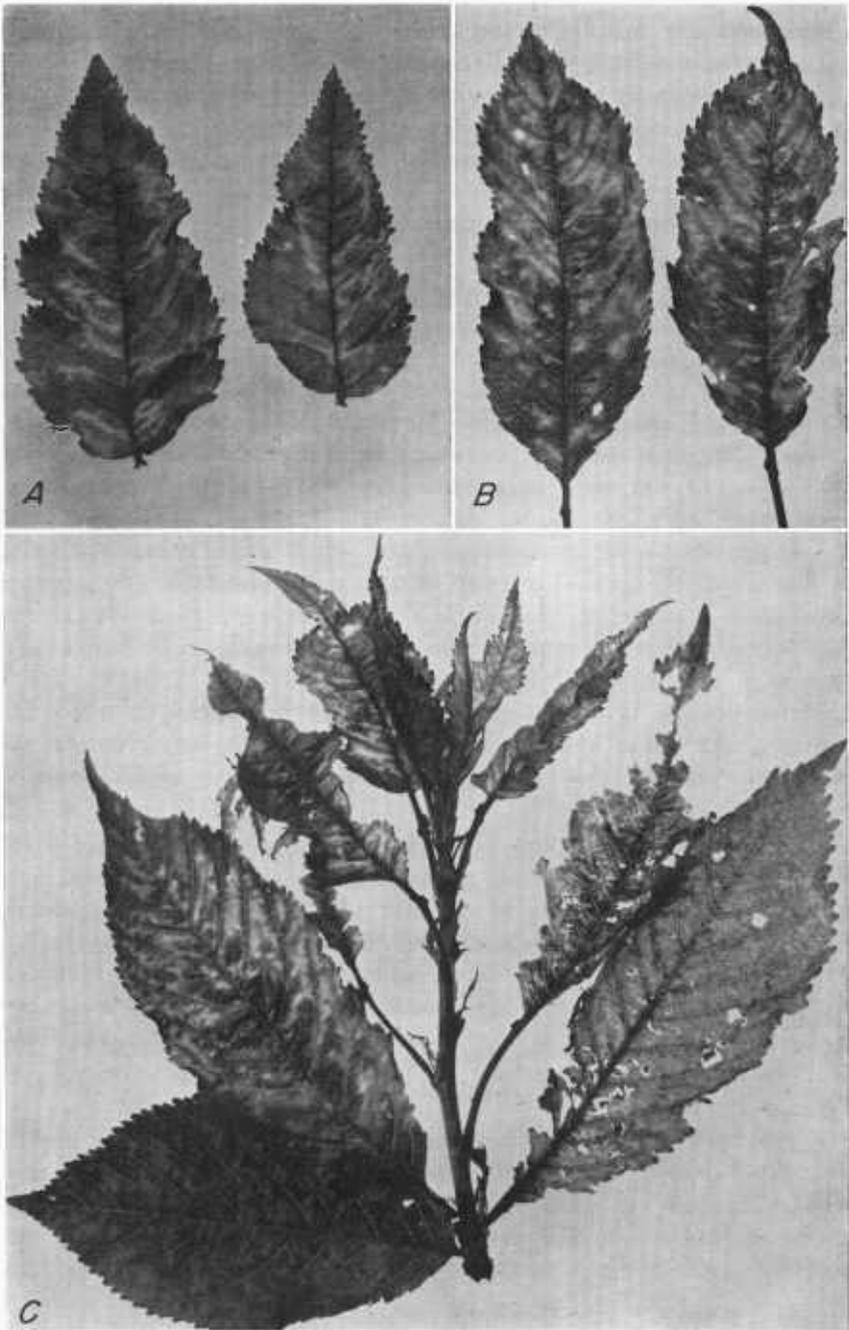


FIGURE 102.—Mottled and deformed leaves of *A*, Napoleon and *B*, Bing cherry affected by cherry mottle leaf. *C*, Shoot of Bing cherry showing severe leaf symptoms in the new tip growth.

temperatures may partially suppress symptoms on some leaves. The amount of pucker, tatter, shot hole, and reduction in leaf size varies with the severity of the virus strain as well as the cultivar of the host involved. There is no dieback or leaf cast. Fruits of severely affected trees are small, late ripening, and lack flavor, but are not misshapen. Diseased trees may produce a marketable crop for many years. A lack of terminal growth and shortening of the internodes cause CMLV-infected trees to appear rosetted.

Transmission

Transmission is readily obtained by budding or grafting any root or shoot tissue from a diseased tree into a susceptible host. Under field conditions, leaf symptoms were found to occur within 32 days after inoculation, whereas symptoms were noted in the greenhouse in 9 to 14 days (553). Graft transmission of CMLV at 26° C. ranged from 0 percent at 66 hours to 100 percent at 106 hours. Viruses with similar speed of transmission are cherry twisted leaf, apricot ring pox, peach wart, and green ring mottle viruses (222). CMLV is not seed transmitted (219, 430).

A minute scale mite, *Eriophyes inaequalis*, which normally lives and feeds on bitter cherry, was found to be an efficient vector of CMLV (L. S. Jones, unpublished data). Single tree infections, widely separated from bitter cherry trees or other diseased sweet cherry trees, indicate that mites can be carried long distances in air currents or by some other means.

CMLV has not been mechanically transmitted or purified. It cannot be eliminated from scion wood by hot-water dip at 50° C. for 20 minutes or hot-air exposure at 37° for 21 days (P. R. Fridlund, personal communication).

Control Measures

In areas where there is a high incidence of cherry mottle leaf, diseased trees, which are no longer producing profitable crops, should be removed and replaced with noninfected trees of a tolerant cultivar. In districts where cherry mottle leaf is uncommon and where susceptible cultivars (Bing and Napoleon) are important, infected trees should be removed immediately to prevent them from being a source of infection for the rest of the trees. Furthermore, tolerant trees used as pollenizers should be indexed; if found to be carrying CMLV they should be rogued.

Remarks

Where cherry mottle leaf is prevalent, it is of considerable economic importance. Fruits of infected Bing and Napoleon trees are reduced both in size and number. The practice of replanting infected Bing orchards with tolerant cultivars (primarily Lambert) to offset this loss is only partially effective because the price of Lambert cherries is usually lower than that of Bings.

CHERRY RASP LEAF

GEORGE NYLAND

Causal Virus

Cherry rasp leaf virus (CRLV).

Synonyms

The disease has been called leaf enation, ruffled leaf, and cockscomb.

History and Distribution

The disease was reported first from Colorado in 1935, and its virus nature was established in 1942 (84). The disease is known to occur in most cherry-growing districts in the West and British Columbia (51, 310, 442, 554). Usually only a few trees are involved at a particular site. In Europe, leaf enation is a prominent symptom of several cherry diseases (74, 167, 173, 380, 381, 480, 532, 591), and a single report from New Zealand describes a rasp leaf disease of cherry (290). Arabis mosaic virus and raspberry ring spot virus, alone or in combination with cherry leaf roll virus, are associated with most of the leaf enation symptoms of the European diseases. Leaf enation in sour cherry or sweet cherry in Europe and North America is also associated with infection by certain strains of *Prunus* ring spot virus (27, 347, 349, 490, 495) and with peach yellow bud mosaic virus (=tomato ring spot virus) (407, 457, 700). CRLV is considered distinct from the other viruses that cause enations on cherry leaves.

Economic Hosts

Rasp leaf has been reported primarily in sweet cherry cultivars and mazzard seedlings but also occurs in Montmorency sour cherry (80). Sweet cherry on mahaleb roots became infected and developed symptoms when buds from diseased Bing were placed in the mahaleb understocks (C. W. Nichols and George Nyland, unpublished data). Mahaleb seedlings became infected when grown in soil that had been obtained near infected orchard trees (700).

Other Hosts

CRLV has been transmitted mechanically to *Chenopodium amaranticolor* and *C. quinoa* and recently to *Gomphrena globosa* (490 and author's unpublished data).

Symptoms

The most characteristic symptom of the disease is prominent enations on the underside of the leaves (fig. 103). The enations take the form of leafy outgrowths or raised protuberances between the lateral veins and along the midrib. The leaves are more or less deformed; many are extremely narrow, folded, and distorted. The upper surfaces of the leaves have a rough, pebbly texture with depressions corresponding to the enations on the lower surface. Some or all of



FIGURE 103.—Napoleon sweet cherry leaves affected by cherry rasp leaf, showing distortion and prominent enations on the lower surface.

the leaves on a shoot or branch may be affected. The virus is essentially systemic in the tree or branch below the highest leaves with symptoms, but sometimes affected spurs may be scattered among healthy ones. Newly infected trees in the orchard usually develop symptoms first on leaves low in the tree. Spread within the tree is slow unless the virus reaches a growing point. Then spread is more rapid, seemingly by cell division in this case. Different isolates may move at different rates, indicating variation in the virus. Many affected spurs and branches in the lower portions of infected trees die, giving the tree an open, bare appearance and reducing fruit production. Trees that are infected at an early age grow poorly and produce few fruits. Sometimes young trees are killed by the virus, especially if they are tissue-inoculated when they are 1 to 2 years old. Mazzard seedlings infected by viruliferous *Xiphenema americanum* or by

implants of infected tissue of *Chenopodium* spp. have a high rate of mortality (George Nyland and S. Lowe, unpublished data).

Symptoms in *C. amaranticolor* and *C. quinoa* consist of a very fine stipple or dusty mottle that often shows only at the base of the leaf and persists for only a few days before fading out. No local lesions or other symptoms are produced on inoculated leaves; the best symptoms are in the partially expanded terminal leaves. If infected plants are cut back, growth from lateral buds may again show symptoms. *C. quinoa* usually shows stronger symptoms than *C. amaranticolor*.

Transmission

CRLV is readily transmitted by tissue grafts, by sap mechanically to a very limited host range, and by the nematode *Xiphinema americanum*. Cherry trees inoculated by bud grafting (80), by placing infected pollen under the bark (700), by approach grafting, or by placing tissue from infected *Chenopodium* spp. under the bark (George Nyland and S. K. Lowe, unpublished data) develop symptoms from 8 to 9 months to 2 or 3 years later. CRLV is retained in soil and infects mahaleb and mazzard seedlings when they are planted in infested soil in containers or in the orchard (700, 727). *X. americanum* was among several plant parasitic nematodes detected in infested orchard soil (700). Handpicked *X. americanum* acquired CRLV from mechanically inoculated *Chenopodium* plants and transmitted it to healthy *Chenopodium* and to mazzard seedlings. *X. diversicaudatum* in a parallel test did not transmit the virus (497).

Characteristics of the Virus

CRLV is a polyhedral, soil-borne, mechanically transmitted, nematode-vector virus of the type referred to by Cadman (104) as NEPO viruses. The particle associated with infectivity is approximately 21 m μ in diameter, with longevity of 10 days in expressed sap at room temperature, and a dilution end point of 10⁻⁴ in *Chenopodium quinoa* sap. The isoelectric point is lower than pH 4.5, and the thermal inactivation point in vitro is about 65° C. for 5 minutes. The virus is fairly stable and is easily concentrated from sap clarified by acidification and butyl alcohol. Yield of virus from *Chenopodium* spp. is low (George Nyland and S. K. Lowe, unpublished data).

Control Measures

Removal of infected trees and trees immediately adjacent to infected trees and deep-soil fumigation with a nematocide are suggested to eradicate the disease in bearing orchards. Disease-free propagating stock should be used for replants or for setting out new orchards.

Remarks

CRLV did not react with antiserum of raspberry ring spot, arabis mosaic, or grapevine fanleaf viruses. Symptoms incited by CRLV in *Chenopodium* most nearly resemble those described for arabis mosaic and fanleaf virus infection in species of *Chenopodium* (George Nyland and S. K. Lowe, unpublished data).

CHERRY TWISTED LEAF

A. J. HANSEN and P. W. CHENEY

Causal Virus

Cherry twisted leaf virus (CTLV).

History and Distribution

Twisted leaf was reported in 1943 from the Okanagan Valley, British Columbia (408), and Yakima County, Wash. (420). It is also known to occur in Chelan County (420) and Stevens County, Wash. (558). It has appeared occasionally in Idaho (A. W. Helton, unpublished data) and at The Dalles, Oreg. (J. A. Milbrath, unpublished data).

Economic Hosts

Sweet cherry is the only known economic host. Bing appears to be the most severely affected cultivar. Other symptom-expressing cultivars are Lamida, Long Stem Bing, and Rainier. Lambert and Napoleon are affected by a severe form of twisted leaf (558). Van and Velvet react variably. Black Tartarian, Deacon, Sam, Star, and Sue, as well as most other cultivars tested, can be symptomless carriers of the virus (415, 416, 558).

Other Hosts

The only other host which has been found naturally infected is western chokecherry (415). Apricot frequently shows ring pox or pit pox symptoms when budded with CTLV-infected material. Almond, Burbank plum (*Prunus salicina*), mahaleb cherry, sour cherry, and peach can be experimentally infected but remain symptomless. Abundance plum (*P. salicina*), Italian Prune (*P. domestica*), myrobalan plum, and bitter cherry are immune to CTLV (413, 558, and P. W. Cheney and C. L. Parish, unpublished data).

Symptoms

The most consistent symptom is an abrupt kink in the midrib or the petiole, which causes the leaf to appear twisted (fig. 104). This is accompanied by an even downward and slightly sideward curl (see smaller leaves in fig. 115, A, p. 244) of the whole leaf. Leaves frequently develop unsymmetrically or stay small. Parts of the midrib and of the lateral veins may become necrotic. In addition, a severe stunt and shortening of the internodes causes the spurs to appear bunched (fig. 105). Both stunt and curling can be noticed from a distance. Once a tree has become infected, the disease spreads rapidly to most or all branches, although scattered spurs may seem to be healthy. Symptoms appear year after year, but their severity in field trees varies. In Washington (554), symptoms of irregular fruit distortion accompanied by pedicel necrosis have occasionally been observed in trees affected by cherry twisted leaf. The available evidence indicates that this distortion is a characteristic induced by certain isolates

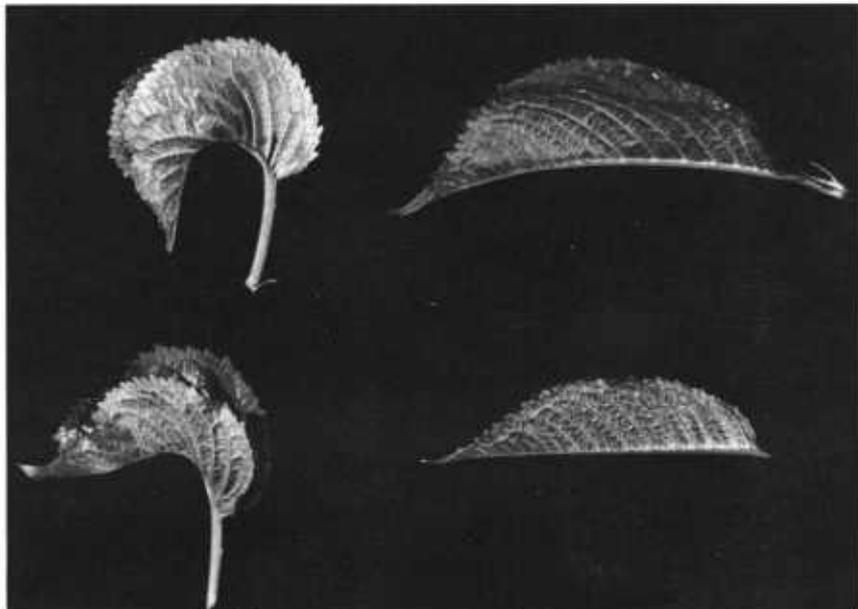


FIGURE 104.—Leaves from healthy (*right*) and cherry twisted leaf-affected (*left*) sweet cherry, showing the typical kink in the midrib.

of the virus, and that it is more severe in older trees of medium vigor (556). For diagnostic purposes, the leaf-twisting reaction of Bing is recognized as the most reliable means of identification. Although the severity of the twisting in Bing varies according to the strain, this cultivar has so far reacted with all isolates. Fruit distortion has been less diagnostic, as it varies more from strain to strain. Moreover, fruit distortion can be caused by other unrelated viruses and probably by physiological factors. Symptoms similar to twisted leaf may be induced by heavy infestation of black cherry aphid (*Myzus cerasi* Fab.). However, these aphid-induced symptoms are characterized by absence of the petiole kink, by the presence of live or dead aphids, and by an uneven distribution of affected spurs in the tree.

Transmission

The mechanism of field transmission of CTLV has not been investigated in detail. Twisted leaf occurs in the orchard at irregular intervals and in unpredictable, scattered patterns. In British Columbia (415, 416), the virus is present in wild chokecherries even in places far removed from cherry orchards. This points to the possibility that it is carried by an unknown vector from a native host or a symptomless carrier into orchards.

CTLV can be transmitted easily from infected cherry to Bing by budding or grafting. Symptoms will appear generally in the first and certainly in the second year after inoculation. Seed transmission of CTLV is not known to occur.



FIGURE 105.—Branch from healthy, *right*, and cherry twisted leaf-affected, *left*, sweet cherry, showing severe bunching of leaves.

Characteristics of the Virus

CTLV has never been transmitted to herbaceous plants, has not been purified directly from *Prunus* spp. tissue, and has not been detected with the electron microscope. Attempts at heat treatment (20 min. at 50° C.; 21 days at 38°) have been unsuccessful (P. R. Fridlund, unpublished data). However, transmissibility and other indications point to a virus as the causal agent of cherry twisted leaf.

Control Measures

At present, only preventive measures can be taken to keep twisted leaf under control. Infected trees should be removed as soon as twisted leaf symptoms appear. Native chokecherry should be eliminated from and around orchard areas wherever feasible. Virus-indexed budwood should be used for all new plantings including pollenizers.

Remarks

Twisted leaf occurs erratically in a very limited geographic area, and is therefore of minor economic importance. It has been locally important in the past where symptomless infected cultivars were budded into susceptible trees as pollenizers (420).

Control depends entirely upon preventive measures, but these have apparently been successful in the last 25 years, as no major outbreaks have occurred. How-

ever, as long as the source of field infections and the hypothetical vector are not known, a potential threat of sudden epiphytotic remains.

Twisted leaf can be confused under field conditions with various other diseases. A fruit distortion similar to that caused by CTLV is induced by tomato bushy stunt virus in Windsor cherry (16). This disease, which is sap transmissible and at present restricted to Ontario, differs from twisted leaf in the absence of the leaf kink, in having a more severe leaf necrosis, and in having a slower advance and less complete distribution of the virus in affected trees. Fruit distortion can also be induced by xylem aberration, a disease occurring occasionally in British Columbia. The short stem disease (8) differs from twisted leaf mostly in that there is an extreme shortening of the peduncle. The spur cherry disease (68) resembles cherry twisted leaf in that the foliage of some cultivars (for example, Sam and Windsor) may be twisted in a similar way. However, the extreme rosetting and the twig epinasty caused by spur cherry disease differ from the symptoms of twisted leaf. Similarities and possible relationships between cherry twisted leaf and apricot ring pox (216, 414, 415), and between these two and the spur cherry disease have been discussed in the section on apricot ring pox.

FRECKLE FRUIT

H. E. WILLIAMS and H. R. CAMERON

This disease of sweet cherry was first observed in 1952 in two Lambert trees growing in an orchard at The Dalles, Oreg. (724). Symptoms were most conspicuous at harvest time. Most of the fruit on the affected trees was undersized, immature, and spotted with necrotic brown flecks (fig. 106). Severely affected fruits were deformed by irregular, shallow depressions; mildly affected fruits were nearly smooth, with most of the brown necrotic area located near the stylar end. No leaf symptoms were observed.

A virus was transmitted by bud-inoculation to Lambert and Bing sweet cherry trees. Fruit symptoms developed in the inoculated trees but were less severe in Lambert. The small affected fruits of Bing and Lambert matured about 2 weeks late. Leaves on the inoculated Bing trees were distinctly mottled. The fruit of inoculated Napoleon sweet cherry trees did not develop fruit symptoms, but the leaves were mottled. Inoculated peach and apricot trees did not develop fruit or leaf symptoms. No natural spread of freckle fruit has been observed, and the disease is of no economic importance.

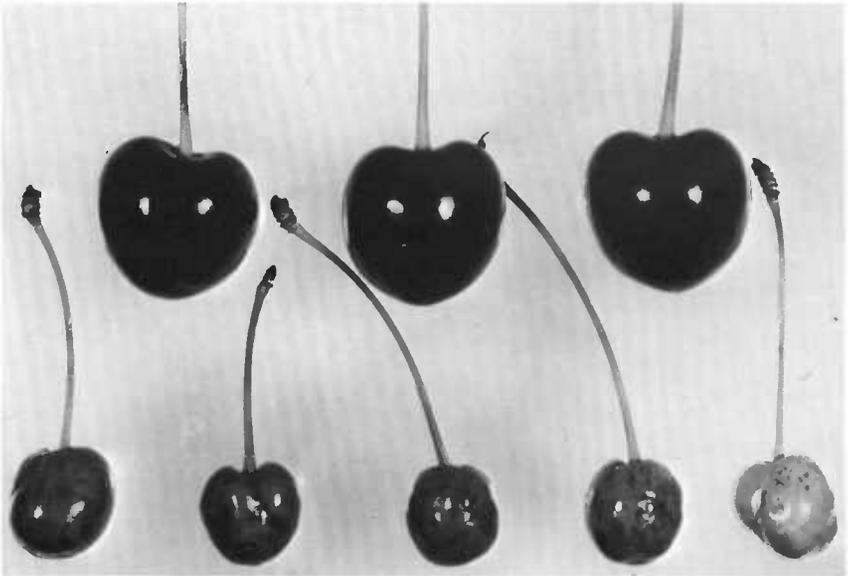


FIGURE 106.—Freckle fruit in Lambert cherry fruit, showing distortion, reduced size, late ripening, and necrotic lesions, *below*; normal fruit, *above*.

FRUIT PITTING OF SWEET CHERRY

T. R. DAVIDSON and WAYNE R. ALLEN

Causal Virus

Tomato bushy stunt virus (TBSV).

Synonyms

Carnation Italian ring spot virus (CIRV), petunia asteroid mosaic virus (PAMV), and pelargonium leaf curl virus (PLCV).

History and Distribution

Infected Windsor sweet cherry trees were first observed in 1962 (16). The virus was isolated in 1963. The disease in sweet cherry is known to occur in only two orchards near Stoney Creek, Ontario. The infected trees, all of the Windsor cultivar 25 to 30 years old, were produced by the same nursery, now defunct. The trees are on mahaleb rootstock, but the source of rootstock and budwood is unknown.

Economic Hosts

Strains of TBSV have been found occurring naturally in the following economically important plants: sweet cherry (16), tomato (618), grape (39), pelargonium (315), petunia, muskmelon and privet (421), primula (462), and apple (W. R. Allen, unpublished data).

Other Hosts

More than 60 other plants have been recorded as hosts of one or more strains of TBSV (16, 315, 408, 420, 421). From a diagnostic point of view, the most important are *Nicotiana tabacum*, *Datura stramonium*, *Chenopodium amaranticolor*, *Petunia hybrida*, *Ocimum basilicum*, *Phaseolus vulgaris*, and *Antirrhinum majus* (16, 315, 421).

Symptoms

The leaves of sweet cherry trees infected with this virus tend to develop in tight clusters. Necrosis of the midvein and some of the lateral veins causes a sideways and downward twisting of the leaf that can be very extreme (fig. 107). Irregular necrotic areas in various parts of the leaves drop out, leaving them with a wind-whipped appearance. The first leaves each year are most severely affected, but symptoms are not confined to these. There is a very low fruit set due to high flower abortion (fig. 108). Flowers that do not set often have very short stems, occasionally twisted by necrosis. The fruit is misshapen by sunken circular pits, and the flesh below these pits is often necrotic (fig. 109). About 95 percent of the seeds in these fruits are aborted.

The twig wood is very brittle. Annual shoot growth is extremely short (fig. 108). Thus, from a distance, the trees appear rosetted.

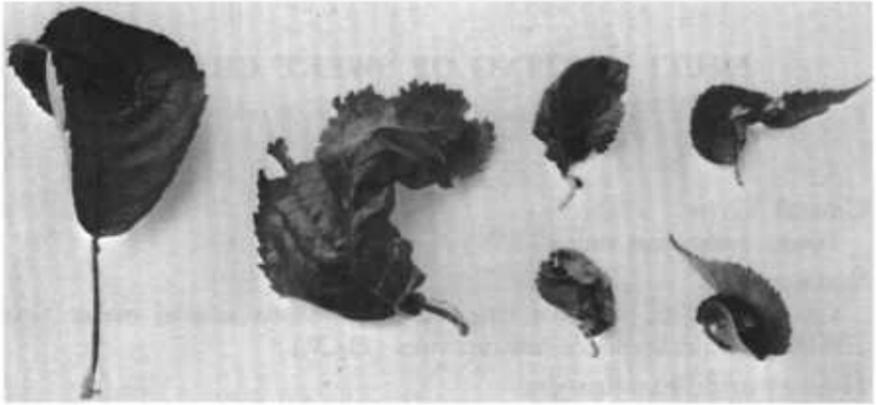


FIGURE 107.—Leaves from Windsor sweet cherry tree systemically infected with tomato bushy stunt virus. Upper leaf surface shows severe twisting and veinal necrosis.



FIGURE 108.—Branch from a Windsor sweet cherry tree systemically infected with tomato bushy stunt virus, showing leaf tufts, leaf twisting, shortened growth, and poor fruit set.

Transmission

TBSV is easily isolated from infected sweet cherry trees by triturating young leaves, petals, anthers, pollen, flesh from pitted areas of deformed fruits or seeds in a mortar with carbamate-phosphate buffer pH 7.0 and then mechanically rubbing the mixture over carborundum-dusted leaves of herbaceous test plants (16). Mechanical inoculation of *Prunus mahaleb* seedlings has resulted

in infection of the inoculated leaf, but the virus has not become systemic. To date, pollen transmission in sweet cherry has been unsuccessful. Also, attempts at soil transmission using *Olpidium* have failed (16, 115, 644). Graft transmission from infected to healthy Windsor sweet cherry trees has been successful, but no natural means of spread is known.

Characteristics of the Virus

The TBSV particles from *Prunus* are more spherical than hexagonal and average 30 m μ in diameter. The sedimentation coefficient in sucrose-density-gradients was determined as 132s. Density-gradient-purified TBSV from *Prunus* has a maximum thermal inactivation point (TIP) at 97° C. The TIP was affected by the virus concentration and the purity of the preparation. The TIP of the virus in raw extracted cucumber sap was 60°. TBSV from *Prunus* was biologically and serologically active between pH 2.0 and 10.3. Antisera to TBSV from *Prunus* cross-reacted with both TBSV and CIRV (15, 16).

Control Measures

This seems to be a very rare disease in sweet cherry. Conclusive evidence of orchard spread is lacking. Therefore, removal of diseased trees will likely eliminate the problem. If virus-indexed budwood and rootstocks are used for propagation of new trees, there is little likelihood of this disease becoming serious.

Remarks

This disease has been found in only two sweet cherry orchards and appears to be confined to the cultivar Windsor. The virus is very readily isolated from trees with symptoms as described, but indexing of symptomless trees, Windsor or other cultivars, has failed to reveal any carriers of the virus. Because no means of natural spread is known and conclusive evidence of orchard spread

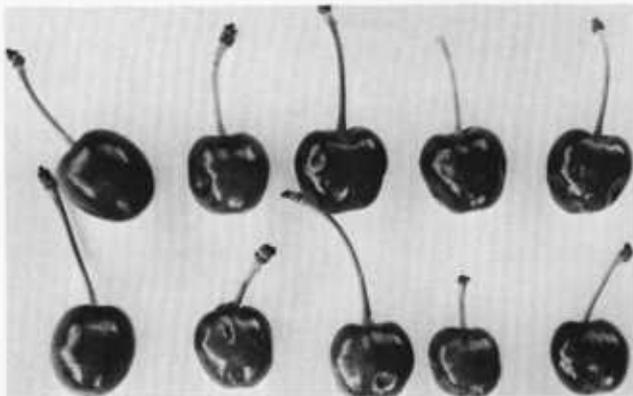


FIGURE 109.—Pitted fruits from a Windsor sweet cherry tree infected with tomato bushy stunt virus, *right*; two normal fruits on *left*.

is lacking, this disease is of minor economic importance. However, the infected trees produce little or no marketable fruit so the loss as related to these trees is considerable. Therefore, the disease is potentially serious.

TBSV from *Prunus* is serologically related to TBSV and, to a lesser degree, to CIRV (16). Other workers have shown that CIRV, PLCV, and PAMV are all serologically related to TBSV and are therefore strains of this virus (316, 421). In Germany, PAMV has been isolated from grapes (39), and a similar strain to TBSV from *Prunus* has been isolated from grapes in Canada (H. F. Dias, unpublished data). More recently an isolate from apple has been found distantly related to TBSV (W. R. Allen, unpublished data).

The symptoms caused by TBSV in sweet cherry are very similar to those described for twisted leaf in western North America (408, 420). However, those working with twisted leaf have been unable to isolate a causal virus on herbaceous hosts.

LITTLE CHERRY

MAURICE F. WELSH and PHILIP W. CHENEY

Causal Virus

Little cherry virus (LCV).

Synonyms

K & S little cherry; sour cherry decline (Oregon).

History and Distribution

Little cherry disease was observed first in 1933, affecting sweet cherry trees in one orchard near Nelson, British Columbia (207). During the ensuing 15 years, almost every cherry tree in the fruit-growing districts of the Kootenay region of southeastern British Columbia became infected. There has been no evidence of spread from this region to other fruit-growing districts of the Pacific Northwest. Bud transmission was effected by Foster in 1941, and more extensively by Lott in 1944 (208).

Reeves, Cheney, and Milbrath (562) demonstrated the common occurrence in Kwanzan and Shiro-fugen (*Prunus serrulata*) of a virus that induces small cherry symptoms in sweet cherry. Subsequent investigations in Washington and British Columbia (557, 561, 719) provided evidence that LCV and K & S virus are indistinguishable in host range, symptoms on known hosts, rates of movement in plant tissues, and ease of bud transmission. *P. serrulata* trees that had been planted near the first infected British Columbia orchard, before the original little cherry outbreak occurred, have been indexed and shown to be infected (719).

Oriental flowering cherries, popular ornamentals in most countries where sweet cherries are grown, were introduced from Japan to Europe in 1822 and to North America about 1845 (561). Reports of the virus indexing of flowering cherries growing in several countries suggest that LCV infects most trees of a number of flowering cherry cultivars in several species wherever these are grown.

Natural occurrence of little cherry disease has been reported in Utah plantings of Burbank Black Giant (688). The virus that causes a decline of Montmorency sour cherry orchards in Polk County, Oreg., when bud-transmitted to sweet cherry cultivars, induces symptoms resembling those of little cherry disease (459).

LCV is believed to occur commonly in sweet cherry in Japan (643). East Malling Research Station workers have reported widespread occurrence of LCV in English orchards, and have detected its presence in sweet cherry cultivars newly introduced to England from France, Italy, and Sweden (534, 535, 540). Its detection has been reported in a number of cultivars in Switzerland (601), and its presence in sour cherry is suspected in Poland (25). Transmission of

LCV from sweet to sour cherry has been reported in Denmark (382). There are unpublished reports of its occurrence in other European countries. In the State of Victoria, Australia, LCV infects a number of cherry cultivars, but spread is not apparent (P. R. Smith, personal communication).

Economic Hosts

All tested sweet cherry cultivars are susceptible, although they vary considerably in type and severity of reaction. In British Columbia, LCV was bud-transmitted from sweet cherry to Montmorency sour cherry and returned to sweet cherry, but there was no such passage of LCV through peach, apricot, and prune (718, 722). In Washington, the virus was passed through numerous duke and sour cherry cultivars but not through peach, apricot, prune, plum, almond, and apple. All tested duke and sour cherry cultivars display fruit symptoms.

Other Hosts

Some indexed bushes of wild bitter cherry, *Prunus emarginata* var. *mollis*, growing near diseased orchards in British Columbia proved to be infected with LCV; other seedlings of this wild cherry were not systemically infected after bud inoculation (722). In Washington, seedlings of *P. mabaleb*, *P. emarginata*, and *P. tomentosa* were shown to be tolerant hosts, and *P. pensylvanica* displayed small fruit symptoms (author's unpublished data and P. R. Fridlund, personal communication). The chokecherries *P. virginiana*, *P. virginiana* var. *demissa*, and *P. virginiana* var. *melanocarpa* have not become infected when bud-inoculated (560, 718).

Indexing at Wenatchee, Wash., of flowering cherry cultivars growing in the United States and Canada (561), has provided positive tests for the following: *P. serrulata*, 25 of 34 cultivars; *P. sieboldii*, 1 of 1; *P. subhirtella*, 2 of 6; *P. yedoensis*, 1 of 3. Negative tests have been given by one cultivar each of *P. sargentii*, *P. incisa*, and *P. nipponica*. Similar indexing in England (114, 535 and A. F. Posnette, personal communication) has detected presence of LCV in 17 cultivars of *P. serrulata*; two cultivars each of *P. subhirtella*, *P. yedoensis*; and one each of *P. fontanesiana*, *P. avium* cv. *Flore-pleno*, *P. incisa*; but not in two tested cultivars of *P. serrulata* and *P. subhirtella* and one each of *P. cerasus* var. *rhexii* cv. *Flore-pleno* and *P. lannesiana*. The virus has been detected by indexing in 13 flowering cherry cultivars in Japan (643).

Symptoms

Symptoms have been seen in sweet, sour, and duke cherries. Those on the fruit are the most apparent and economically important, but foliage symptoms are displayed by many cultivars; tree vigor is usually impaired when trees have been infected for a few years.

Fruit symptoms on Lambert, the cultivar most extensively planted in the region of disease occurrence in British Columbia, are fairly consistently displayed and can usually be diagnosed with reasonable assurance. The fruits develop normally through the early part of the season but never fully ripen.

At picking time, the cherries are one-half to two-thirds normal size, dull red, angular, and pointed, usually with three flat sides tapering toward the distal end (fig. 110). In the season following infection, the fruits are usually light to medium pink and even smaller than in subsequent years. In all seasons, they may hang on the trees for weeks without ripening fully.

On most other cherry cultivars, the symptoms resemble those on Lambert, but are less severe and more variable. On Bing, the fruits are severely affected in the season after infection, but in subsequent years they may vary from small and medium red to large and black and from irregular to normal in shape. On cultivars with firm, broad-shouldered fruits, such as Van, the color and shape are altered little, but the size and sweetness are reduced. On most white cherries, such as Napoleon, the symptoms are characteristically milder than on black-fruited cultivars. Montmorency fruits usually vary from normal size and color to small and light-colored with little change in shape.

Fruit symptoms vary considerably in severity for all cultivars from season to season, from district to district, and from orchard to orchard. Zinc deficiency has been shown to accentuate their severity in many Kootenay orchards (720). Limbs of affected trees that are severely shaded bear normal fruits, and fruits on nonfoliated portions of branches are usually normal (716).

For all tested cultivars, the symptoms are comparable whether trees are grown on mazzard or mahaleb rootstocks (560, 718).

Foliage symptoms are displayed by many cultivars. On diseased Lambert

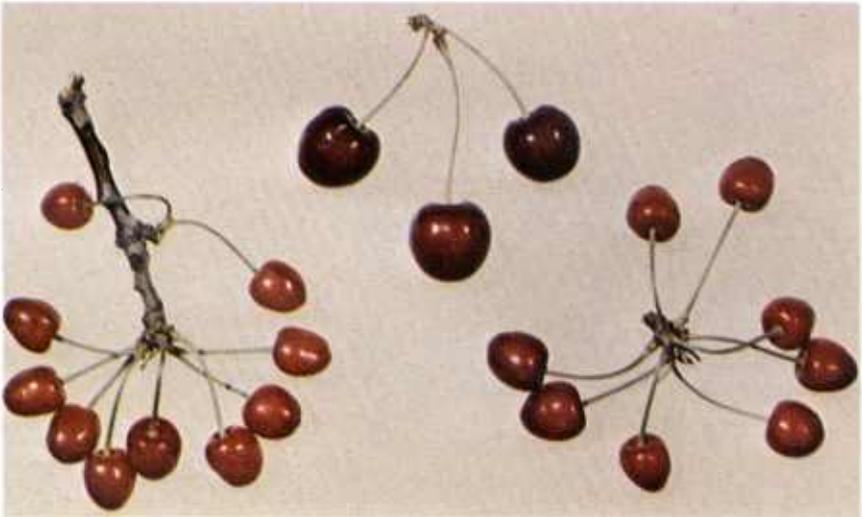


FIGURE 110.—Lambert cherry fruit affected by little cherry, *left* and *right*, showing smaller size, duller color, and angular and pointed shape, compared with normal Lambert fruit, *center*.

trees, the fruit crop is readily visible at picking season—less hidden in the leaf canopy than on normal trees. Careful examination indicates that most leaves have slight marginal upcurl. On a number of other cultivars, this foliage upcurling is more apparent and is accompanied by marked reddening or bronzing of the leaf surfaces. The coloration may appear during summer months when there is an interval of cool nights and sunny days. It is usually most apparent in September and October.

Often the coloration is apparent first on the under surface of the uprolled margins of the leaves, but characteristically it develops most strongly in the interveinal areas of the upper surface of the leaves; the tissues bordering the midrib and main veins retain their normal color (fig. 111). Usually symptoms appear first on the basal leaves of the current season's growth.

Foliage reddening is mild to moderate in Bing. Cultivars that are recorded as displaying strong coloration and that therefore have had frequent use as indicators for LCV include Sam, Deacon, Mazzard F12/1, Star, and Van (534, 560, 721).

Observations in orchards and test plots suggest that trees infected with LCV suffer gradual loss of vigor. Cultivars that display severe foliage symptoms decline most rapidly. In a LCV tolerance breeding program in British Columbia, several of the seedlings which were selected for expanded trials because they produced desirable fruit in the several seasons following inoculation, have



FIGURE 111.—Star cherry leaves affected by little cherry, showing red coloration in the areas between the main veins and the retention of green along the midrib and larger veins. Leaf on *right* is normal.

displayed severe foliage upcurling. Subsequently, the foliage has become sparse, vigor has declined, and fruit size has diminished.

Transmission

The remarkably rapid natural spread of LCV in British Columbia between 1933 and 1950 (208) suggested transmission by an agent that was abundant and efficient. Natural spread has been recorded in experimental plantings of sweet cherry in Washington (561) and Oregon (458). Surveys indicate that natural spread has occurred in commercial plantings in England (535, 540).

In transmission tests at Creston, British Columbia (717), 18 orchard trees and seven trees under screen displayed little cherry symptoms after caging of *Macrosteles fascifrons* (Stal.) on the foliage. Similar caging of *Scaphytopius acutus* (Say) and *Psammotettix lividellus* (Zett.) gave symptoms in one tree for each species. Proportions of successful transmissions were low for all three species. All tests were performed after 1950 when the rate of natural spread is believed to have dropped to a low level (723), so there is justification for doubting whether any of the three leafhopper species is the vector responsible for the original rapid spread of LCV in the Kootenay region. Transmission was not effected by similar tests with 21 other leafhopper species in 11 tribes of seven subfamilies; with nine species of insects in other families and with six species of mites.

There is considerable evidence that infected flowering cherries can be maintained adjacent to sweet cherries for long periods without natural spread of LCV to sweet cherry (561, 688), although once the virus is transmitted to sweet cherry by budding there is spread to other sweet cherries. It seems possible that all spread from flowering cherry to sweet cherry can be attributed to natural root grafting or to vector transmission from suckers that frequently arise from mazzard rootstocks on which flowering cherry trees are grown.

Bud transmission is usually 100 percent successful when buds are taken from any part of a tree that has been infected for 2 years or longer. Characteristically, fruit and foliage symptoms appear on inoculated limbs within 1 year of bud inoculation, and throughout the tree within 2 years. However, LCV transmission requires a relatively long period of graft union contact and falls into Fridlund's Rate 1 group (222). This suggests that LCV may be phloem-limited in tree tissues.

Establishment of dodder (*Cuscuta subinclusa* Dur. & Hilg) on LCV-infected flowering cherry and on *Vinca rosea* has induced symptoms on the latter that include stunting, diminution of flower size, chlorosis, and slight proliferation (R. M. Gilmer, personal communication).

There have been no reports of success in juice transmission of LCV.

Control Measures

In British Columbia, two approaches have been made to control of little cherry: prevention of its establishment in parts of the province where it does not

occur, and development of resistant varieties to allow reestablishment of a cherry industry in the Kootenay region.

Under the British Columbia Plant Protection Act (1954), a Little Cherry Control Area was established, embracing the province's principal disease-free cherry-growing districts. Preventive measures included the prohibition of all movement of fruit trees and fresh fruits from the Kootenay region to the Control Area and enforced removal of affected cherry trees that might be found in surveys of the Control Area. Subsequently, these measures were complemented by compulsory eradication of all ornamental flowering cherry trees within the Control Area and prohibition of their further planting. So far, the disease has not become established within the Control Area.

Among hundreds of sweet cherry cultivars tested by various workers, all have become infected when bud-inoculated with LCV. In British Columbia, a tolerance breeding program has been attempted. Thousands of seedlings of the more tolerant commercial cultivars have been inoculated and grown within the region of disease occurrence. Several have shown promise during the first three seasons of fruiting. A sweet cherry of Lambert type, found within the Kootenay region, does not appear to become infected by natural spread, although it becomes severely affected when bud-inoculated.

LCV has been inactivated in buds of several flowering cherry and sweet cherry cultivars by low-intensity, relatively low temperature, long exposure heat therapy (37.5° C. for 3 to 4 weeks (499); 37° for 21 days (P. R. Fridlund, personal communication); or 35° for 14, 21, and 25 days (A. F. Posnette and R. Cropley, personal communication). Several cultivars of flowering cherry, thus rendered free from LCV, have been made available to nurserymen. LCV has not been inactivated in sweet cherry by dipping budsticks for 20 minutes in hot water at 50° (P. R. Fridlund, personal communication).

Remarks

In England, (A. F. Posnette and R. Cropley, personal communication), most isolates from sweet cherry plantings give milder symptoms on standard indicators than those induced by isolates from flowering cherry. Symptoms of widely differing severity have been obtained during indexing at Wenatchee, Wash., of flowering cherry collections from various parts of United States and Canada and in the indexing of sweet and sour cherry in Oregon (722). This has been interpreted as evidence of strain differences among LCV isolates.

X-disease virus in sweet cherry has been reported as "little cherry" in a number of publications. The only obvious similarity between the two viruses is in the symptoms that they induce in sweet cherry growing on mazzard rootstocks. The many differences that have been noted (560, 718) include symptoms induced in sweet cherry on mahaleb rootstocks, ease of bud transmission, interval between inoculation and appearance of symptoms, differences in foliage

symptoms on sweet cherry, ability of X-disease virus to infect chokecherry, *P. mahaleb*, and peach, (none of which become infected with LCV), demonstrated occurrence of LCV in *P. emarginata* and its varieties (not hosts of X-disease virus), and vector relationships to the extent that they are known.

CUCUMBER MOSAIC VIRUS INFECTION IN *PRUNUS*

J. H. TREMAINE

Cucumber mosaic virus (CMV) and *Prunus* necrotic ring spot virus (PRSV) were transmitted together to cucumber seedlings by mechanical inoculation with leaves from a Montmorency sour cherry tree showing green ring mottle symptoms and with leaves from a Black Tartarian sweet cherry tree showing tatter leaf symptoms (713, 736). After several transfers in cucumber plants, the viruses were separated by sap transmission to tobacco seedlings, which separated CMV and showed primary necrotic etching, systemic chlorotic rings, and vein-banding patterns (fig. 112). Sap inoculation of cucumber seedlings with extracts of infected tobacco leaves induced chlorotic and necrotic spots on the inoculated leaves and a systemic mosaic pattern (fig. 113). The cucumber seedlings sometimes appeared to recover. The thermal inactivation point of CMV from *Prunus* was between 65° and 70° C. and the dilution end point between 10^{-3} and 10^{-4} . The isolate of CMV from sour cherry was purified from infected cucumber plants and had a sedimentation coefficient of 100 s (667). The CMV isolates from both sour cherry and sweet cherry reacted with antiserum of cucumber mosaic virus strain Y (667.)

The isolation of CMV from *Prunus* on herbaceous hosts was not always successful. However, its frequent isolation from peach (*P. persica* L.) seedlings bud-inoculated from the two original sources and failure of isolation from other trees is good evidence that CMV was not a contaminant. The symptoms induced by CMV alone in *Prunus* hosts are not known. Peach and sour cherry seedlings infected with PRSV and CMV together did not show symptoms differing from similar plants infected with PRSV only. CMV is probably not involved in the production of the green ring mottle or tatter leaf symptoms observed in

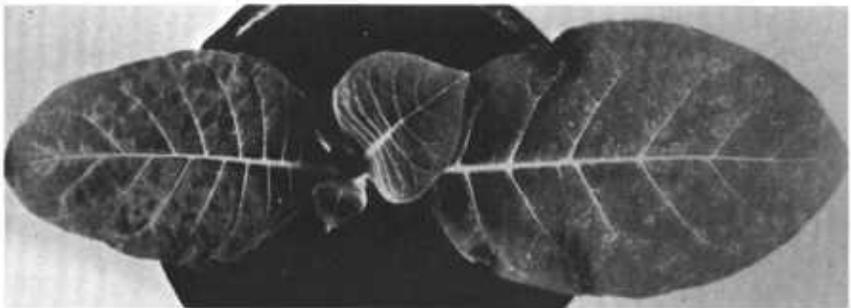


FIGURE 112.—Primary necrotic flecking and systemic chlorotic ring- and vein-banding patterns in tobacco infected with cucumber mosaic virus (CMV) from *Prunus*.

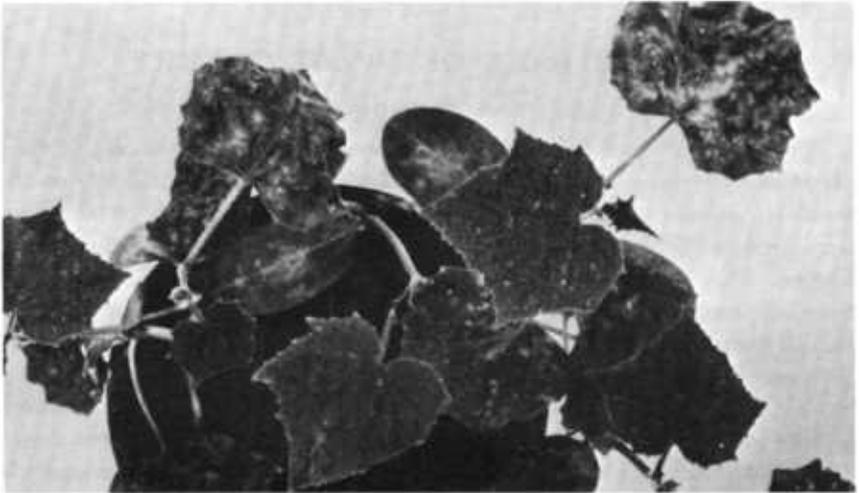


FIGURE 113.—Cucumber inoculated with cucumber mosaic virus (CMV) from *Prunus*, showing necrotic and chlorotic spots on inoculated leaves and typical mosaic in systemically infected leaves.

the original sources, because CMV was not isolated from other trees showing these symptoms.

CMV from *Prunus* was transmitted from cucumber to tobacco and back to cucumber by the aphids *Aphis gossypii* and *Myzus persicae*. However, natural spread from infected peach seedlings to adjoining peach and sour cherry trees was not observed in tests over a 10-year period.

ROUGH BARK OF SWEET CHERRY

CARL W. NICHOLS

Rough bark of sweet cherry (*Prunus avium*) has been reported only in California near the community of Valyermo in Los Angeles County and only on the Lambert cultivar (484). The symptoms on sweet cherry resemble certain symptoms produced by the necrotic rusty mottle (580, 632) and black canker (774) viruses. They consist of severely roughened bark on the trunk and scaffold branches from which masses of gum exude (fig. 114, A). They differ from the rough bark and gumming symptoms that occur on the trunks and scaffold branches of Burbank cherry trees (fig. 114, C) wherever this cultivar is grown in California. The symptoms on Burbank are more severe, are always associated with enlarged nodes on branches 7 years old or older, and have not been shown to be caused by a graft-transmissible entity. Apparently, this latter disorder is the rough bark disease of cherry reported in California by Thomas (647, 648). Leaf symptoms typical of those produced by the *Prunus* ring spot virus (152) are the only ones that have been observed on the infected Lambert cherry trees.

Rough bark and gumming symptoms identical to those seen on Lambert cherry trees in the Valyermo area were produced by bud inoculation on Lambert and on trees of a mazzard seedling clone that resembled Black Tartarian (fig. 115, B and D) but not on Napoleon and Bing. Trees inoculated in the fall produced the first bark symptoms the second summer after inoculation. These symptoms were discrete, raised bark blisters a few centimeters across, which were similar to those of cherry bark blister (necrotic rusty mottle) in California (632). These blisters appeared to coalesce and rupture, forming rough areas from which masses of gum exuded. The rough areas with exuding gum continued to develop until, in the third summer after inoculation, they covered large portions of the trunk and scaffold branches of the inoculated trees. During the first and subsequent dormant seasons after bark symptoms appeared, the nonroughened bark on the trunks of the inoculated trees had a dark, shiny appearance in comparison with the uninoculated checks (fig. 115, D).

Typical *Prunus* ring spot leaf symptoms (152) were produced by inoculation from the Valyermo source on Guame peach; Bing, Lambert, and Napoleon sweet cherry; and the mazzard seedling clone. A positive ring spot reaction, gumming and bark necrosis (462), was produced on Shiro-fugen flowering cherry. This reaction suggests that either there was more than one virus present in the infected Valyermo trees or the rough bark virus is related to *Prunus* ring spot virus.



FIGURE 114.—*A*, Lambert sweet cherry tree near Valyermo, Calif., with rough bark and gumming (*arrows*) symptoms of cherry rough bark; *B*, mazzard seedling clone with rough bark on trunk during third summer after inoculation with cherry rough bark virus; *D*, same tree as in *B* showing gumming (*arrows*) and dark shiny appearance of trunk during third dormant season after inoculation. *C*, Burbank sweet cherry tree with nontransmissible big node and canker symptoms similar to those caused by rough bark virus.

RUSTY MOTTLE GROUP

BRYCE N. WADLEY and GEORGE NYLAND

Causal Viruses

Rusty mottle virus (CRMV), mild rusty mottle virus (MRMV), necrotic rusty mottle virus (NRMV), and Lambert mottle virus (LMV) incite the diseases included in the rusty mottle group. Rusty mottle has also been called severe rusty mottle to distinguish it from mild rusty mottle (772). CRMV and MRMV are considered to be closely related, based on certain similarities in the diseases they cause in cherry. Both rusty mottle and mild rusty mottle originally were included in the description of rusty mottle (569). NRMV and LMV are considered to be closely related, based on certain similarities in the diseases they cause. Because some characteristics are common to all four diseases, they are included here in a rusty mottle group under two names — rusty mottle and necrotic rusty mottle. Cherry bark blister is a synonym for necrotic rusty mottle.

History and Distribution

Rusty mottle was reported by Reeves in Washington in 1940 as a new virosis of cherry that had been repeatedly transmitted during the previous 5 years by grafting (552). In 1947, Zeller and Milbrath (770) distinguished mild rusty mottle from the Washington disease complex. In 1945, a rusty mottle disease was described in Utah, which is now recognized as necrotic rusty mottle (574, 576, 582). Later, Reeves and Richards (569) described differences in the types of rusty mottle found in Utah and Washington, but reached no conclusions as to their relationship. Lambert mottle, which appears to be related to necrotic rusty mottle, was reported from British Columbia in 1945 (409) and Montana in 1951 (11). Cherry blister bark, now recognized as necrotic rusty mottle, was described in 1949 in California (632).

The various diseases of the rusty mottle group are distributed in sweet cherry-growing areas in Washington, Oregon, Idaho, Montana, Utah, California, and British Columbia. The rusty mottle disease as described in Washington and designated subsequently as severe rusty mottle (772) is not known to occur in California or Utah. Necrotic rusty mottle occurs where cherries are grown in Western United States and was reported in Great Britain (539).

Host Range

All sweet cherry cultivars tested, mazzard and mahaleb seedlings, Manchu cherry, peach, apricot, damson plum, and sour cherry are susceptible to the rusty mottle viruses, although symptoms may not be evident in some.

Necrotic rusty mottle is most severe in Lambert, Seneca, Sam, Hudson, and Bing, and moderate in Napoleon, Black Republican, Van, Windsor, Lyons, Macmar, Chinook, and Rainier (580, 682, 685). Cultivars in which symptoms

may be very mild or masked are Black Tartarian, Burbank, Orb, Schmidt, Napa Long Stem Bing, Deacon, Cardofer Fruhe, and Dicke Braune Blankenburger. In Great Britain, Frogmore, Noble, Lambert, and Bing are damaged (539).

LMV caused symptoms only in Lambert, Seneca, and Starking Gold Giant cherries of 10 cultivars tested (465) and could not be recovered from inoculated trees of Shiro-fugen flowering cherry, Montmorency sour cherry, chokecherry, Italian Prune, apricot, plum, *P. emarginata* var. *mollis*, and four cultivars of apple and one of pear (418, 465, 685).

Rusty mottle and mild rusty mottle were equally severe on the sweet and sour cherry cultivars tested and also infected peach, apricot, damson plum, some flowering cherry cultivars, and *P. mahaleb*, *P. virginiana*, *P. tomentosa*, and *P. serotina* (7, 684, 770).

Symptoms

Necrotic rusty mottle.—Symptoms in sweet cherry vary considerably with the cultivar, strain of the virus, and the weather during the early growing season. Symptoms appear earlier and are more severe during a cold, late spring. The first leaf symptoms appear 3 to 6 weeks after full bloom. With Lambert mottle, the buds on upper portions of the shoot fail to open or die soon after opening (411). Angular necrotic spots form in the leaves. The necrotic areas may be limited to discrete spots, or they may involve large areas of the leaves (fig. 115). Leaves that are more than 50 percent necrotic usually are cast, whereas those that remain develop the characteristic senescent yellow mottle with green islands or bands 2 to 3 weeks before harvest (580). The leaf symptoms at this stage are very similar to those of rusty mottle or mild rusty mottle. Other leaves with scattered angular necrotic spots may develop shot hole as the necrotic areas fall out, but the leaves remain on the trees. Leaf symptoms usually are inconspicuous during the summer and early fall in areas with continuous high temperatures. Autumnal senescence of the leaves occurs somewhat earlier in diseased than in healthy trees. Striking leaf patterns of prominent green rings and lines on a background of yellow, brown, or brilliant red are produced following the onset of cool fall temperatures (fig. 116).

As the disease develops in the tree, part of the buds and leaf spurs may be killed, resulting in bare, rangy branches with terminal tufts of foliage (fig. 117). In more advanced stages of the disease, the older branches of the tree are killed and water sprouts develop from the lower portion.

Bark symptoms consist of shallow, necrotic areas, discrete shallow gum blisters, or deep gum pockets that originate in the wood. Both the virus isolate and the host cultivar influence the type and degree of bark symptoms. Bark blisters and deep gum pockets are common in Bing, but general bark necrosis is common in Lambert.

The brown, angular, necrotic spots, rusty chlorotic areas, shot holes of the leaves, and the blisters, gum pockets, and general necrosis of the bark constitute the most important diagnostic symptoms of the disease (580, 684). Infected



FIGURE 115.—*Top*, Leaves from Bing cherry, *below row*, inoculated with necrotic rusty mottle virus; small Bing cherry leaves, *above row*, have twisted leaf symptoms. *B*, Leaves and fruit of Napoleon cherry affected by Lambert mottle.

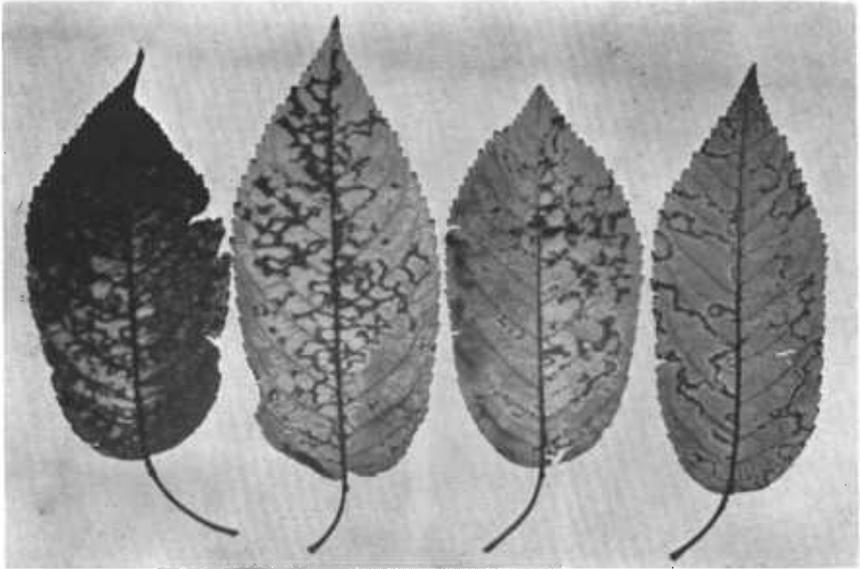


FIGURE 116.—Senescent leaves in late fall from Lambert cherry tree with necrotic rusty mottle, showing characteristic rings and line patterns.

trees are killed more quickly in areas with severe winters than in areas with relatively mild ones. Lambert, Sam, Seneca, and Hudson often fail to survive the second season after infection in Utah.

Rusty mottle and mild rusty mottle.—The first foliage symptoms in cherry appear 4 to 5 weeks after full bloom (552). The small basal leaves are the first to show chlorotic mottling. The chlorotic areas remain light green, and the remainder of the leaf rapidly develops late-season coloring of bright yellow, brown, or red (fig. 118). The affected leaves soon abscise. Thereafter, variable chlorotic mottling of most of the foliage takes place (555). Symptoms of mild rusty mottle do not change much after initial leaf fall and appearance of mottling except for some bronzing of the leaves through the summer (fig. 119) (772). With rusty mottle, many of the older leaves rapidly develop autumnal colors and subsequently drop, resulting in the loss of 30 to 70 percent of the leaves by the time fruits are mature (552). Fruits are smaller, have insipid flavor, and ripen late. Infected trees decline, and the main limbs die back. Trees affected by mild rusty mottle decline more slowly than those affected by rusty mottle (770). Foliage symptoms occur in peach trees inoculated with buds from infected cherry trees and are not unlike those produced in cherry (444, 552).

Montmorency sour cherry trees in the field from which MRMV was recovered in Oregon (444, 772) and trees in Oregon and California which were inoculated with MRMV from sweet cherry had leaves that were smaller than normal,

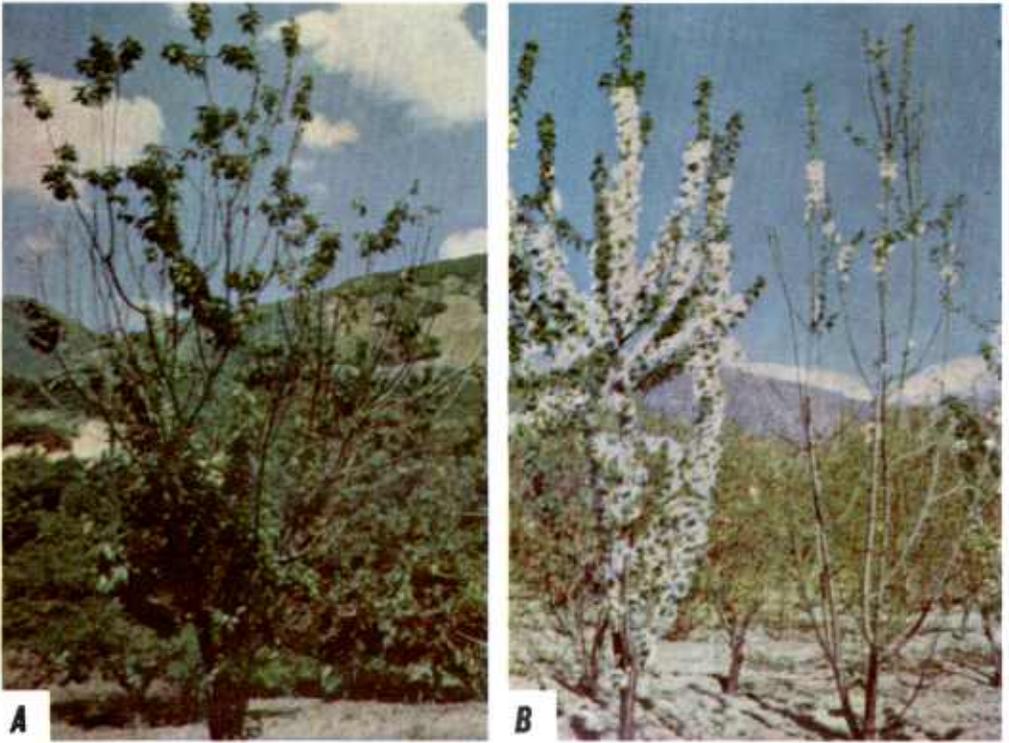


FIGURE 117.—*A*, Fourteen-year-old Lambert cherry tree showing bare, rangy limbs and tufted terminals characteristic of advanced stage of necrotic rusty mottle; *B*, *right*, young Lambert tree inoculated with buds from tree in *A* and showing retarded leafing and blossoming compared with uninoculated tree, *left*.

showed mild mottling and rustiness, and, later, necrotic areas. Some affected leaves were cast.

No distinctive symptoms have been observed in apricot, Italian Prune, damson plum, or chokecherry. Occasionally, Kwanzan and Naden flowering cherry showed slight leaf symptoms (417, 772).

Transmission

The rusty mottle group viruses are easily transmitted through tissue grafts to susceptible host plants. They have not been transmitted mechanically in expressed juice. The incubation period is usually less than 1 year but may be as much as 3 years. Sometimes, only part of a tree will show symptoms the first season after infection, but by the second season the virus will be distributed throughout the tree. Trees inoculated in August will develop symptoms the following spring. Trees inoculated as the buds are breaking dormancy often develop symptoms within 30 days (6, 555, 682, 770). Natural spread of rusty

mottle disease is slow in sweet cherry orchards and often occurs only to nearby trees. Most of the disease in orchards results from propagation of infected trees by nurseries. Mild or symptomless cultivars often play an important part in perpetuation and spread (9, 411, 685).

Characteristics of the Viruses

NRMV was inactivated in Lambert cherry budsticks treated in a hot-water bath at 50° C. for 10 or 13 minutes or at 52° for 5 minutes (487), but MRMV was not inactivated by hot air in 4 weeks at 38° (George Hyland's unpublished data). The virus nature of this group of diseases is assumed, but the easy cure by heat of necrotic rusty mottle is similar to some diseases of suspected mycoplasma nature. The viruses have not been purified, so nothing is known of their physical or chemical properties.

Control Measures

Planting of virus-free nursery trees should give economic control of this disease. Diseased trees should be removed as they are discovered to prevent natural spread. Several cultivars commonly used to pollinate major cultivars of sweet cherries may be symptomless hosts and have transmitted NRMV when infected buds were grafted to sensitive varieties (682). Symptomless cultivars should be indexed before budwood is taken for propagation.

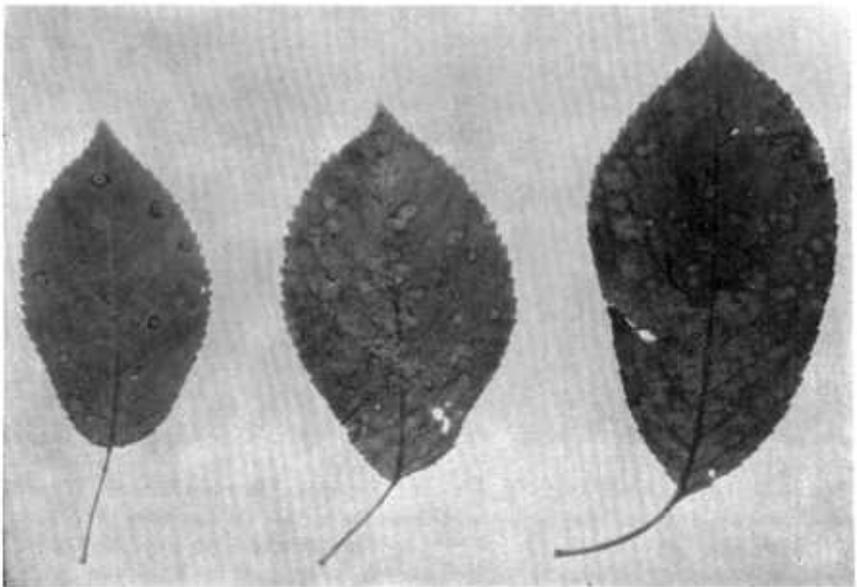


FIGURE 118.—Bing cherry leaves affected by mild rusty mottle, showing chlorotic rings and mottle.

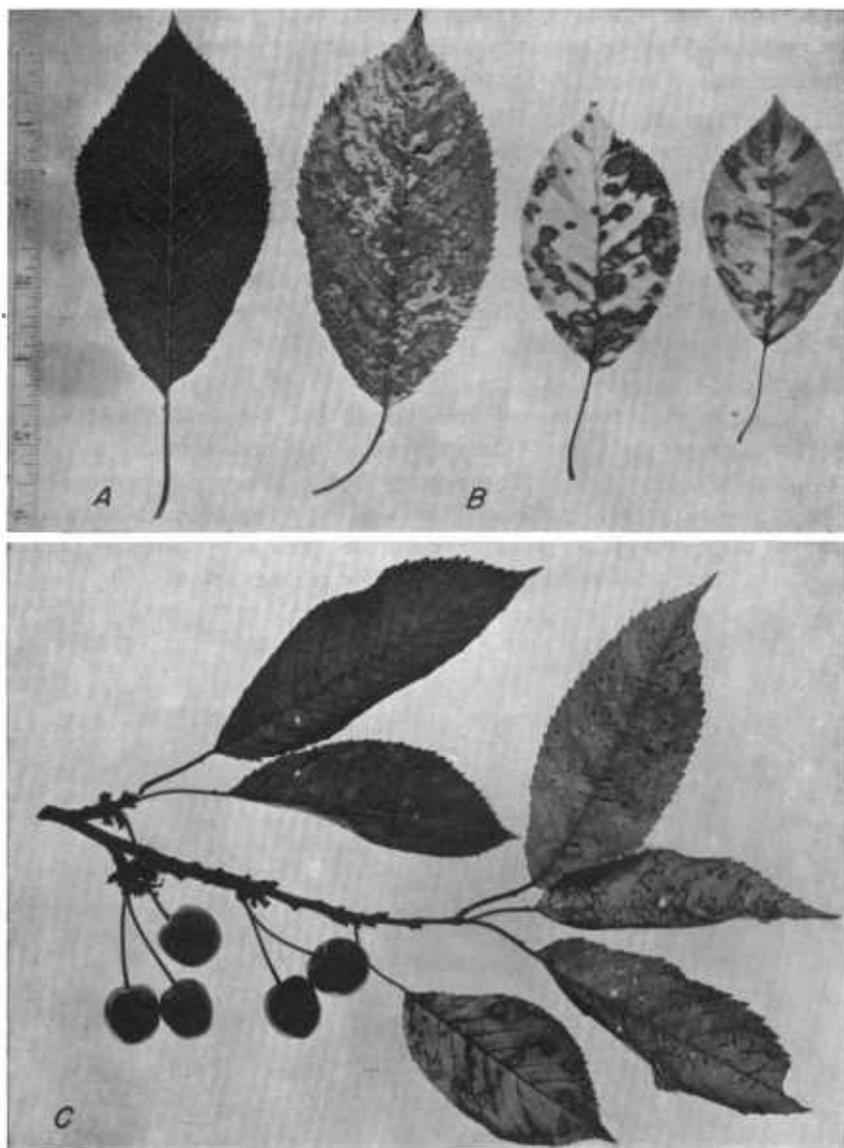


FIGURE 119.—*A*, Normal leaf of Bing cherry; *B*, comparable leaves affected by rusty mottle, showing various patterns that develop on leaves that are cast prior to harvest; *C*, branch from a Bing cherry tree affected by rusty mottle, 1 week before harvest, showing that the fruits are not malformed but that the leaf cast has been heavy.

Remarks

Evidence that necrotic rusty mottle is quite different from mild rusty mottle is provided by the difference in heat stability of the viruses. NRMV was inactivated by relatively short hot-water treatments, but MRMV was not inactivated when infected cherry trees were heat-treated for 4 weeks in air at 38° C. Evidence that NRMV and MRMV may be related is provided by similarity of symptoms in cherry and because mild rusty mottle interferes with or partially protects against necrotic rusty mottle (682, and authors' unpublished data).

SHORT STEM

C. L. PARISH and PHILIP W. CHENEY

Causal Virus

Short stem virus (SSV).

History and Distribution

Short stem was first discovered at Flathead Lake, Mont., in 1958 (4, 5, 8, 10). It was later found at Hood River, Oreg., in 1962, although it was not identified as such until 1964.

Economic Hosts

All sweet cherry cultivars tested.

Other Hosts

The virus when introduced by bud inoculation affected sweet cherry, Abundance plum (*Prunus salicina*), and seedlings of *P. maackii* and *P. davidiana* which exhibited symptoms resembling those of twisted leaf. Drake almond, Riland and Tilton apricot, Elberta and J. H. Hale peach, and seedlings of *P. besseyi*, *P. capuli*, *P. pensylvanica*, and *P. serotina* were symptomless carriers of SSV.

Indexing of the following plants inoculated with SSV failed to reveal the presence of the virus: Golden Delicious apple, *Malus platycarpa*, Russian apple seedling (R12740-7A), *Prunus andersonii*, *P. virginiana*, *P. tomentosa*, Chilcott and Fiebing plums (*P. americana*), Italian Prune and Tragedy prune (*P. domestica*), Burbank plum (*P. salicina*), Shiro plum (hybrid), and Bartlett pear.

Symptoms

Symptoms of the short stem disease occur on leaves, fruit, and fruit stems. Constriction of the veins or midrib causes part, or all, of the leaf to tip or roll downward, resembling aphid injury. The twisting and rolling of the petiole or blade first appears in April or May on the spur leaves. Later, some leaves on the current season growth may become affected. This malformation is permanent, and affected leaves are retained by the tree until the time of normal abscission. Fruit symptoms usually occur as shock symptoms on a small percentage of cherries on newly infected trees. Rough misshapen fruits can usually be observed the first and second year following infection. In some instances, color break may also occur on the misshapen cherries, resulting in dappled, bumpy fruits. As the name of the disease indicates, shortened fruit stems are the most characteristic symptom (fig. 120). The shortened stems usually do not occur to an appreciable extent until the second year following infection; this symptom develops more slowly throughout the tree. The fruit stems vary from nearly normal length to as short as one-quarter inch in length. At this stage, the shock



FIGURE 120.—A branch from a short stem-infected Lambert cherry tree showing twisted leaves and both long and short fruit stems on the same branch.

symptoms of bumpy, dappled fruit no longer occur; the only symptoms are twisted leaves and short fruit stems.

Transmission

The virus can be transmitted easily by tissue grafts. It spreads naturally in the field, but the vector is not known.

Control Measures

It is strongly recommended that infected trees be removed so they will not serve as sources of inoculum. Virus-free propagating materials should be used for replanting.

Remarks

Symptoms incited by SSV resemble those incited by tomato bushy stunt virus in sweet cherry in Ontario (16). However, attempts to isolate tomato bushy stunt virus from short stem infected trees have been negative (authors' unpublished data).

SPUR CHERRY

EARLE C. BLODGETT and MURIT D. AICHELE

Causal Virus

Spur cherry virus (SCV).

Synonyms

Plath Bing; spur Bing.

History and Distribution

An 8-year-old Bing cherry tree (*Prunus avium*), thought to be a mutant, was discovered in an orchard near Yakima, Wash., in 1962. The tree was producing a good crop of what appeared to be normal Bing fruits on a compact or spur-type growth. Indexing on Bing showed that this type growth was caused by a previously undescribed virus (68).

Natural occurrence has been limited to the original tree. Nursery stock has been propagated by several nurserymen and sold as dwarf Bing cherry trees and budwood has been distributed for experimental purposes to other areas and States.

Economic Hosts

Of 41 cherry cultivars inoculated with SCV, 18 showed reduction in growth. The responses were cultivar specific and variable (136, 269). Inoculated Tilton and Riland apricot were affected (136).

Other Hosts

J. H. Hale peach, Kwanzan flowering cherry, Mazzard F12/1, seedlings of *P. capuli*, *P. maackii*, *P. serotina*, and *P. tomentosa*, when artificially infected, are symptomless carriers of SCV. Similarly infected mazzard and mahaleb seedlings are known to be carriers. The virus could not be recovered by indexing inoculated plants of Shiro plum, Italian Prune, Bartlett pear, and Russian apple (R/12740-7A). Inoculated Elberta peach did not show symptoms, and the trees have not yet been reindexed.

Symptoms

Effects on sweet cherry trees vary from none on some cultivars to severe leaf epinasty and bark necrosis of current season's growth on others. Some cultivars are dwarfed in varying degrees because of reduced internodal space (fig. 121), and a few show a spiral growth. Tips of stunted shoots are thicker, rougher, and more brittle than normal. As a group, sour cherry cultivars are less affected by the virus than sweet cherries and, in some cases, appear to be stimulated (68, 136 and P. W. Cheney, M. D. Aichele, C. L. Parish, and E. C. Blodgett, unpublished data). Apparently there are no fruit symptoms on cherries. Cultivars observed in fruit include Badacsoner, Bing, Deacon, Early Richmond, English Morello, Montmorency, Napoleon, Schmidt, Sue, and Van (P. W.



FIGURE 121.—Effect of the spur cherry virus. On the *left* are 10 Van nursery trees simultaneously budded and inoculated the year before. On the *right* are 10 Van nursery trees similarly propagated, but not inoculated. All trees are on mazzard root.

Cheney and C. L. Parish, personal communication). Tilton and Riland apricot, when bud-inoculated with SCV, show typical apricot ring pox symptoms.

Transmission

Transmission is easily accomplished through budding or grafting. Virus movement within a sweet cherry tree (e.g., Bing) appears to be rapid because trees single-bud inoculated in the fall will exhibit good symptoms early the next season. There is no evidence of natural spread in orchards, although spread through natural root grafts of closely spaced cherry trees has occurred.

Control Measures

Use certified nursery stock for propagation. Stock propagated from SCV-infected mother trees is not eligible for certification.

Remarks

Propagation in the nursery from budwood from infected Bing cherry trees results in poor bud stands and irregular and variable growth (68). A few near-normal appearing shoots from the original diseased tree grow as normal Bing trees in the nursery row, but most nursery budlings grown from affected shoots appear to make dwarfed trees (E. C. Blodgett and M. D. Aichele, unpublished data). The virus appears to affect susceptible sweet cherry cultivars more severely when the trees are propagated on *Prunus avium* rootstocks than on *P. mahaleb* (P. W. Cheney, M. D. Aichele, C. L. Parish, and E. C. Blodgett, unpublished data).

SWEET CHERRY ROUGH FRUIT

BRYCE N. WADLEY

Cherry rough fruit was discovered in 1960 in plots at the Howell Field Station, Utah State University, North Ogden. Symptoms were observed in Van (*Prunus avium*) and in other sweet cherries with labels indicating Iranian origin (683, 687). Budwood was apparently brought into the station by students from Iran in 1957. Because of lack of suitable rootstocks for propagation, some of the buds were grafted to Van and others were placed in mahaleb trees. The virus nature of the disease was determined during 1960–64. All affected and inoculated trees were destroyed in 1965. No evidence of natural spread was obtained.

All sweet cherry cultivars tested are hosts of cherry rough fruit virus (CRFV). These include Van, Bing, Lambert, Napoleon, and Valera, and 32 sweet cherry selections from Iran. Peach, apricot, Italian Prune (*P. domestica*), and Montmorency sour cherry were inoculated but developed no symptoms. No attempt was made to recover the virus from these plants.

The most characteristic symptoms are the roughened and crinkled surface of affected fruit at maturity (fig. 122). Fruit symptoms on dark-fruited cultivars

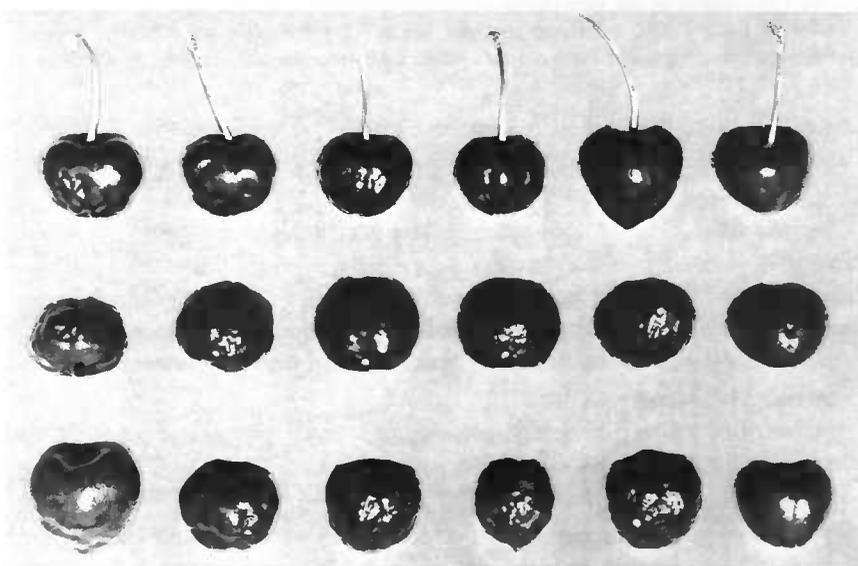


FIGURE 122.—Three fruits at *right edge* are normal Van sweet cherries; the others are fruits of Iranian cherry selections and Van affected by cherry rough fruit, showing roughening and crinkling.

initiate as small, irregular-sized red spots. As the fruits mature, these spots intensify in color and become bright red before color develops in the surrounding tissue. The flesh beneath the spots appears to stop growing and matures earlier than the surrounding tissue. This leads to depressions and an uneven, roughened surface. As the fruits mature, the colored spots become obscured but the crinkled surface remains. No diagnostic leaf symptoms have been observed, although leaves on an infected Van tree were smaller and paler green, and terminal growth was less than that on an adjacent healthy Van tree. Fruit symptoms vary from year to year but are generally more severe on large fruit than on small. Symptoms are milder on light-colored fruit cultivars; they do not develop red spots but do produce some rough fruit.

Transmission was readily achieved in bud or tissue grafts to sweet cherry. However, no symptoms developed until the second or third year after inoculation. Then, only fruits growing near the inoculum buds developed rough fruit symptoms. The virus moved slowly through the inoculated trees, and, after 5 years, fruits on many branches were still healthy. The virus moved somewhat faster in Van than in Bing or Lambert.

Infected trees should be removed and destroyed. Van is suggested as the index host for this virus. This virus disease was inadvertently introduced into the United States in budwood from a foreign source and illustrates how easily such diseases can be brought in if the budwood is allowed to bypass plant quarantine inspection.

TOBACCO MOSAIC VIRUS INFECTION IN *PRUNUS*

R. M. GILMER

Tobacco mosaic virus (TMV) was initially isolated in 1967 in New York from sour and sweet cherry trees (251). TMV has since been recovered from mahaleb rootstocks that originated in Washington and Oregon, a fact suggesting that the virus may be widely distributed in stone fruits.

TMV has been isolated from trees of sour cherry, sweet cherry, and from seedlings of mahaleb cherry. Other stone fruits are probably infected, although TMV was not isolated from any of eight peach cultivars or from peach seedlings in limited indexing.

TMV was isolated from apple (251, 373), pear (263), and grape (257). Isolates from sweet cherry infect a large number of herbaceous plants, including *Chenopodium quinoa*, a useful host for recovering the virus from stone fruits. Havana 423 tobacco, a local lesion host, is useful for confirming virus identity.

TMV is evidently a parasite but not a pathogen in stone fruits because none of the trees from which TMV was isolated have shown any symptoms attributable to TMV infection. The foliage of infected mahaleb seedlings was normal in appearance and the seedlings grew vigorously. A Montmorency sour cherry tree in the Geneva virus-indexed foundation planting contained TMV, but the tree appeared completely normal in all respects. Several infected sweet cherry trees of different cultivars were also without symptoms.

All of the isolates of TMV recovered from the stone fruits closely resemble isolates obtained from apple, pear, and grapes. All are "yellow" strains, similar to the tomato or *Dahlemense* isolates. The thermal inactivation point is between 90° to 95° C., and dilution end point in expressed *C. quinoa* sap is 10⁻⁴ or greater. Electron micrographs depict straight rods about 300 m μ in length.

It is doubtful that the occurrence of TMV infections in cherries is of any economic significance. Infected trees are without symptoms, and the present, very limited data suggest that the concentration of TMV in infected cherries is quite low (251). In direct isolations from cherry foliage, inoculum usually induced only 1 to 3 local lesions per plant in the sensitive indicator *C. quinoa*, and attempts to recover TMV from leaves of known infected trees often failed. The virus was transmitted more readily from petals than from leaves (251).

How TMV is transmitted in the stone fruits is not known. Recovery of TMV from yearling mahaleb seedlings suggests that TMV may be transmitted through seeds of stone fruits as it is in apple, pear (263), and grapes (257).

XYLEM ABERRATION

A. JUERGEN HANSEN

Xylem aberration is a little-understood disease syndrome of sweet cherry, sour cherry, and apricot. It has been reported only from the Okanagan Valley, British Columbia (412, 419). Field symptoms consist of stunting and severe distortion of terminal shoots, irregular weaving growth of sideshoots, distortion of leaves similar to that encountered in the twisted leaf disease, irregular fruit distortion, development of cankers on shoots and branches, and extensive gumming throughout the framework. After bud transmission from infected field trees, the same range of symptoms was encountered in indicator trees. In addition, a severe necrotic streaking and pitting in the wood of the trunk and major branches was observed (fig. 123). All symptoms were most severe on sweet cherry, notably Sam, and occurred in a milder form in Montmorency sour cherry. In Wenatchee (Wenatchee Moorpak) apricot, leaf twisting was not observed, but pitting was similar to that in sweet cherry.

Field spread of the syndrome has not been observed, although affected trees were found in scattered locations. However, natural spread has occurred over short distances in experimental plots. In experimental work, inoculated trees have not always developed the full range of symptoms; especially the wood pitting has been absent in several cases where other symptoms were present.

The nature of the xylem aberration syndrome has not been definitely established. Transmissibility, natural spread, and the type of symptom induced are the only indications that a virus may be involved. The disease differs in several aspects from *Prunus* stem pitting (206, 396, 470) observed in the East and should be considered a separate disease unless future research shows that a relationship exists.

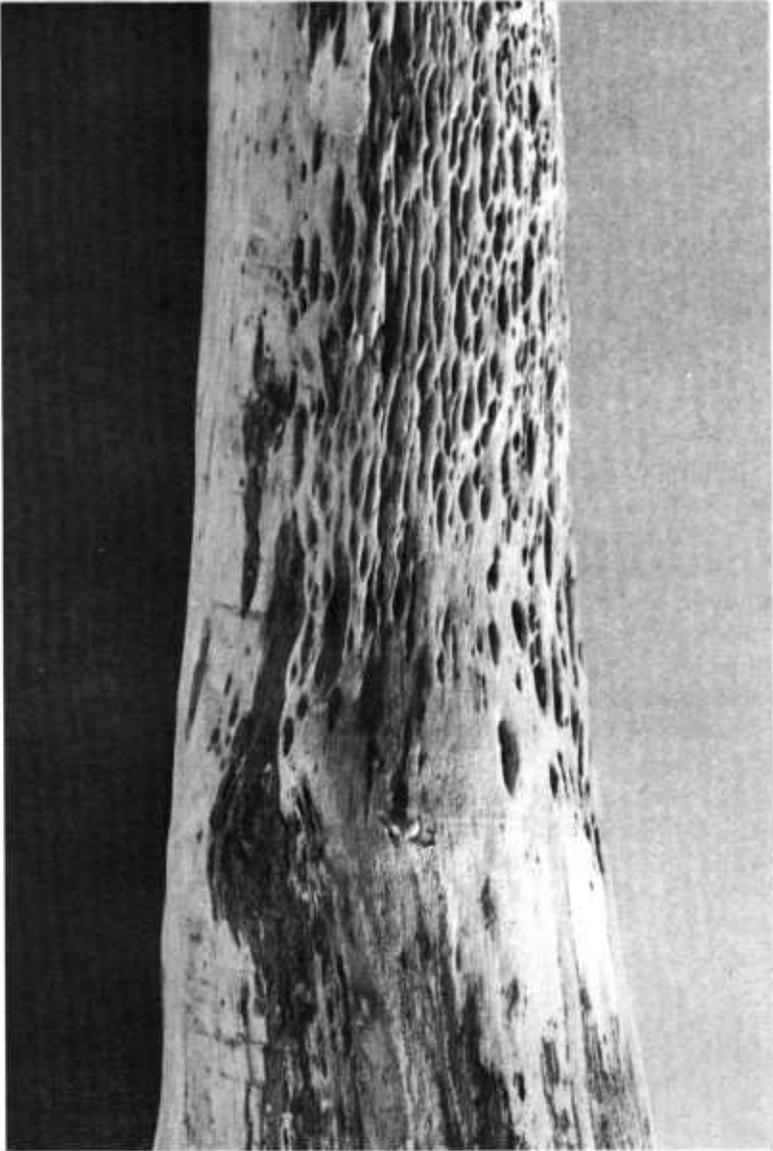


FIGURE 123.—Xylem aberration. Severe pitting in trunk section of a cherry tree (bark removed).

NONTRANSMISSIBLE DISORDERS THAT RESEMBLE VIRUS DISEASES

ALMOND CORKY SPOT

H. K. WAGNON, H. E. WILLIAMS, and J. A. TRAYLOR

An excessive necrotic leaf spotting disorder of Nonpareil almond trees was first noted in 1936 in a young orchard of mixed almond cultivars in Butte County, Calif. From 1936 to 1938, C. F. Kinman conducted virus transmission studies on the disorder, and from 1940 to 1944, G. L. Stout, L. C. Cochran, and E. E. Wilson carried out additional studies. During 1957-63, the problem of virus transmission was further investigated, particularly in regard to disease-free almond nursery stock and to verify previous tests (634, 701).

The cause of almond corky spot has not been determined but is known to be associated with occasional almond seedling rootstocks. Early work provided evidence that virus might be involved because symptoms developed on healthy Nonpareil scions grafted to affected orchard trees; however, graft-inoculation tests on almond seedlings (634, 701) and on healthy Nonpareil trees failed to demonstrate a graft-transmissible agent and also failed to show the disorder to be bud-perpetuated. The disorder occurs often on Nonpareil but occasionally on Jordanolo. Corky spot develops only on cultivars budded to seedling almond rootstocks, particularly those grown from bitter almond (*P. amygdalus*) seed.

The outstanding symptom is the development of extensive necrotic spotting of the leaves, the spotting being predominantly near the leaf margins (fig. 124). The necrotic spots begin to appear as early as April and increase in size and number as the season advances. By late summer, all or parts of the necrotic areas may fall away, thus creating a perforated leaf with irregular margins. The roots of a limited number of trees that were examined had bark that was thicker than normal and somewhat spongy in texture. Stem pitting symptoms have not been observed on these affected roots.

The disorder should not be confused with oedema, which is characterized by similar symptoms developing under certain environmental conditions. Oedema is somewhat rare in almond and is not restricted to the Nonpareil cultivar. Usually, when oedema occurs, it affects many trees in a planting and does not occur consistently each year on the same tree as does corky spot.



FIGURE 124.—Four Nonpareil almond leaves showing necrotic spotting symptoms of corky spot, compared with normal leaf on the *left*.

APRICOT GUMBOIL

GEORGE NYLAND

Gumboil is a nontransmissible disorder of apricot in California (652) known to occur in Contra Costa, San Benito, Santa Clara, San Joaquin, and Stanislaus Counties. Scattered affected trees occur in orchards of Royal and Tilton.

The disease is most striking in trees at least 10 years old. Affected trees are smaller and less fully branched than unaffected trees. There is some dieback of small shoots, and annual terminal growth is less than normal. The most conspicuous symptom is the extremely rough, cracked bark of the trunk and older branches (fig. 125). The roughness becomes less pronounced toward the younger portions of the tree, where it gives way to more or less discrete swellings in the wood and bark. Surface bark removed from these areas reveals masses of brown gum in pockets of necrotic tissue (fig. 126). In older branches, the pockets extend from the inner wood to the surface of



FIGURE 125.—Apricot gumboil. A 15-year-old tree of Royal apricot on Myrobalan 29C, showing rough bark and overgrowth at the union.

the bark (fig. 127). On 3- to 6-year-old shoots, only discrete blisters, or gumboils, can be seen. Gumboils are difficult to find in wood younger than 3 years.

On myrobalan roots, affected trees are markedly overgrown at the union

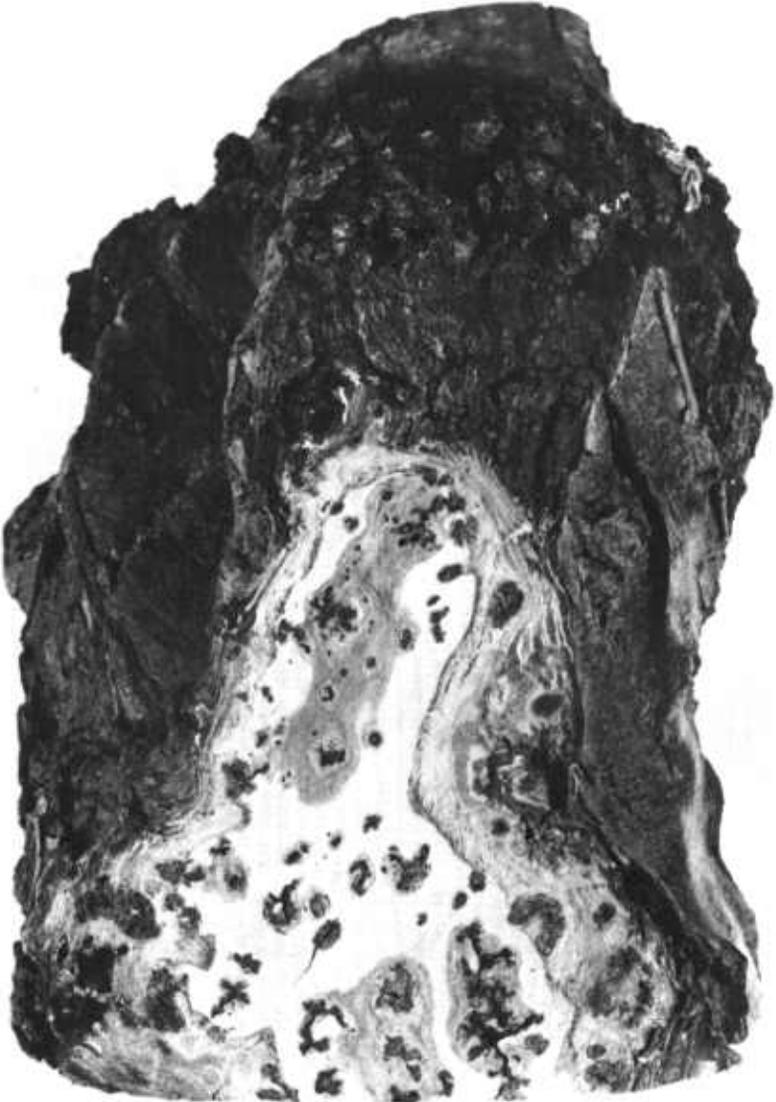


FIGURE 126.—Apricot gumboil. Scaffold limb of a 15-year-old tree of Royal apricot (tangential face) showing pockets of gum and necrotic tissue.

(fig. 125). Such trees exhibit excessive development of root suckers. The understocks are much smaller than those of normal trees of the same age. Affected trees are usually the first in the orchard to show signs of water stress between irrigations.

Fruits are fewer on affected trees than on healthy trees, but otherwise appear normal for the variety. Typically, they mature a few days earlier than fruits on unaffected trees.



FIGURE 127.—Apricot gumboil. Scaffold limb of Royal apricot (cross section) showing gum-filled necrotic lesions extending radially from 4th-year wood.

Distribution of diseased trees in the orchard suggested that propagation was the means by which the disease was spread. Most affected trees of the same age showed the same degree of symptom expression. A tree was occasionally found with symptoms more severe or advanced on one side or in one or two main branches than in the others. No partially affected trees were found. Affected trees occurred singly or in scattered groups of two or more. In one case, it was possible to trace the origin of affected trees to a specific orchard containing diseased trees.

The disorder is perpetuated by buds or scions from affected trees placed on Myrobalan 29C, myrobalan seedlings, or apricot seedlings, but not when peach seedling rootstock is used. Healthy scions of Royal and Tilton grafted on an apricot tree with good symptoms of gumboil did not develop symptoms in 5 years. When buds from trees with symptoms of gumboil were placed in healthy trees of Royal and Tilton, no transmission occurred during the following 10 years (652 and author's unpublished data). All attempts to transmit the disorder in tests reported earlier failed.

There are at least three types of rough bark disorders of apricot, none of which has been transmitted; gumboil is the type most often encountered.

Gumboil and other rough bark disorders of apricot can be avoided by using healthy trees as sources for propagation.

CHLOROTIC FLECK OF MYROBALAN PLUM

R. M. GILMER

History and Distribution

The name, "chlorotic fleck," suggested by Brase and Parker (97), appears preferable to the term "asteroid spot" applied by Hildebrand (307), who originally described the disorder. Asteroid spot had been previously used as the name for a virus disease of peach (162) not related to the present disorder.

Chlorotic fleck was initially observed about 1941 in New York. Hildebrand (307) reported an incidence as high as 90 percent in myrobalan nursery stock.

The disorder is common every year in New York nurseries, but its prevalence fluctuates considerably.

Schuch (603) observed a similar or identical leaf flecking of Brompton plum (*Prunus domestica*) in Germany in 1957. Although chlorotic fleck has not been reported specifically from other areas, it is undoubtedly more widespread than the limited published observations indicate.

Economic Hosts

Myrobalan plum and Brompton plum are the only reported hosts. A closely related disorder, yellow spot of peach, occurs in California (746) and has been observed on peach seedlings in New York and Pennsylvania (R. M. Gilmer, unpublished data).

Symptoms

In New York, symptoms of chlorotic fleck are evident from late June until October but are more prevalent in late August and September. Chlorotic fleck appears only on fully expanded, mature leaves, and the symptoms persist for the remainder of the growing season after they have developed.

Chlorotic fleck is most prevalent on 1- or 2-year-old myrobalan seedlings and is much less common on large myrobalan trees. When symptoms occur on large trees, they are commonly restricted to the foliage of water sprouts or suckers arising at the base of the trees.

Circular chlorotic spots or flecks, ranging in size from mere pinpoints to spots 1 to 2 mm. in diameter, develop on mature leaves. Although spots may occur anywhere on the leaf lamina, they are often concentrated along the midrib or lateral veins (fig. 128). Margins of the smaller flecks are often diffuse, but in the larger spots, the margins are usually sharply defined. Such spots may show indications of one or more concentric rings.

The number of chlorotic flecks on a single leaf may vary from one to many; if flecking is abundant, the leaf lamina is often distorted and twisted. Severely affected terminals are rosetted with many leaves reduced in size, and the entire myrobalan seedling may be severely stunted.

Symptoms may also develop in the young bark of affected shoots. Spots on

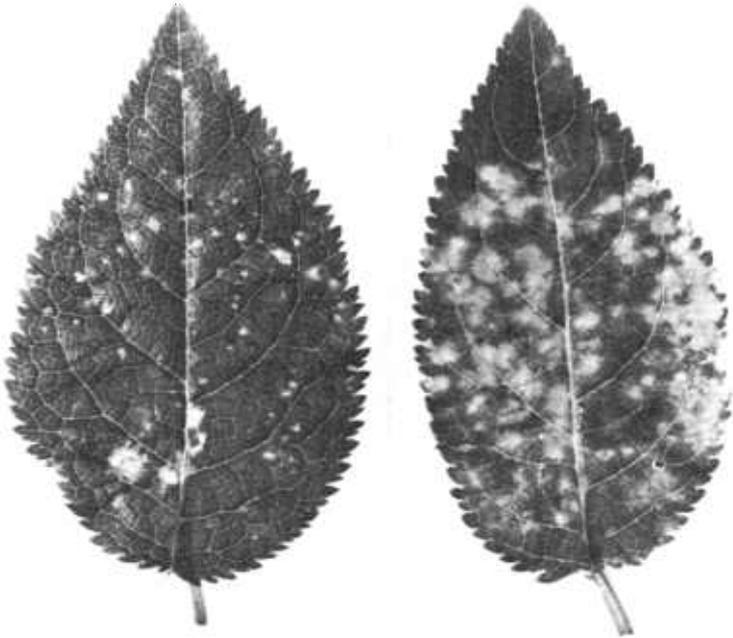


FIGURE 128.—Symptoms of chlorotic fleck on leaves of myrobalan plum infested with the eriophyid mite, *Aculus fockuei*.

young bark are generally oval rather than circular, generally larger than foliage flecks, and are often reddish.

Chlorotic fleck of myrobalan plum is a toxicogenic injury resulting from feeding by an eriophyid mite, *Aculus fockuei* (Nal. & Trt.). Schuch (603) associated the same mite with leaf flecking of Brompton plum in Germany. A related disorder, yellow spot of peach (p. 292), is induced by a closely related mite, *A. cornutus* (Banks), in California (746), and what is presumably the same mite has been found on peach seedlings with yellow spots in New York.

Mites feeding on mature foliage did not induce chlorotic fleck, but symptoms appeared 13 to 33 days after the mites fed on immature leaves (258). Wilson and Cochran (746) similarly found that yellow spot of peach occurred only after *A. cornutus* had fed on immature foliage and that about 12 days elapsed A related disorder, yellow spot of peach (p. 292), is induced by a closely related from the injection of a toxin by the mites.

The possibility that chlorotic fleck is a virus disease with *A. fockuei* as its vector appears to be conclusively excluded by the failure to transmit the disorder by bud inoculation or propagation from affected seedlings (258).

Control Measures

Chlorotic fleck was completely eliminated in nursery plants by three applications of a miticide at 2-week intervals from late June through early August (258).

Remarks

Hildebrand (307) reported that the occurrence of chlorotic fleck in myrobalan rootstocks resulted in poor budtake of Italian Prune buds and subnormal growth of Italian Prune budlings. Brase and Parker (97) also found that Italian Prune, Stanley, and Abundance plum budlings were less vigorous when propagated on myrobalan seedlings with severe chlorotic fleck. Since the disorder is easily controlled, increases in grade of nursery stock may repay the costs of application of a miticide.

CONSTRICTION DISEASE OF STANLEY PRUNE

K. G. PARKER and R. M. GILMER

Synonyms

Decline of Stanley prune.

History and Distribution

Brase (96) discovered a constriction of the rootstock immediately below the union with the scion in declining trees of Stanley prune (*Prunus domestica*) in New York. The affected Stanley trees were propagated on myrobalan seedling rootstocks and appeared in orchards planted after 1943 but not in those planted before this date. Brase (personal communication) considered that the presence of constriction in plantings made after 1943 was possibly associated with the change from European to domestic sources for myrobalan rootstocks caused by World War II.

Constriction is common in Stanley prune orchards in New York. Few or many trees of an orchard may be affected—occasionally, so many that the entire planting is removed. As far as the authors are aware, constriction is not a serious problem in other areas because Stanley prune is not widely grown elsewhere.

Economic Hosts

Stanley prune is the only cultivar of commercial importance affected with constriction but identical symptoms have been observed in selections of several breeding lines of prunes developed at the New York State Agricultural Experiment Station. Very similar disorders have been reported in apricot or Japanese plum propagated on myrobalan rootstocks. (See "Remarks.")

Symptoms

Constriction may develop at any time after a Stanley tree is propagated until at least the 10th year of growth in the orchard. Incipient constriction may occasionally be visible in 1-year-old nursery trees, but more commonly the disorder is not evident until 1 to 5 years after the tree is planted in the orchard. Since the scion-rootstock union is commonly planted below ground, the early stages of constriction are generally overlooked until it has progressed to the point that tree growth is affected. Many affected trees grow vigorously for 2 to 5 years before foliage symptoms are observed.

The initial symptoms are weak growth of terminals and yellowing and upward rolling of the foliage. Kirkpatrick et al. (375) reported loss of chlorophyll from the interveinal areas of the leaves. These areas gradually change from green to yellow to yellow-orange and ultimately become bright red. The areas immediately contiguous to the veins remain green for some time but finally also become orange or red. At this stage, the leaf margins are necrotic, and areas of necrosis may develop in the lamina.

Affected trees are weakly anchored in the soil. When the scion-rootstock union is examined, a pronounced overgrowth of the scion is evident ("inverted shoulder") and the rootstock is markedly reduced in diameter (fig. 129). The overgrown scion tissues are cheeselike in consistency. Numerous, abnormally large lenticels appear in the scion bark immediately above the union. Below the union, the bark of the rootstock is necrotic in large areas or completely dead (fig. 130).



FIGURE 129.—Overgrowth of the scion and constriction of the myrobalan rootstock in a Stanley prune tree *right*. A normal Stanley tree of the same age at *left*.

Cross sections at the union area reveal considerable disorganization of the phloem and xylem of both scion and rootstock.

Affected trees usually die within 1 to 2 years after the foliage symptoms, which result from constriction, become evident. Less commonly, affected trees collapse completely within a short time, particularly under conditions of severe moisture and temperature stress.

Control Measures

Brase and Parker (97) found that Italian Prune trees propagated on myrobalan seedlings did not develop constriction even though Stanley trees propagated on seedlings of the same lot became constricted. Brase (personal communication) therefore suggested the use of Italian Prune as an intermediate stem piece between the Stanley scion and myrobalan rootstock. During the past 8 years, Stanley trees with such interpieces have not developed constriction.

Brase (unpublished data) also tested selections of myrobalan as prospective seed source trees. The seedling progenies from these selections are currently being tested as rootstocks for Stanley. Some of them appear promising for direct propagation with Stanley.



FIGURE 130.—*Left*, Three Stanley prune trees with constriction, showing varying degrees of necrosis of the myrobalan rootstocks after removal of bark; *right*, normal Stanley prune tree of the same age with bark removed.

Remarks

Parker and Brase (unpublished data) compared the occurrence of constriction in five individual selections of Stanley scionwood propagated on myrobalan seedlings from six seed source trees. The presence and severity of constriction were consistently associated with the source of rootstocks and were not influenced by scion source. Constriction commonly developed in trees propagated on seedlings of a myrobalan tree infected with *Prunus* ring spot virus (PRSV); however, constriction also developed in several trees propagated on seedlings of a second myrobalan tree not infected with PRSV.

Brase and Parker (97) suggested that the later development of constriction in Stanley trees was perhaps related to the occurrence of a chlorotic spotting in the leaves of some myrobalan seedlings. Hildebrand (307) had previously suggested that this leaf spotting might be of genetic origin. Later studies by Gilmer and McEwen (258) demonstrated that this chlorotic leaf spot in myrobalan resulted from feeding by an eriophyid mite.

Progenies of certain breeding lines of plums, particularly those derived from either of the two parents of Stanley (Agen x Grand Duke) also developed constriction when propagated on myrobalan seedlings. Of selections of four breeding lines in which Grand Duke was a parent, 30 to 50 trees propagated on myrobalan in 1961 had died of constriction by 1968 (J. P. Watson, unpublished data). Occasionally, constriction also developed in selections of breeding lines derived from Imperial Epineuse and Golden Transparent Gage (J. P. Watson, unpublished data).

Day (185) noted a disorder much like constriction in Kelsey plum (*P. salicina*) and Sergeant plum (*P. domestica*) on seedling myrobalan rootstocks and in French Prune and Sergeant trees on clonal myrobalan rootstocks. A similar disorder was reported in apricot propagated on myrobalan seedlings (543). Whether these difficulties are identical with constriction or not, myrobalan seedlings from different seed source trees obviously vary considerably in their compatibility with certain plum cultivars.

Although a virus causation has not been completely excluded, there is no direct evidence that constriction is caused by a virus. The absence of constriction in Stanley trees separated from the myrobalan rootstock by an intermediate stem piece of Italian Prune suggests that a virus is not involved. Clearly, the incidence of constriction is higher in certain lots of myrobalan rootstocks, and constriction develops only when such rootstocks are propagated with certain prune cultivars. These observations suggest that constriction is a type of scion-rootstock incompatibility affected by the genetic composition of both scion and rootstock. Yet this explanation is difficult to reconcile with the fact that in many instances constriction develops only after the tree is several years old. It is possible that initial mild stages of constriction weaken the trees just enough to predispose them to low temperature injury at the union and that this further injury leads to the further development of constriction (375).

HIGH TEMPERATURE MOTTLE OF MONTMORENCY CHERRY

PAUL R. FRIDLUND

Leaves on rapidly growing shoots of apparently virus-free Montmorency sometimes expand with mild to severe mosaic patterns of light and dark green. The symptoms resemble those of a virus-induced mosaic or a chimaera (fig. 131). The patterns may continue to appear in the succeeding leaves that are formed on the shoot. However, because a shoot usually ceases to elongate when these mosaic symptoms appear, the symptoms are primarily limited to a few of the tip leaves. The mosaic persists after the leaves enlarge, but symptoms never appear in older, expanded leaves.

The upper surface of an affected leaf appears to have a pebbled texture with raised dark and sunken light-green areas, but this is probably an optical illusion. Additionally, the lighter areas appear to border on the veins, but this is not always true.

The exact cause of this nontransmissible abnormality is unknown, but it is primarily a greenhouse problem associated with high temperatures, particularly at night. At Prosser, Wash., greenhouse-grown Montmorencys frequently produce this symptom if constant temperatures are maintained at 26° C. or higher, but symptoms rarely occur at lower constant temperatures. Symptoms

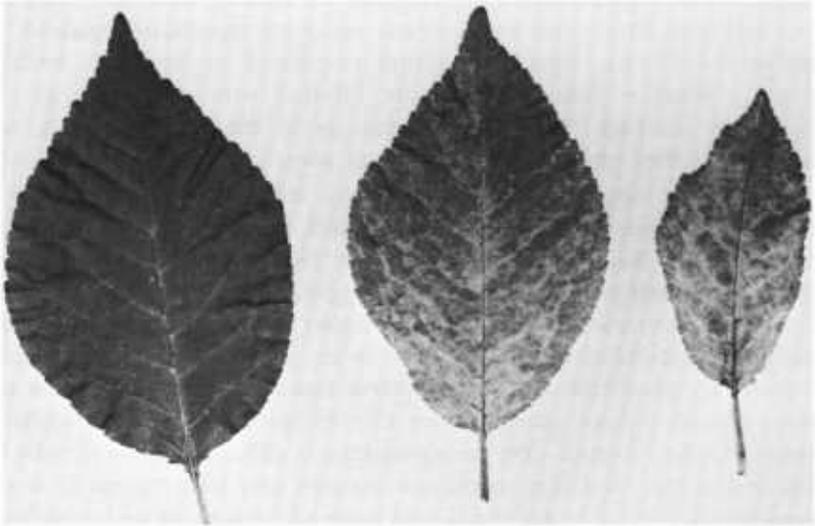


FIGURE 131.—*Right*, Two leaves of Montmorency cherry showing mottle induced by high temperature, compared with normal leaf, *left*.

occur more frequently late in the spring than during the winter, but this may result from a seasonal rise in greenhouse temperatures. When the conditions for symptom production in Montmorency appear ideal, a few plants of other species may also produce abnormal tip leaves. For instance, oriental flowering cherry, *P. serrulata*, may have distorted tip leaves with abnormal veins, and apricot may form tip leaves similar to those affected by 2, 4-D. However, the abnormality is invariably more frequent in Montmorency than in the other species.

Field grown Montmorency at Prosser rarely are affected. A late-summer mildew infection of the tip leaves can be confused with this abnormality.

ITALIAN PRUNE LEAF SPOT

A. W. HELTON

Synonyms

Italian Prune leaf spot sometimes is called shot hole (296, 303), and it may be referred to by a number of descriptive terms indicative of any of the several associated symptoms (294, 297).

History and Distribution

Symptoms of leaf spot generally are found wherever the Italian Prune (*Prunus domestica*) cultivar is grown (294, 297). These symptoms often are severe and have caused increasing concern among prune growers since the condition was first reported in Idaho in the 1930's (49, 54). Leaf spot occurs in serious proportions in the prune-growing districts of Oregon, Washington, Idaho, and Utah (577). Nursery improvement programs have reduced the severity of the problem in parts of the West, but no known clone of standard Italian Prune is entirely free of the condition in all orchard environments.

Economic Hosts

Italian Prune is a common host, but conditions with similar appearance have been observed on Stanley prune (294) and Beaty plum (610).

Symptoms

First growth in the spring generally is normal, with symptoms developing early in the summer. The necrotic spots, from which the name of the disorder is derived, may vary from very small (1 to 2 mm.) to large blotches of dead tissue (fig. 132) that sometimes coalesce and engulf most of the leaf. Trees affected with leaf spot often express several symptoms which may be classed together as the leaf spot complex (292, 297). The characteristic brown spots may not always be present, but where they occur they frequently develop concentric lines in the browned tissues. Such spots sometimes drop out, leaving a shot hole condition that can be confused with infections of *Coryneum* blight (303) or *Prunus* ring spot virus (PRSV) (294). The most common of the symptoms that may accompany the brown spots is a chlorotic mottle (59, 577) reminiscent of virus or powdery mildew infection or of prune leaf casting mottle (255). The mottling symptom often is more prevalent than the brown spotting symptom, and it generally precedes or accompanies the brown spotting. The mottling may be very indistinct or very striking, most often consisting of light-green to rusty areas scattered over the surfaces of affected leaves.

The various symptoms of leaf spot grade into each other and may disappear and reappear in affected branches (297). This is true also of the genetic abnormality in prune trees known as crinkle (64, 298). Such behavior does not typify the classical concept of a genetic disorder. Severity of the various symp-



FIGURE 132.—Italian Prune leaves showing variations of the brown leaf spotting symptoms most characteristic of leaf spot. Note the accompanying chlorotic spotting where the necroses are less advanced.

toms seems to be governed primarily by environmental conditions (60), and, in extreme cases, extensive defoliation results (49).

The presence of leaf spot can lead to depressed yields (584), severe fruit drop (49, 584), and production of shriveled, sunburned, gum-spotted fruits (49, 584).

Nursery trees with leaf spot generally produce dwarfed and unsatisfactory orchard trees. Growth of less affected trees may be near normal. Bloom and fruit set seem to be affected only in the most severe cases. Trees propagated on peach rootstocks seem to be more adversely affected than those on myrobalan plum roots.

Control Measures

Removal of affected trees is advisable when extreme symptoms develop or when symptoms are detected in newly planted trees. Propagating wood should be taken only from selected clones with a minimum leaf spot history. Even with the best clones now in use, occasional leaf spot trees have been observed in the propagations.

Remarks

Italian Prune leaf spot symptoms have not been reproduced in unaffected trees by exhaustive tests employing standard grafting techniques (49, 297, 577). However, the symptoms can be perpetuated to varying degrees in growth from

carrier buds that have been grafted to other trees. Therefore, leaf spot is a bud-perpetuated condition, and hence a genetic disorder, despite the fact that perpetuated symptoms often are unstable and may disappear after 4 years or more (297). Although the symptoms are similar, infection with PRSV does not cause leaf spot, but leaf spot is common in PRSV-infected trees (296).

ITALIAN PRUNE SPARSE LEAF

EARLE C. BLODGETT

This abnormality of standard Italian Prune is characterized by lack of foliage (57). Trees affected with sparse leaf are large, round-headed, tend to be umbrella shaped, and develop an open type of growth. When the trees are young, growth is probably nearly normal. In the orchard, affected trees can be identified easily when in leaf by their lack of foliage and in the dormant stage by their shape. There may be an abnormal delay of about 10 days in time of blooming (62) and of leafing out.

Affected trees bear very sparingly. The prunes are perhaps larger than normal, but they seem to be normal in color, shape, and texture.

The foliage on affected trees is extremely sparse in comparison with that on normal trees. The sparseness seems to be due principally to lack of small-twig growth on which leaf and fruit buds are normally borne. Apparently the lower and inner small branches either do not form or die soon after they are formed. The leaf surface of affected trees is little more than an estimated 50 percent of that on normal trees. The foliage is confined largely to twig terminals on the periphery of the tree. The early leaves in particular are deformed somewhat similar to sweet cherry leaves affected with crinkle leaf. They are smaller than normal and have irregular margins and diffuse mottling.

Although transmission tests were not completed, the disorder is regarded as a genetic abnormality that can develop as a mutation. Budwood for propagation should be taken from normal bearing trees.

NONINFECTIOUS BUD FAILURE IN ALMOND

DALE E. KESTER

Synonyms

Shatter-bud; crazy-top; muletail.

History and Distribution

This disorder was first described by Wilson and Stout (742) in 1944, but trees affected by noninfectious bud failure were observed in California almond orchards many years prior to that time. The disorder first occurred in Nonpareil and Peerless and for many years was not considered a serious problem. It began to cause serious concern when Jordanolo, a variety introduced in 1937, began to be affected in high percentages in commercial orchards (743). The disorder has become so severe in Jordanolo that this cultivar is no longer planted. In 1953, it was discovered in Jubilee. Since about 1950, the incidence of affected trees of Nonpareil, the leading commercial cultivar, has increased significantly, particularly in newly established areas in the San Joaquin Valley of California. Since then, the disorder has appeared in such other cultivars as Harpareil and Merced.

Trees affected by the disorder are found throughout the almond-growing districts of California. It is particularly prevalent in the upper end of the Sacramento Valley and the southern part of the San Joaquin Valley. Affected trees have been reported from Turkey, Israel, and Tunisia (H. K. Wagnon, P. Spiegel-Roy, and E. E. Wilson, personal communication)

Economic Hosts

Almond cultivars affected include Nonpareil, Peerless, Jordanolo, Harpareil, Jubilee, and Merced. The last four cultivars are offspring of Nonpareil. In addition, numerous unnamed seedlings and selections, have been affected, particularly when Nonpareil or Jordanolo is one of the parents. The disorder has been reported in Marcona, a cultivar rarely grown in California (689).

Symptoms

The disorder is manifested through two principal symptoms or characteristics: (1) failure of vegetative buds to grow out in the spring (bud failure) and (2) cracked or roughened areas on shoots (rough bark). The level of expression can vary continuously from slight, where only a few buds are affected, to severe, where practically all buds are dead and extensive rough bark areas occur.

Bud failure results from the fact that vegetative buds become necrotic some time during late summer or fall (590, 597). These seemingly dormant buds fail to grow out in the spring and abscise and drop about 6 weeks later. Consequently, bud failure characteristics are best determined in the spring within 6 to 8 weeks after growth begins. If examined later than that, identification may be obscured by new growth and the inability to distinguish between bud

failure and other factors causing lack of set. Not all of the buds will fail on the branch, and those that remain viable will grow into normal-appearing shoots during that same summer. These few shoots are usually vigorous and grow at right angles to the branch. Growing points on these shoots are normal during the early part of the growing season but lose their viability sometime during the summer or fall, although this fact is not apparent until they fail to grow the following spring. Repeated annual cycles of this bud failure result in a recognizable atypical growth pattern (fig. 133).

Sometimes bud failure is limited to the center part of the shoot, with normal growth at the end to give the so-called muletail look. If failure occurs in buds at the end of the shoot, dieback results. Foliage is sparse.

Flower buds on affected branches are usually normal unless the tree is very severely affected. Very often the flowers on moderately affected branches will bloom late—1 to 2 weeks after those on nonaffected branches. Fruit set is not affected.

Cracked bark results from necrosis of a portion of bark cortex (597). Affected areas may vary from small lesions one-half inch or less in length at the node to areas extending a foot or more along a branch. The beginning of such cracked bark areas can often be observed on new shoots by late summer or early winter. The rough bark areas become more pronounced and are particularly characteristic of branches 2 to 3 years old or more. The wood beneath the rough areas is often discolored. The rough bark bands do not necessarily develop every year on a given tree but may occur in 2 to 3 year cycles.

Control Measures

Control of noninfectious bud failure in a commercial planting is achieved either by replacing the entire tree or by topworking. Scions can be taken either from a different cultivar known to be free of the disorder or from the same cultivar but from a nonaffected source. Which procedure is best to follow is a horticultural decision. A tree that is not seriously affected might well be left until production falls to an uneconomic level.

Prevention of the disorder depends either on developing or identifying clonal lines not affected by the disorder or by developing propagation sources for budwood with no or low potential for the disorder. Unfortunately, no reliable method exists to carry out these objectives.

The following guidelines may be helpful in reducing the probability of propagating trees with bud failure.

1. Select budwood source trees that are at least 5 to 6 years old or older. Since symptoms are most apt to appear during the early life of a tree, this precaution may increase the chances of avoiding potential affected sources.

2. Examine the trees carefully for bud failure or rough-bark symptoms. If some trees of a given orchard are affected, it is likely that others, though outwardly normal, will carry the potentiality. Consequently, it would be better to avoid a source where some trees are affected.



3. Maintain the identity of trees propagated from specific budwood sources. If a program of followup inspection of orchards propagated from specific sources is practiced, it should be possible in time to identify sources that have less bud failure potentiality than others.

Remarks

Graft transmission tests.—Tests for graft transmission from Nonpareil, Peerless and Jordanolo have been negative although the disorder is readily bud-perpetuated (741). Affected Nonpareil trees have been topworked with scions from normal trees. The growing scions have then remained normal for observation periods up to 20 years. Topworking has become an accepted practice to control the problem, although it is difficult to obtain completely nonaffected scionwood of some varieties. Bud failure in other cultivars later found to be affected (Merced, Harpareil, and Jubilee) has not been tested specifically, but the disorder appears to be the same as that in Nonpareil, Peerless, and Jordanolo. Heat treatments do not overcome bud failure (George Nyland, unpublished data).

This disorder has some similarity to almond virus bud failure in which set is reduced, fruit are misshapen, and foliage is upright, greener, and persists later into fall than with noninfectious bud failure.

Seed transmission.—Noninfectious bud failure is a genetic abnormality readily transmitted by seed (740). Transmission from parent to offspring appears to account for the origin of the disorder in current almond cultivars. Two aspects of the bud failure problem exist. One is the distribution of affected trees within specific clones with vegetative propagation. The other is the identification of susceptible clones arising as selections from families of seedling trees.

The distribution of bud failure trees in affected clones tends to follow a random pattern, although some observations suggest a directional aspect. It is not possible at present to identify an affected tree before the external symptoms actually appear. Bud failure is bud-perpetuated but can also develop in trees propagated from source trees that were outwardly normal (744). The bud failure phenotype can develop at any age. There is likely to be a gradual increase in the number of affected trees within an orchard, although the increase is more likely to be greatest while the trees are relatively young. The percentage of affected trees that develops in any one orchard can range from a few percent to 75 percent or more. There is usually a continuous gradation in severity from none to slightly affected to severely affected, not only in different trees but also

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FIGURE 133.—*A*, Nonpareil almond affected by noninfectious bud failure, showing, in center, one vigorously growing branch less affected than the others; *B*, Nonpareil almond tree showing, at right, unaffected branches that developed from a healthy graft placed on understock 7 years previously and, at left, the branches that developed from the affected understock.

within the same tree. If a mildly affected tree is pruned severely, the new growth will be normal at first but eventually will develop bud failure, often more severely than before.

Wilson (744) has described progressive increase in percentage of affected trees with consecutive vegetative propagations from a single source beginning with the original seedling tree. The vegetative generations have been designated as S (original seedling tree), S + 1 (first scion generation), S + 2 (second scion generation), and so on. The differences in phenotypic levels of expression within a clone are gametically transmitted (366). An association between incidence of bud failure, high summer temperatures, and rapid shoot growth has been noted, but a cause and effect relationship has not been established experimentally.

The inheritance of bud failure from normal and affected parental trees has been investigated (365, 366). The pattern of inheritance found involves the production of families of seedling individuals, each of which exhibits all the potential instabilities described in the previous paragraph but differ in the level of bud failure potentiality that each inherits. In crosses involving affected and nonaffected Nonpareil and Peerless, there was a continuous gradation from those individuals that developed extremely severe symptoms when 1 or 2 years old to those that developed symptoms only after many years and after the trees had been propagated into advanced scion generations. The age when the bud failure phenotype develops within a family of seedlings is directly related to the bud failure severity in the two parents. Bud failure is transmitted through both male and female gametes. Families derived from crosses where both parents were affected showed early and pronounced development of bud failure; families from crosses where only one parent was affected were less precocious in bud failure development; and families from crosses where neither were affected produced some bud failure individuals but only after much delay and after trees had been propagated into advanced scion generations. The development of individuals of this latter group that are normal but with bud failure potential appears to account for the existence of bud failure susceptible varieties from breeding programs.

NONINFECTIOUS PLUM CRINKLE LEAF

T. S. PINE

Noninfectious plum crinkle leaf is a severe leaf distortion of Japanese plum, analogous in many ways to prune crinkle leaf and sweet cherry crinkle leaf, that occurs occasionally in southern California and Washington (525). Single branches or an entire tree may be affected. The disorder is found most often in the Santa Rosa cultivar and its derivatives.

Affected portions of trees are conspicuously dwarfed. The leaves are one-third to one-half normal size and distorted in a variety of patterns: strap-shaped, pronounced lobes along the margins, or severely indented margins (fig. 134). Some leaves show a pale-yellow pattern along the margins or a mottle extending over the whole blade. Affected leaves are thickened and tend to become brittle early in the growing season. Leaf midveins are enlarged, and the entire leaf venation is formed irregularly. Leaves produced early in the growing season are



FIGURE 134.—Leaves from a Santa Rosa plum tree affected by noninfectious plum crinkle leaf and showing various leaf distortions.

always deformed; later leaves may be nearly normal. A few blossoms may appear on affected branches, but they are imperfectly formed. Affected trees and branches are almost entirely unfruitful. Many leaf and fruit buds abort, so that affected trees appear sparse and stringy. Although this disorder is readily bud-perpetuated, all attempts to transmit it through tissue grafts have failed.

Plum crinkle leaf apparently results from a genetic instability. The disorder is of economic importance only when affected budwood is introduced into nursery operations. Although symptoms of plum crinkle leaf resemble closely those of prune crinkle leaf and sweet cherry crinkle leaf, the leaves of affected plum trees are more severely distorted than those of prune or sweet cherry.

NONINFECTIOUS PLUM SHOT HOLE

T. S. PINE and MAURICE F. WELSH

Noninfectious plum shot hole occurs occasionally in commercial plums in the West and commonly in some hardy plum hybrids in the Great Plains States and the Canadian Prairie Provinces. The disorder was described first on Beaty plum, a hybrid of *Prunus angustifolia* x *P. munsoniana* (610). Because this condition occurs frequently in native *P. munsoniana*, it was thought that the trouble might be the result of a genetic weakness in *P. munsoniana* and that this weakness was carried in plums arising as seedlings and hybrids of this species (611). However, the appearance of plum shot hole in varieties and hybrids not related to *P. munsoniana* indicates the disorder is not species-limited.

Spring growth of affected *P. salicina* trees is normal. As the season progresses, the older leaves at the base of the shoots develop translucent flecks that enlarge rapidly. The centers of these spots turn brown or purple-brown, and in most cases the dark-colored areas separate from the rest of the leaf at a definite line of cleavage and drop out (fig. 135). The resulting shot hole condition develops progressively toward the shoot apex, and by midsummer the foliage is riddled with holes. The spots and holes in most leaves range in size from pinpoints to 1 to 2 mm. in diameter, their size usually being inversely proportional to their number in the affected leaf. The spots are generally circular but may become irregularly shaped as several spots coalesce. Affected trees vary individually in



FIGURE 135.—Noninfectious plum shot hole in leaves of Beaty plum, showing dark necrotic spots leading to shot hole.

symptom expression, some being severely and others only mildly damaged. In some trees, the spots are small and the centers do not fall out; in other trees, the spots are large and the centers fall out as they are formed. The general vigor of affected trees is not reduced. The disorder is known to be seed borne. Although noninfectious plum shot hole has never been transmitted from affected to healthy trees by bud grafts, it is readily perpetuated in growth from the inserted buds.

Throughout the Great Plains States and the Canadian Prairie Provinces similar symptoms are common on western sand cherry x plum hybrids, such as Sapa and Opata (fig. 136). Yellow blotching occurs on the leaf lamina on one or both sides of the midrib, usually accompanied by leaf puckering. The yellow tissue becomes necrotic and is shed. Frequently all leaves on long portions of shoots are reduced to midribs only.

Other hardy "prairie" plums such as Grenville, Radisson, and Tecumseh frequently suffer similar leaf tattering. However, the loss of leaf lamina is not preceded by yellowing or necrosis, but by the appearance of lines of fine shot holes that radiate from the point of pedicel attachment to the upper leaf margins. Mild symptoms of this type are occasionally apparent on Italian Prune



FIGURE 136.—Noninfectious plum shot hole in leaves of Sapa plum, showing various stages of yellow blotching, puckering, necrosis and shedding of necrotic tissue.

(*P. domestica*) and other plum cultivars. These syndromes have persisted in plum clones subjected to heat therapy.

Noninfectious plum shot hole apparently is a genetic abnormality. It is not economically important but can be confused with infectious diseases having similar symptoms. It has many characteristics in common with Italian Prune leaf spot. It differs from plum rusty blotch in symptomatology and by being a midseason rather than an early season disease.

PEACH FALSE WART

C. G. WEIGLE

Peach false wart (578) is seen occasionally in the West on fruits of several cultivars of peach. It bears this name because it shows some resemblance to the symptoms on fruit caused by peach wart virus. As with peach wart, there are no observable symptoms except on fruit. But false wart differs from virus-caused wart in several ways.

False wart differs in the number of fruits it affects on a tree. Only one to five affected fruits, rarely 10 to 15, are found on an orchard tree. The warty fruits are not confined to one branch or spur, and trees with affected fruits are randomly scattered in the orchard. The warts are characteristically different from those found on virus-infected fruit. The erumpent growths are usually large and somewhat variable in shape. They may be found any place on the sides of fruits, but generally not at the styler end. Generally, there is only one wart on a fruit. The center of the wart often has bony tissue, which compares in hardness with the seedcoat. The bony tissue in some warts extends, like a needle, up to three-eighths of an inch into the flesh. Gumming is not characteristic (fig. 137). Moreover, the condition does not persist in the tree from year to year. Buds from trees with affected fruits failed to produce the condition



FIGURE 137.—False wart on peach fruits. Two fruits at *left* show "crease wart."

when grafted to healthy trees, and warts did not appear in the growth from the inserted buds.

While the cause of peach false wart has not been determined, it is generally accepted to be the result of feeding punctures made by one or more species of hemipterous insects. False wart may be the same as "beady wart" mentioned by Blodgett (51) but differs from "crease wart," which Blodgett considers to be a varietal reaction. False wart is not economically important. It is described here since it presents a diagnostic problem in the field because of its similarity to virus-caused peach wart.

PEACH VARIEGATION

A. W. HELTON

Peach variegation was reported first in 1940 from Idaho (48). Since that time, the condition has been recognized on several peach varieties in Idaho, and it probably occurs in most peach-growing districts. The disorder sometimes is referred to as a chimaera (55, 63). Symptoms generally appear on a single branch of affected trees. Whole trees seldom are affected even when buds in the axils of symptom-bearing leaves are used to produce new trees.

Naturally occurring symptoms have been observed in J. H. Hale, Elberta, Redhaven, and peach cultivars and seedlings of unknown identity. Similar symptoms have been found in Idaho in sweet cherry and sweet cherry seedlings, mahaleb cherry, myrobalan plum, Italian Prune, apple, and strawberry (*Fragaria vesca*) (293).

Peach leaves affected by variegation produce streaky blotches, characterized by three distinct, sharply delimited shades (fig. 138)—normal green, light green, and cream to white. Growth of affected twigs generally is reduced, and a yellowish discoloration of the bark tissues sometimes is associated with the

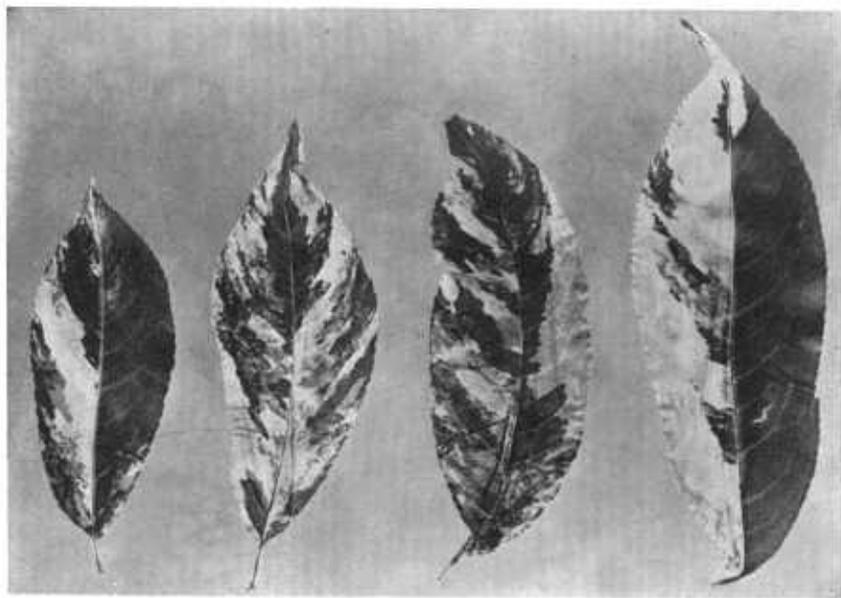


FIGURE 138.—Leaves of J. H. Hale peach showing typical symptoms of noninfectious variegation.

foliar symptoms. Trees only partially affected need not be removed, but wood from them should not be used for propagation.

Peach variegation symptoms may be confused with those of peach calico, but the latter is not characterized by the three distinct shades of color. Variegation is not transmissible. It is bud-perpetuated, but the perpetuated symptoms may not be of the same intensity as those of the original tree. This is true for the other affected species as well as peach. Observed variation in symptoms on a tree from one year to another suggests that environmental conditions influence symptom intensity. Peach variegation is of minor economic importance.

PEACH YELLOW SPOT

NORTON S. WILSON and T. S. PINE

Peach yellow spot is a nontransmissible disorder caused by the peach silver mite, *Acalus cornutus* (Banks). This is the eriophyid mite that also causes widespread silvering of peach foliage just before leaf drop in the autumn. The disease is characterized by yellow spotting, vein-associated leaf chlorosis, longitudinal leaf rolling, and stem spotting (746). The leaf discoloration, which closely resembles the symptoms of several mosaic-type virus diseases, is caused by a toxin injected by the mite during feeding. The toxin may move short distances within the leaf, either by diffusion among the parenchyma cells or along vascular bundles, but cannot be considered generally systemic.

Affected peach seedlings develop light-yellow chlorotic spots and vein feathering in the young leaves approximately 10 days after the mite feeding (fig. 139). In severe cases, the spots may coalesce to produce a vivid leaf mottle. Leaves of some cultivars (e.g., *Salberta*) develop spots with red borders that become necrotic and drop out, producing a shot hole effect. Spots also appear in current-season growth, being obscure and pale yellow at first but increasing in size to become slightly raised and darker. Affected leaves of some cultivars (e.g., *J. H. Hale*) often show puckering and longitudinal rolling.

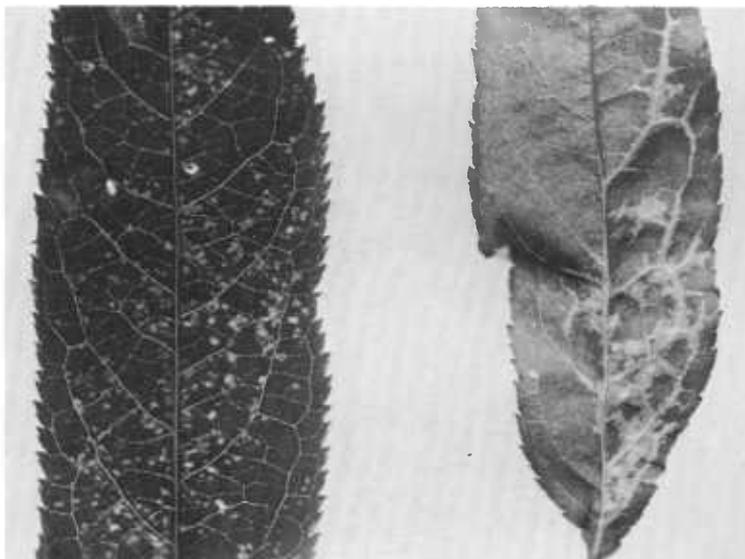


FIGURE 139.—Damage caused by the mite *Acalus cornutus* in Lovell peach leaves: chlorotic spotting on left; heavy vein feathering on right.

Symptoms develop only in leaves that are rapidly growing and unfolding and not in fully mature leaves. Production of vein feathering seems to be correlated with infestations of very young leaves in which the toxin is introduced directly into the vascular bundles. Affected trees are not permanently damaged. Populations of *A. cornutus* fluctuate greatly, and traces of earlier infestations can be difficult to detect by the time yellow spot symptoms are evident.

Symptoms similar to those described above, and associated with infestations of *A. cornutus*, were seen on both peach and apricot in British Columbia (Maurice F. Welsh, unpublished data). Leaf patterns in apricot usually were larger than those in peach, and the leaves were most severely affected. Plum rootstocks (*P. cerasifera*), growing in the same planting, also developed a strong mottle in the tip leaves.

PLUM RUSTY BLOTCH

T. S. PINE

History and Distribution

Plum rusty blotch was reported first in 1960 (524) as occurring in Santa Rosa and Late Santa Rosa plums (*Prunus salicina*) in California, but the disorder had been seen in several parts of the State prior to that time. It is now known to occur in all the plum-growing areas of California (519). Similar disorders have been reported in Israel (67) and Argentina (678).

Economic Hosts

The only known stone fruit trees affected by this disorder are Japanese plums and hybrid plums closely allied to *P. salicina*. Symptoms of the disorder have been seen in Elephant Heart, Gaviota, Howard Miracle, Kelsey, Laroda, Late Santa Rosa, Nubiana, Redroy, Santa Rosa, Shiro, and Star Rosa growing in California orchards. The disorder has been induced experimentally in Abundance and Beauty plums. Affected trees have been found growing on their own roots and on apricot, plum (Marianna and myrobalan), and peach rootstocks.

Symptoms

In new spring growth of affected trees, chlorosis develops at the base of the leaf blade and progresses toward the apex along one or both margins (fig. 140). This chlorosis tends to be continuous at first but may eventually take the form of chlorotic blotches. Within a few weeks, the blotches become reddish-brown, and many small red spots appear in the discolored areas. These spots may increase to about one-eighth inch in diameter or remain the size of a pinpoint. Eventually they all become necrotic and drop out, leaving the blotchy portions of the leaves irregularly shot holed. The rusty blotch and necrosis may cover the whole leaf blade when leaves are severely attacked. Affected leaves are reduced to one-third to one-half the size of healthy leaves and tend to become misshapen as the growing season progresses. Young or old leaves on a branch may show the onset of symptoms first, but by midsummer all the leaves on an affected branch usually show some damage.

Symptoms in orchard trees generally are observed first on a single branch. Because the leaves are discolored and reduced in size, these branches appear stringy and are readily noticed. Affected branches produce few blossoms and almost no fruit. However, leaf symptoms and reduced fruit production may not occur simultaneously.

Control Measures

Plum rusty blotch was not eliminated from buds of affected trees by heat treatment for 7 weeks at 38° C. (George Nyland, unpublished data). Removal of affected branches does not prevent the appearance of symptoms in other



FIGURE 140.—Leaves of Santa Rosa plum affected by plum rusty blotch showing typical reddish-brown blotches.

portions of a tree. Damage from this disorder is reduced by selecting budwood for propagation from source trees that have a history of producing healthy foliage and consistently high yields of fruit.

Remarks

Plum rusty blotch originally was described as a transmissible disorder resembling a virus disease (524). Healthy-appearing experimental trees had developed clinical symptoms of rusty blotch 4 years after being inoculated with buds from affected trees, and the appearance of the disorder in nearby untreated trees was assumed to result from the natural spread of a virus by an unknown vector. Although these experiments were repeated several times, the continued appearance of rusty blotch in untreated trees and the widespread occurrence of genetic disorders in plums generally suggested that more evidence was needed before this disorder could properly be called a virus disease. Nontransmissible

leaf shot holing, although quite different in symptomatology from rusty blotch, occurs in many plums and plum hybrids (710) and makes definite characterization of plum disorders a difficult procedure.

In recent investigations, healthy-appearing Beauty plum trees, which have not been found naturally affected in orchards, developed symptoms of rusty blotch the second year after they were approach-grafted to affected Santa Rosa trees. Abundance plum, in which rusty blotch has not been seen in commercial plantings, developed symptoms the first year after they were bud-grafted or approach-grafted to affected Santa Rosa. Although untreated trees of these two varieties remained normal-appearing, it is not known at the present time if the symptoms were induced by a virus, by another graft-transmissible pathogen, or if the association of affected tissues merely hastened the appearance of a genetic change.

The appearance of plum rusty blotch in the cool, humid coastal valleys of California as well as in the hot, inland valleys seems to indicate that climatic conditions are not a factor in its development. Good cultural practices do not diminish the severity of symptoms or reduce its incidence in affected orchards. Development of foliar symptoms and reduction in fruit set are more evident in Santa Rosa than in other plum cultivars, probably because Santa Rosa and its related cultivars are grown more extensively in California. Although other cultivars may develop severe symptoms, the disorder is found in proportionately fewer orchard trees and is of less economic importance. Plum line pattern and noninfectious plum shot hole sometimes occur in trees with rusty blotch, but this coexistence does not interfere with the development of the symptoms of each disease.

PRUNE CRINKLE LEAF

EARLE C. BLODGETT

The Italian Prune, like Bing and Black Tartarian sweet cherry, is subject to genetic abnormalities. Crinkle leaf symptoms are similar and common in both prune and cherry, although there is no apparent connection.

Prune crinkle leaf is known in both regular and early sports of Italian Prune cultivars and is widespread in the Northwest. It has been noted on the prune cultivars Emily, Merton, and Hungarian.

Leaves on crinkle leaf affected trees show a wide range of leaf deformities, the most common of which are uneven margins and distorted blades (fig. 141). Affected leaves generally are smaller and lighter in color than normal leaves and often show chlorotic areas near the restricted portion of the blade. The mottled or marbled appearance is dull and quite distinct from the even green of normal leaves. In older trees, prune crinkle leaf symptoms may occur only on one spur or one branch, or on larger portions of the tree. These frequent occurrences on formerly healthy branches probably represent genetic instability. The symptoms and characteristics are quite similar to those described for sweet

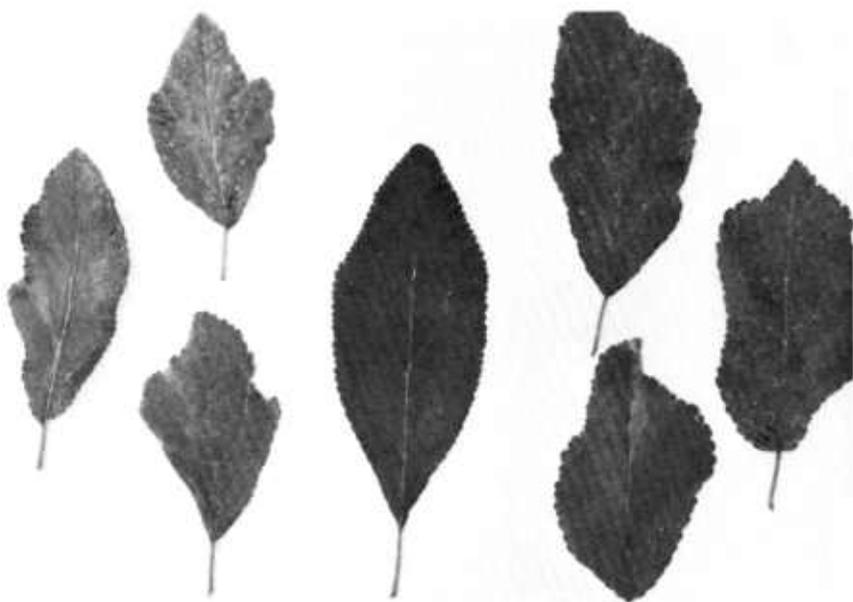


FIGURE 141.—Six Italian Prune leaves affected by prune crinkle leaf, showing irregular margins, distortion, and chlorotic areas; normal leaf in center.

cherry crinkle leaf, which occurs most commonly on the Bing and Black Tartarian sweet cherry. Trees with prune crinkle leaf usually are dwarfed; the growth is spindly, and the fruit may ripen earlier than normal. In general, crinkle leaf-affected trees tend to be unfruitful. Fair crops may set, but most of the fruit falls before maturity. The fruit stems on affected trees are noticeably shorter than those on normal trees. There are cases, however, in which trees with prune crinkle leaf are extremely vigorous but unproductive. A similar condition is often found in sweet cherry trees affected with crinkle leaf. Examples are known in which normal branches developed on crinkle leaf-affected prune trees.

Trees affected by crinkle leaf usually are dwarfed and have a denser foliage than those with sparse leaf, another genetic abnormality of prune. Transmission tests consisted of topworking crinkle leaf trees with scions of healthy prune. No transmission was obtained after 3 to 4 years in two separate tests (64). Limited trials indicated that a small percentage of seedlings grown from Italian Prune seeds, collected from a commercial cannery, developed symptoms of crinkle leaf.

In nursery stock grown 15 to 20 years ago in Washington, crinkle leaf was very common. Under the present certification program, the disease is virtually unknown in the nurseries. The frequency of crinkle leaf mutations is very low in prune as compared with crinkle leaf in Bing cherry.

PRUNE LEAF CASTING MOTTLE

K. G. PARKER and R. M. GILMER

Synonyms

Prune mottle; prune leaf mottle.

History and Distribution

In the original description of prune leaf spot, Blodgett (49) reported a mottle was often present in leaves with leaf spot. In some years, only mottle symptoms developed on certain trees, and the leaf spot symptoms were absent. In this and a later report (53), he presented evidence that prune leaf spot was bud-perpetuated but not transmissible. He suggested that leaf mottle might be a preliminary phase of leaf spot, but considered the evidence inadequate to determine whether leaf mottle was bud-perpetuated (53).

Prune leaf mottle is apparently common in the West, because the complex of leaf spot and mottle was observed in Oregon, Washington, and Utah (72). The senior author observed that leaf mottle symptoms similar to those occurring in New York were common in prune orchards in Washington in 1951.

Leaf mottle symptoms were observed in Italian Prune trees in the East for many years prior to an initial report in 1952 (110). The disorder was fully described as leaf casting mottle in 1957 (255).

Economic Hosts

In New York and other Eastern States, leaf casting mottle is common and widely distributed on Italian Prune. Identical symptoms have been observed on several other prunes, including Albion, Tragedy, Grand Duke, and Hungarian (255). The disorder has never been observed on Stanley prune.

Symptoms

The foliage of affected Italian Prune trees is initially normal. In midsummer, chlorotic spots appear on many leaves; these spots finally become brownish yellow and translucent, with indefinite margins (255). Somewhat later, the leaf lamina between the spots becomes yellow with a green halo surrounding individual spots (fig. 142). Many individual spots become necrotic in their centers, and severely affected leaves soon drop.

Richards et al. (586) suggested that prune leaf mottle and prune leaf spot were distinct. They described prune leaf mottle appearing in mature leaves as small, diffuse, light-green or chlorotic areas irregularly scattered over the leaf lamina with chlorotic areas coalesced in places to form a mosaic pattern. During the summer, gray or purple-brown necrotic centers often developed in the chlorotic areas. Affected leaves often dropped prematurely but sometimes persisted with a muddy or gray appearance and irregular marginal chlorosis.

Both reports (255, 586) indicate that symptoms are more severe with increasing defoliation at high temperatures. Environmental conditions are ob-

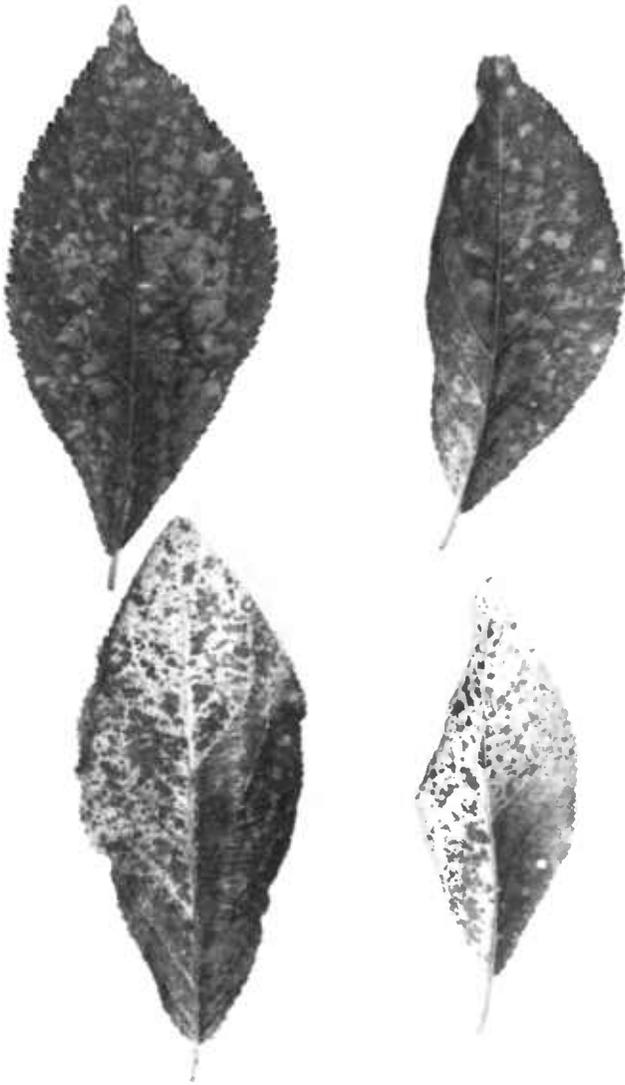


FIGURE 142.—Prune leaf casting mottle in Italian Prune leaves: Chlorotic spots, *upper*, and green halos around spots on yellow leaf lamina, *lower*.

viously significant in severity of symptoms. Minor variances in symptoms observed in Utah and New York very likely result from differences in the environment of the two areas.

Control Measures

As indicated in the following section, leaf casting mottle is bud-perpetuated. The sole practicable control is to propagate trees from scion sources free of the

disorder. Scionwood source trees should be examined carefully for foliage symptoms, particularly at midseason and after extended periods of high temperatures. In trees with very mild symptoms, foliage may appear quite normal in many years, and symptoms may not become evident until the trees have reached considerable size.

Richards et al. (586) suggested that Italian Prune may have an inherent weakness because most trees of the variety are affected by leaf mottle or leaf spot. It would, perhaps, be advisable not to use varieties affected with such disorders as parents in breeding programs.

Remarks

In New York, trees were propagated on peach rootstocks from four sources of Italian Prune scions: symptomless; mild leaf casting mottle; severe leaf casting mottle; and leaf crinkle (also bud-perpetuated). The resulting Italian Prune trees were cross-budded in all possible combinations, and growth of the original scion and of the inserted buds was observed for 4 years. During this period, the identities of each of the original scions and of growth from the inserted buds were not affected, i.e., symptomless scions were free of symptoms; those from the severe mottle source evidenced severe mottle symptoms. No evidence of possible virus transmission from scion to scion was observed.

Symptoms very similar to leaf casting mottle developed in the greenhouse on seedlings derived from crosses in which Italian Prune, Tragedy, or Grand Duke were parents (255). Characteristic leaf casting mottle symptoms later developed in many of these seedlings when grown in the field. These observations suggest that leaf casting mottle is a genetic abnormality.

Helton (297) presented data indicating that symptoms were often absent in trees that had previously developed symptoms of leaf mottle, leaf spot, or other disorders. Such variations perhaps again result from environment or other conditioning factors.

Cain and Boynton (110) reported yield reductions in trees with leaf casting mottle, with the amount of fruit reduction positively correlated with severity of foliage symptoms. Set of fruits was reduced on affected trees, and a large number of fruits dropped prematurely. Because leaf casting mottle is extremely common in Italian Prune trees in New York, it undoubtedly causes a considerable reduction in yields.

PUSTULAR CANKER OF TRAGEDY PRUNE

T. S. PINE

Pustular canker was described from California in 1941 as a disease limited to Tragedy prune (*Prunus domestica*) and possibly caused by a virus (621). It has not been reported from other States. Small, diamond-shaped pustules break through the bark of affected trees, usually in the young growth of the upper branches (fig. 143). The pustules develop into hard, roughened cankers that may remain small or become much elongated along the axis of the branch. Cankers have appeared in yearling trees in nurseries, but some trees have not shown the abnormality until they were 5 to 10 years old. Growth of cankered trees is somewhat stunted, twig necrosis is present occasionally, and fruit production is progressively reduced. Repeated attempts to transmit this disorder from affected Tragedy trees to healthy trees of other *P. domestica* cultivars or to other *Prunus* spp. have all failed. However, pustular canker is easily bud-perpetuated (527).



FIGURE 143.—Small pustular cankers on branch of Tragedy prune.

Pustular canker is less evident in orchards that receive maximum attention under good climatic conditions but increases in severity during exceptionally warm weather or when cultural practices are relaxed. Although high summer temperatures may favor the development of this disease, affected trees have been found in both the cooler coastal valleys and the warmer interior valleys of California. As a cultivar-limited genetic abnormality, pustular canker is not a threat to the culture of other prune cultivars. "Little diamond canker," a common name for this disorder, is no longer used because of possible confusion with prune diamond canker, a disease known to be caused by a virus.

STANDARD PRUNE CONSTRICTING MOSAIC

H. KEITH WAGNON

Standard prune constricting mosaic is known only in California and occurs only on Standard prune (*Prunus domestica*) (655, 691, 701). The disorder is of minor economic importance because Standard now is being grown on a very limited scale in California. Originally considered to be caused by a virus, on the basis of inoculation tests on Standard prune and peach (655), subsequent observations and tests indicated that the disorder is a genetic abnormality peculiar to the Standard prune (691, 701).

Symptoms consist of few-to-many chlorotic spots, $\frac{1}{16}$ to $\frac{1}{8}$ inch in diameter, in the leaf blade. A distinctive leaf-constricting feature of the disorder is the result of the concentration and coalescing of the spots in a band across the top half of the leaf (fig. 144). Often the chlorotic areas become necrotic and



FIGURE 144.—Leaves of Standard prune affected by constricting mosaic and showing chlorotic spots and leaf constriction near chlorotic areas, compared with normal leaf, lower left.

fall out, leaving only the midvein. The symptoms are more conspicuous in warm weather and are more severe in growing nursery stocks than in orchard-planted trees. Trees growing in the cooler coastal valleys of California may have few or no symptomatic leaves. Progeny trees produced from symptomless trees have the potentiality of developing symptoms of the disorder when grown under hot summer temperatures.

SWEET CHERRY CRINKLE LEAF, DEEP SUTURE, AND VARIEGATION

EARLE C. BLODGETT and GEORGE NYLAND

Synonyms

Crinkle leaf has been called curly leaf, wild trees, male trees, red bud, maple leaf, and unproductive cherry. Synonyms for deep suture are long leaf and rough leaf. Variegation is also called white crinkle.

Nature of the Disorders

The disorders of crinkle leaf, deep suture, and variegation are genetic. Crinkle leaf and deep suture frequently occur in the same tree. Variegation is most often seen in seedlings and foreign cultivars (18, 89, 533). Repeated experiments by many workers have shown that these disorders are readily perpetuated in buds and scions. Trees propagated from buds from diseased shoots are nearly always diseased. Trees propagated from healthy-appearing trees or from healthy portions of partially affected trees may or may not show symptoms. A single recessive controlling gene is postulated for which certain cultivars prone to crinkle such as Bing and Black Tartarian and to variegation such as Hedelfingen are heterozygous. Symptoms appear in a heterozygous tree when, presumably, a further mutation of the particular gene occurs (364).

History and Distribution

Crinkle leaf was first described by Kinman (368) in 1930 under the name of unproductive cherry. It has been the subject of many investigations and countless observations (166, 364, 369, 533, 564, 578). Deep suture was first described by Reeves (553) in 1941. Distribution of both disorders coincides with the culture of susceptible cultivars, and few if any orchards of these cultivars are free of the diseases. Variegation has been reported from several European countries and from New Zealand (18, 89, 533). Repeated attempts by many workers to transmit the disorders have always failed.

Economic Hosts

Crinkle leaf and deep suture have been observed principally on Bing and Black Tartarian sweet cherry. Crinkle leaf has been noted on Black Republican, Chinook, Eagle, Burbank, Dr. Flynn, Ox Heart (of America), Shelton, Waterloo Heart, and Moreau. Because these cultivars (except Black Republican) are planted sparingly, crinkle is uncommon except on Bing and Black Tartarian. Neither crinkle nor deep suture occur on Lambert or Napoleon. A similar disease is known on plum and prune but there appears to be no specific relation. In most cases, a low percentage of the three disorders is seed-transmitted (554), but in some cases, the percentage is high (18, 364, 578). Variegation is reported on Hedelfingen from Canada (364) and has been seen on other cultivars imported from Europe to the West and Canada. In New Zealand, St.

Margarets cultivar is most commonly affected, and the disorder has been seen on six other cultivars (18).

A small percentage of mazzard seedlings may show deep suture symptoms.

Symptoms

Crinkle leaf.—Trees of Bing and Black Tartarian severely affected by crinkle leaf appear wilted and have foliage that resembles that of maple trees. Leaves on affected trees are variously misshapen and mottled (figs. 145 and 146, C and D). Margins are often indented and abnormally serrated through failure of blade tissue to develop. Some leaves are oddly and severely distorted with deep sinuses and lobes and accentuated tips and bases. If removed from the tree, they would hardly be recognized as cherry leaves. The inhibited areas are usually lighter green and have a silvery upper surface and many more veins per unit area than the remainder of the leaf, which is darker green than normal. The light-green areas are present when the leaf first appears in the spring. Uneven expansion appears to be associated with a mixture of tissues some of which have more chlorophyll than others. Leaves developed later in the growing season, especially on sucker shoots, are often less affected than those produced in the spring. The total leaf surface of a moderately affected tree is approximately three-fourths that of a normal tree; yet affected trees are not noticeably reduced in growth.

Trees affected with crinkle leaf are unfruitful, not because of failure to blossom but because many blossoms are smaller than normal and are visibly defective. The peduncles are short, and most pistils are short and slender and have a tendency to discolor early (fig. 146, A and B). Some of the discolored pistils are brown before the flowers open, others become brown soon afterward, and few develop into fruits. Such fruits are small, pointed, and often have a raised suture (fig. 146, C); they are attached with their long axis at an angle to the stem. Flowers near the base of the preceding season's growth often fail to open fully. The swollen buds on such growth become reddish, and some of them remain in place for a number of weeks.

In many instances, a tree may appear entirely normal, but, upon close examination, small branches, twigs, or even a few leaves, can be found affected. Trees commonly have both normal-appearing branches and branches affected with crinkle leaf. In some instances, an entire tree is badly affected.

Sweet cherry deep suture.—Cherry trees affected with deep suture have variable percentages of malformed leaves (fig. 147) and fruits, and different trees display different degrees of malformation. Sometimes entire trees are severely affected, but at other times scattered branches through a tree or only certain lateral ones exhibit symptoms. A severely affected tree viewed from a distance often has a drooping appearance like one suffering from drought. Nursery trees affected with deep suture are often dwarfed. When such trees are transplanted, they grow very slowly, and the foliage is sparse and malformed.

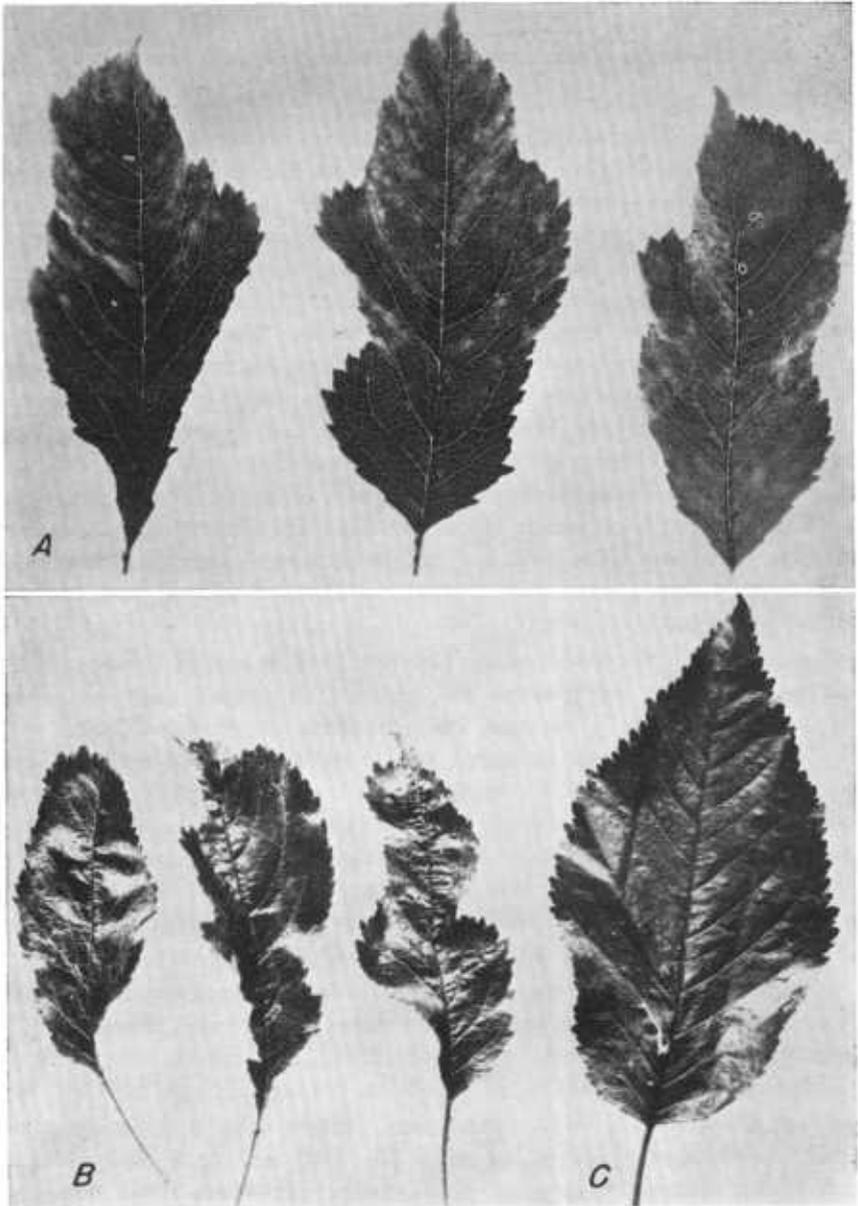


FIGURE 145.—Sweet cherry leaves affected by crinkle leaf: *A*, Bing; *B*, Black Tartarian; *C*, normal Black Tartarian leaf.

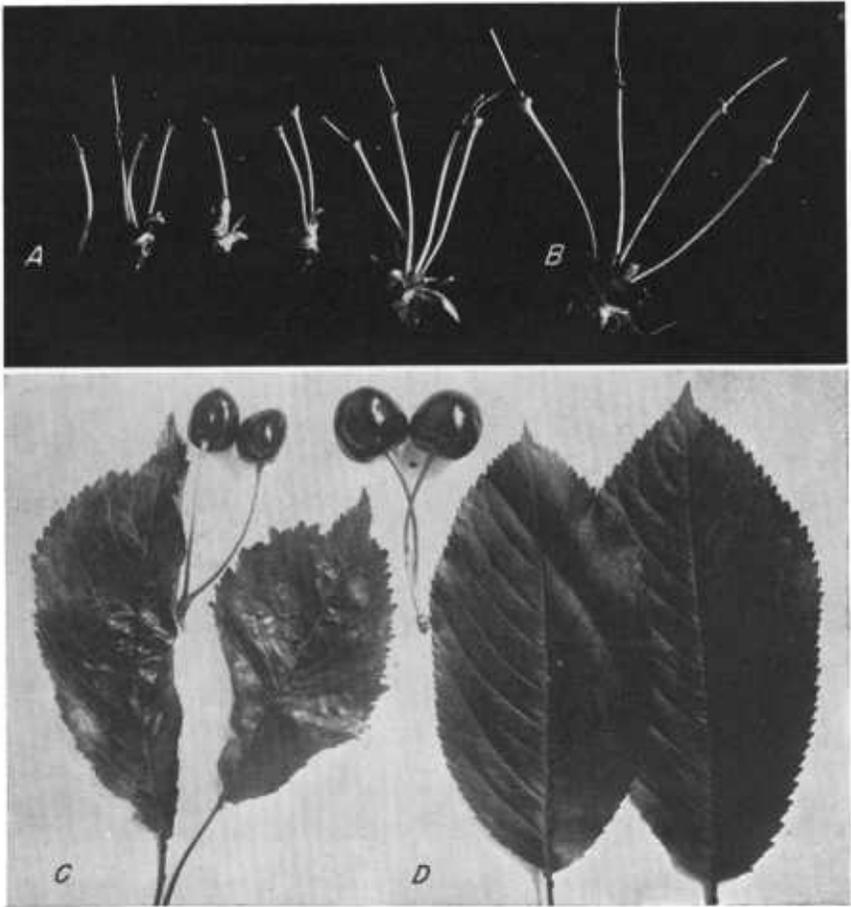


FIGURE 146.—*A*, Exposed pistils of blossom clusters from Black Tartarian cherry tree affected by crinkle leaf; *B*, comparable pistils from an unaffected Black Tartarian tree. *C*, Fruit and leaves from a Burbank cherry tree affected by sweet cherry crinkle leaf; *D*, comparable fruit and leaves from an unaffected Burbank tree.

Some trees show very little leaf malformation other than a thicker, more leathery texture and a slight rugosity, whereas the leaves of others are reduced to narrow ribbons, many of which may be only a midrib in parts and not over 1 inch wide on other parts. The mildly affected trees may show the characteristic deep suture type of leaves in the spring, but the leaves produced in summer may be nearly normal in shape. This makes detection in the nursery more difficult.

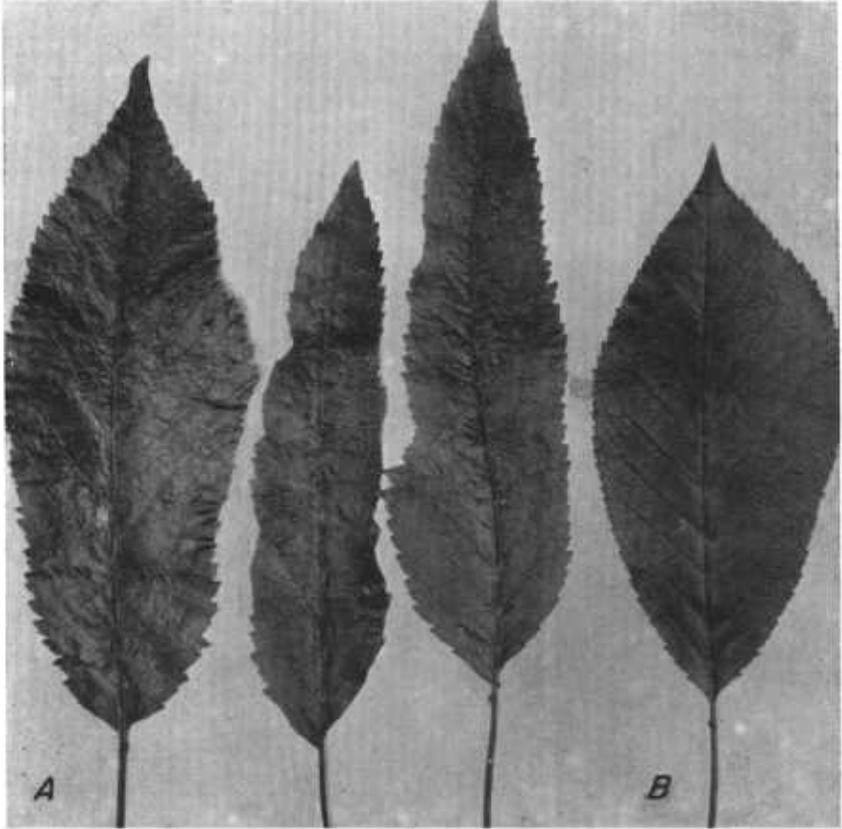


FIGURE 147.—*A*, Leaves from a Bing cherry tree affected by sweet cherry deep suture; *B*, comparable unaffected leaf from a Bing tree.

Some leaves are actually longer than normal, and their lateral veins are shorter on one side or both sides of the midvein, whereas other leaves merely appear to be longer because of their relative narrowness. The surface of the leaf may be very rough, but not puckered. The periphery of some leaves may be irregular or the leaf may be perforated. The leaf margins usually have fewer serrations than normal and sometimes serrations are lacking in portions of leaves. A faint interveinal chlorosis may be present on some leaves during the late spring, but it is less evident later in the season. Pronounced chlorotic areas like those found on trees affected with crinkle leaf do not occur. Affected leaves often tend to develop a bright sheen on the surface and are darker green than normal. Late spring frosts sometimes cause certain leaf malformations to develop on normal leaves. Some of these may easily be confused with the leaf symptoms of deep suture.

Affected fruits have a pronounced depression on the suture side and are normally rounded at the end (fig. 148). Fruits with deep suture are most often borne relatively near leaves that exhibit symptoms.

Vigorous, healthy nursery trees of Bing may have some lower leaves with distortions that resemble the symptoms of crinkle or deep suture, and these make nursery inspection difficult. Only by experience can adequate differentiation be made in the nursery.

Symptoms of crinkle leaf and deep suture may occur together on parts of the same branches and fruit symptoms may not always correlate perfectly with leaf symptoms. It is well recognized, too, that severity of symptoms in affected trees may vary year to year.

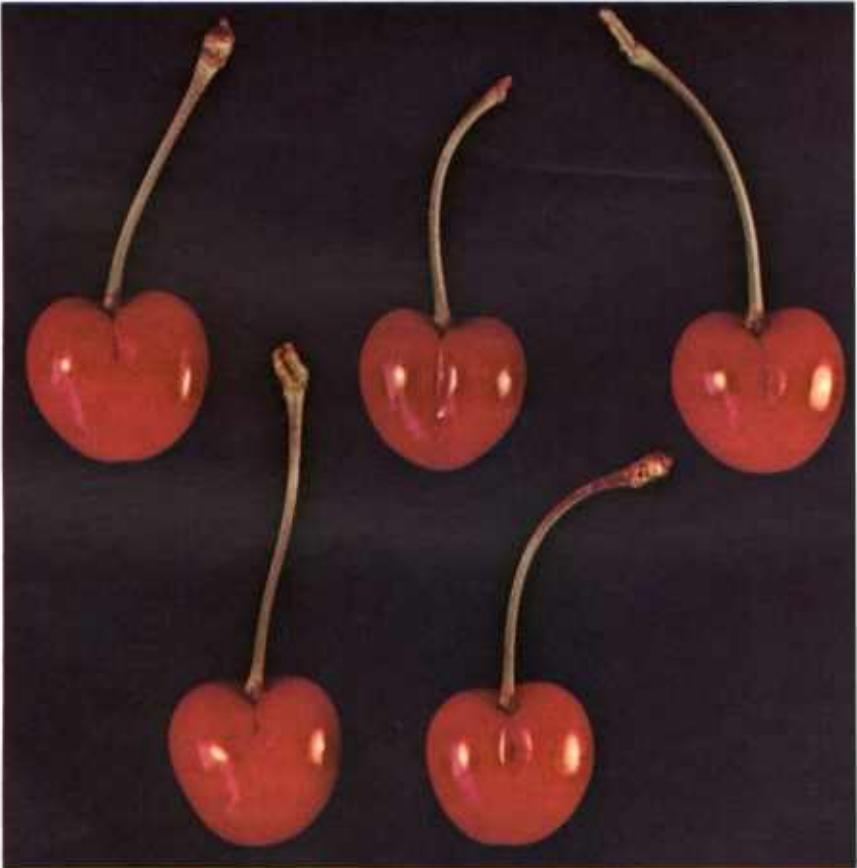


FIGURE 148.—Bing fruits affected by sweet cherry deep suture, showing pronounced depression in the suture.

Variegation.—Trees with this disorder produce leaves which lack chlorophyll in certain areas. Chlorosis may be seen as creamy or white spotting, flecking, or streaking, or sometimes the chlorotic areas give a marbled appearance. Symptoms may be restricted to a few flecks or may involve most of the leaf surface and are best seen in mature leaves. Variegated areas may occur on any part of the leaf but often occur near the margins. Leaves are sometimes distorted, and, when symptoms are severe, they roll upward at the margin (18). Portions of trees or entire trees may show symptoms. Seedlings with severe variegation may die soon after germination (364). Severely affected trees are smaller than normal, lack vigor, and bear undersized fruit (18, 89).

Control Measures

Both crinkle leaf and deep suture are regarded as mutations and not as virus diseases because they have not been transmitted and do not spread. Hence, an affected tree is not a menace to healthy trees. Repeated experiments by many workers have shown that both crinkle leaf and deep suture are readily perpetuated in scions or buds. Tests conducted by the senior writer showed that crinkle leaf may occur in nursery stock even if apparently healthy budwood is used from a partially affected tree.

It is therefore desirable to inspect mother trees carefully and avoid as sources of budwood those with even a trace of these disorders.

In the orchard, young trees showing crinkle leaf or deep suture symptoms should be removed and replaced or they should be grafted over with healthy scions. Older trees normally are kept until they are no longer economical. Topworking affected trees to varieties that are not susceptible has been suggested and is sometimes practiced. The cutting out of affected branches in young trees is recommended if only a few are affected. Crinkle branches should not become main scaffold branches. Healthy nursery stock produced under an adequate certification program is the best control known, but it will not guarantee complete freedom from crinkle or deep suture.

Climate or perhaps other environmental factors greatly influence the incidence of crinkle leaf and deep suture. An experiment was made to observe the effect of geographic location on crinkle leaf and deep suture (J. A. Milbrath and George Nyland, unpublished data). In 1959, alternate buds from current season budsticks of Bing clone 260 were placed into mahaleb seedlings in nurseries at Corvallis, Oreg., and Davis, Calif. At each location, 60 trees were transplanted as yearlings into test orchards and observed for symptoms of crinkle leaf and deep suture. During 8 years, only one shoot with typical symptoms of crinkle leaf was seen in the trees at Corvallis, but many shoots and spurs with symptoms were seen in the trees at Davis, beginning the second year after planting. Of the 57 trees remaining at Davis the eighth year after planting, 23 showed crinkle leaf and 25 showed deep suture in at least one spur or shoot. In general, the symptoms were very mild. Only 12 had neither crinkle nor deep suture. The

trees affected by deep suture averaged two strikes per tree, and those affected by crinkle averaged three strikes per tree at Davis.

Pruning had a marked effect on the number of strikes per tree of both crinkle leaf and deep suture. In the experiment described above, the trees were pruned in three ways: (1) cut heavy, as for budwood, in late August or early September; (2) dormant pruned just prior to budbreak for 2 or 3 years to make a many-limbed, vase-shaped tree; and (3) no pruning, except when planted, and later only to remove serious cross-branches. Terminals were not cut back.

The least strikes per tree, averaging all trees, were in treatment 1; the most, in treatment 2 (32 times greater than treatment 1 for crinkle and three times greater for deep suture). Treatment 3 had 11 times more strikes of crinkle than treatment 1 and four times more strikes of deep suture. These results possibly can be explained on the basis of the total number of buds allowed to produce spurs or shoots in each treatment.

The results are encouraging for the future of cherry mother blocks because trees cut heavily, as for budwood, had the least crinkle and deep suture.

Remarks

Both crinkle leaf and deep suture are very important cherry diseases. Crinkle can reduce the crop by 50 percent or more, and deep suture can cause total rejection of fruit at the packing house if lots are badly affected. Annual losses from these disorders are probably greater than from the virus diseases. Certain lots of Bing fruit from trees affected by crinkle leaf are known to have been marketed as Lambert because of the delay in ripening and the shape of affected fruit.

Repeated attempts to prove that crinkle leaf and deep suture are caused by virus have not been successful. It is difficult to understand why some Bing trees will remain healthy and others from the same source growing in the same general areas will "break down" partially or completely after several years. This may be a case of mutation triggered by certain environmental conditions.

DIAGNOSIS OF STONE FRUIT VIRUS DISEASES AND CERTAIN DISORDERS THAT RESEMBLE VIRUS DISEASES

Virus disease symptoms can be simulated by mineral deficiencies or excesses, damage by herbicides, air pollutant injury, or the effects of low temperatures. To make suitable evaluations of causal factors, the following sections on nutritional disorders, herbicide injuries, and air pollution injuries have been included in the Handbook.

Symptoms that may be induced by nutritional imbalances as well as by viruses include *foliar chlorosis* (for example, by iron or manganese deficiency or by peach yellows, sour cherry yellows); *leaf spotting* and *shotholing* (for example, by arsenic toxicity, nitrogen deficiency, or by *Prunus* ring spot); *little leaf* and *rosette* (for example, by zinc deficiency or by peach rosette); *bark cankering*, *splitting*, and *gumming* (for example by boron deficiency, boron toxicity, manganese toxicity or by necrotic rusty mottle, prune diamond canker, *Prunus* stem pitting).

Foliage and fruit symptoms induced by 2,4-D and 2,4,5-T herbicide injury on most *Prunus* species (for example leaf and shoot distortion of apricot and plum, soft and red sutures on peach fruits) often have been mistaken for symptoms of virus diseases. The foliage yellowing, spotting, and cupping symptoms induced by other herbicides may also be mistaken for virus disease symptoms.

There are several useful criteria that can be used as aids in selecting the most probable causal factor for a given disease or disorder. Distinctive patterns (for example, rings, patterned mosaics, vein-associated flecking) are more frequently characteristic of virus infection than of nutritional disorders. Knowledge of the history of disease development in the orchard and observation of the pattern of occurrence are useful. Thus, if the symptoms have increased in severity gradually through several seasons in most trees of a planting, a nutritional disorder must be strongly suspected; whereas, if one or more trees in a planting develop well-defined symptoms while other trees of the cultivar display no symptoms, there is more likelihood that a virus is responsible. Moreover, although some viruses induce shock symptoms in one season only, virus diseases are most likely to affect the same trees in successive seasons, with or without spread to additional trees.

Assessment of such patterns of disease occurrence, with review of nutritional practices in the orchard, cataloging of herbicide applications in the vicinity, assessment of the likelihood of pollutant injury, and consideration of cold temperature experiences that may have been significant, should be a prelude to designation of any unfamiliar syndrome as a virus disease. Success in transmit-

ting the disease by budding or grafting from affected trees to healthy trees of the same cultivar is usually accepted as confirmation that the disease is virus-induced.

NUTRITIONAL DISORDERS THAT RESEMBLE VIRUS DISORDERS

C. G. WOODBRIDGE²

In 1946, Shear et al. (607) discussed nutrient element balance as a fundamental concept in plant nutrition and pointed out how seldom balance was attained. Not only is the intensity of nutrition important but also the balance between the elements. It is now accepted that optimum nutrient intensity and balance for a given plant vary with the species, cultivar, age, rootstock, and fruit load of the plant. The environment—season, light (quality and intensity), temperature (soil and atmospheric), diurnal fluctuations, and moisture—is also a variable factor affecting optimum nutrient levels. An incipient deficiency may cause a reduction of growth and in crop load in all plants, yet no symptom of the deficiency can be observed. Characteristic symptoms of deficiency are present when nutrient element balance is greatly disturbed.

Nutrient deficiencies in stone fruits are found in all areas. The most commonly needed element is nitrogen, and symptoms of nitrogen deficiency have been well characterized. We feel that the philosophy of nitrogen fertilization has become one of growth regulation rather than deficiency correction. Toxicity symptoms are not so well characterized, but in Washington they are being caused by the overzealous use of high-nitrogen fertilizers close to the trunks of young trees. Phosphorus deficiencies are not common in stone fruits. Potassium is deficient in many areas, and its need in stone fruits has received considerable attention. The results of study of the minor elements, both deficiencies and toxicities, have been fascinating because of the vast range of symptoms that may be observed and because of dramatic responses that the application of a small amount of a deficient element can effect (135, 138, 624, 704). Many deficiencies cause similar twig dieback symptoms. Gumming can be associated with many abnormal conditions. Because of their similarity, leaf patterns, due to mineral deficiencies or excesses, frequently can be identified only after extreme care (for example, the interveinal chlorotic patterns in peach caused by zinc or manganese deficiencies).

Insufficient chilling of trees in parts of an orchard may cause symptoms that may be confused with mineral deficiencies. In this category, a slow breaking of dormancy may be thought to be a boron or a zinc deficiency.

The relationship between the diseases and mineral nutrition has not been well understood nor particularly well classified. Some work has been done on the relationship of virus disease and nutrition. Christensen and Walker (141) in Utah have reported an interaction between virus infection and nutrient

² The names of contributing authors are given in their respective sections.

status of both sweet and sour cherries and between X-disease of peach and nutrition. Affected tissues contained more nitrogen and phosphorus than did noninfected trees. Wilks and Stewart (720) showed that the quality of the fruits on Lambert (*Prunus avium*) trees infected with little cherry virus may be adversely affected by zinc deficiency in the absence of recognized zinc deficiency symptoms.

Nutrient toxicities have been associated with abnormal leaf, twig, bud, and fruit development. Sometimes, the symptoms have been produced first in sand culture and later recognized in the field. Some of the toxicities have been due to high-nutrient levels in the soil or in irrigation water or in both. Arsenic toxicity resulted from the use of heavy applications of arsenic-containing sprays in the 1930's.

Symptoms alone cannot be used to distinguish an actual mineral deficiency from an induced mineral deficiency. For example, iron deficiency symptoms may be induced by high levels of aluminum. Also, multiple deficiencies may occur together. In these instances, diagnosis can be difficult. Positive identification of a deficiency or of an excess cannot always be made from isolated leaves. It is usually best to see the whole tree. Frequently, one also needs to use the results of tissue and soil analysis.

In the diagnosis of virus diseases and nutritional disorders, practical experience is very valuable. In the following sections, deficiency and excess nutritional problems that resemble virus diseases have been described. For more information, the reader is directed to the following publications: "Diagnostic Criteria for Plants and Soil" (135), "Fruit Nutrition" (138), "Hunger Signs in Crops" (624), and "Diagnosis of Mineral Deficiencies in Plants by Visual Symptoms" (704).

Nitrogen Deficiency (E. L. Proebsting, O. W. Davidson, and E. L. Proebsting, Jr.)

On a given soil in California, peach frequently shows symptoms first, but almond and Japanese plum show them a little later. European plum may not show any marked symptoms except reduced growth (544, 545). In the East, also, peach shows more marked symptoms of nitrogen deficiency than other stone fruits, but symptoms may be shown by all *Prunus* species. Chemical analysis of suitably selected leaf samples provides a useful measure of nitrogen status.

The pale green to yellow leaves of all plants grown in a soil low in available nitrogen is a well-recognized symptom of nitrogen deficiency (42). When the supply of nitrogen in the soil is limited, trees gradually redistribute a large portion of their nitrogen from old to new tissues. As a result, the mature and nearly mature leaves turn from dark green to yellowish green (fig. 151). The yellow in these leaves gradually increases, and the petioles and veins become tinged with red. By this time, linear growth of the twigs has been checked, and the leaf size is reduced. Moreover, the twigs and leaves are relatively hard. In

1 to 3 weeks, red or red-brown spots appear between the large veins of the leaf blades if the deficiency is acute. In advanced stages of nitrogen deficiency, many of the reddish spots develop into necrotic areas. Gradual abscission of the leaves from the base of the current-season growth toward the tip follows.

Terminal-shoot growth is frequently short and thickened, and the fruit buds are numerous. The percentage of flowers setting fruit is greatly reduced. In case of severe nitrogen shortage, the shoots also are short and slender, and the number of fruit buds is less than on normal trees. Potassium-deficient trees also frequently have small, light-green leaves similar to those on nitrogen-deficient trees. In the case of potassium deficiency, however, the terminal shoots are more slender and the internodes are long. The red spotting of leaves of severely affected trees is characteristic of deficiency of nitrogen. Lack of this essential element in the soil is the only deficiency that results in reddish discoloration of the leaf.

Fruits growing on trees low in nitrogen ripen earlier and are more highly colored than fruits on trees high in nitrogen (12, 178, 546, 602, 625). Free-stone peach fruits that are nitrogen deficient tend to taste more astringent and be more fibrous than fruits grown on trees that are high in nitrogen (546). Cheeks of nitrogen-deficient fruits may not fill out properly, and the flesh tends to cling to the pit.

Excess nitrogen delays fruit maturity and results in poorer skin color on fruit of a given firmness. Too much vigor can result in shading out lower interior fruiting wood. On young trees, nitrogen toxicity may result if large amounts of nitrogen fertilizer are applied directly at the trunk. Symptoms may include rolling of leaf margins toward the upper side of the midrib and necrosis that tends to be marginal. Chemical analysis will show very high levels in affected leaves.

Nitrogen deficiency is easily corrected by application to the soil of inorganic sources of nitrogen such as ammonium sulfate, sodium nitrate, ammonium nitrate, or organic material containing soluble nitrogen (544, 624).

Phosphorus Deficiency (*W. E. Ballinger*)

Phosphorus deficiency has been reported and described largely for peach (177).

When peach trees are grown in sand culture, the first symptom of phosphorus deficiency is the development of dark, dull-green to purplish-green foliage (fig. 149). The leaves show no puckering or crinkling along the midrib, and the blade is leathery, flat, and narrower than normal for the cultivar. When the phosphorus shortage is severe, the veins on the underside of the leaf and the petiole may be purplish. In orchards in North Carolina, leaves of trees deficient in phosphorus were dark green and, in late summer, became bronzed with a reddening of veins and petioles. Abscission of basal leaves of shoots was premature. Fruit from trees low in phosphorus are small, flat cheeked, cracked, astringent, low in soluble solids, bitter, early ripening, and have a bluish-red overcolor and overall poor quality.

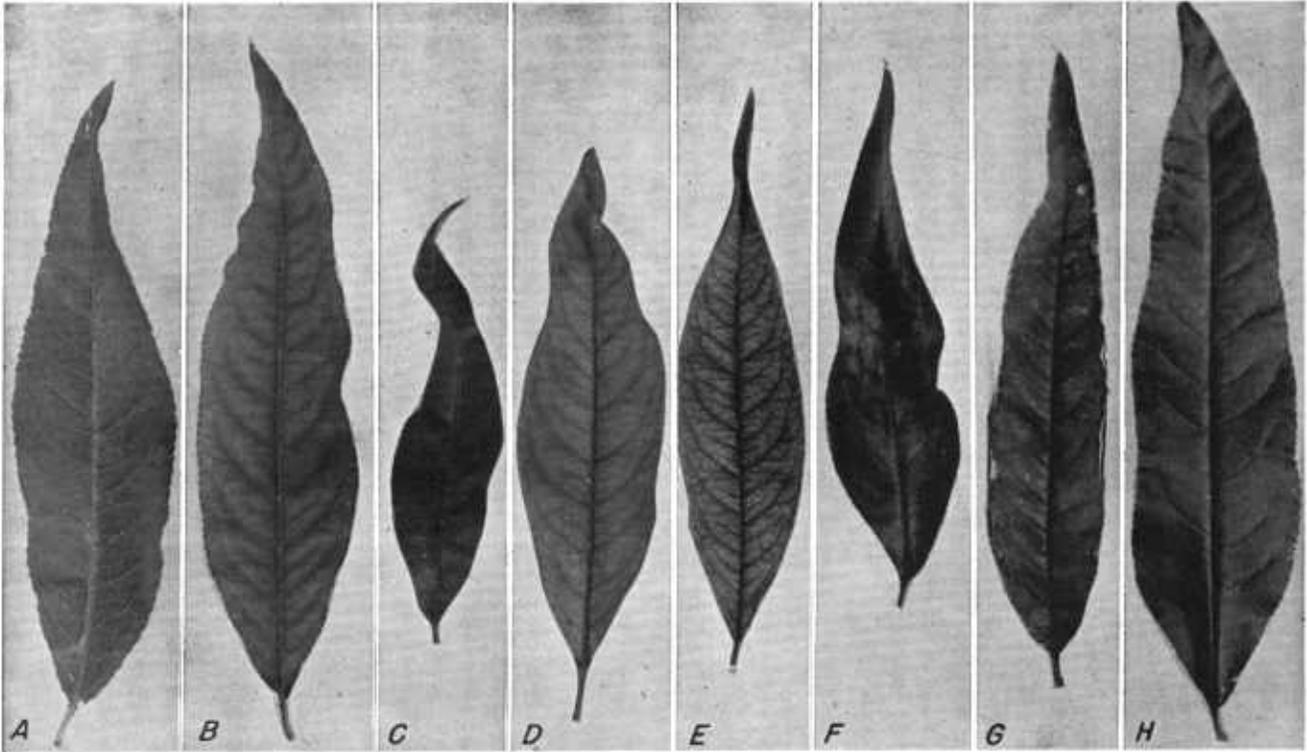


FIGURE 149.—Elberta peach leaves showing symptoms of mineral deficiency when trees were grown in sand cultures with indicated element withheld: *A*, phosphorus; *B*, manganese; *C*, nitrogen; *D*, iron; *E*, magnesium; *F*, calcium; *G*, potassium. *H*, Comparable leaf from tree grown on a complete nutrient.

Where phosphorus deficiency symptoms have been observed under field conditions, the trouble has been corrected by applications of phosphate fertilizer. Fertilizers containing phosphorus should be applied to ensure production of good cover crops in orchards.

Potassium Deficiency (*O. C. Compton*)

Potassium deficiency has been reported and described for almond, apricot, peach, plums (*Prunus domestica* and *P. salicina*), and sour cherry. No reports describing potassium deficiency of sweet cherry growing under field conditions have been found. Chandler (134) observed ". . . sweet cherry trees can obtain enough potassium from soils in which apple trees and some varieties of *Domes-tica* (European) plum trees will be injured seriously by potassium deficiency." Our observations in Oregon confirm these by Chandler. The symptoms have been described recently by Fritzsche et al. (224) for sweet cherry trees grown in nutrient solutions.

Lack of available soil moisture and heavy cropping accentuate the symptoms although young nonbearing trees often show severe deficiency. Removal of top-soil by erosion or grading operations often creates areas of acute deficiency.

In California (398, 400), leaves of affected almond trees are light green and show tipburn early in the season. Marginal scorch often occurs near the leaf tip, and leaves assume a boat-shaped appearance on vigorous shoots. More severe deficiency results in sparse foliage, decreased growth, terminal dieback, and lowered yields. Deficiency symptoms develop in leaves containing 0.7 percent potassium or less dry weight. Reports from California (398, 400) indicate that potassium-deficient apricot trees have leaves that are pale green, tend to roll or cup upward, and are sparse. Shoot growth is reduced. Marginal scorch and dieback occur when the deficiency is severe. Size of fruit and yields are reduced. The deficiency develops when leaf potassium is at 2.0 percent or less, a level considerably higher than for other stone fruits.

Potassium deficiency symptoms in peach are characterized by upward rolling, pale leaves in midsummer or thereafter, which increases in severity from basal to terminal leaves (138, 398, 400, 401) (fig. 150). With acute deficiency, older leaves become crinkled at the midrib, and marginal and submarginal necrosis develops, producing irregular margins and a shot hole effect (fig. 151). Slender shoots, reduced set of fruit buds, and small fruits that ripen prematurely accompany these leaf symptoms. These symptoms are not usually evident at leaf potassium levels above 0.75 percent, although the critical level is 1.0 percent potassium in midsummer leaves (401).

Symptoms of the deficiency on the French (Agen) prune cultivar (398, 400) show first as small, light-green leaves. Marginal necrosis or leaf scorch soon develops, especially with a heavy crop. Severe deficiency results in small, abnormally colored fruits and shoot dieback. Leaves of Italian Prune in New York (94) show midsummer loss of green color starting at the margins. Chlorosis



FIGURE 150.—Two-year-old peach trees: *A*, Adequately supplied with nitrogen and potassium; *B*, adequately supplied with nitrogen but deficient in potassium, showing narrow leaves with a tendency for the margins to roll upward.



FIGURE 151.—Peach leaves showing severe upward rolling and marginal necrosis characteristic of potassium deficiency, *left*, compared with normal leaf, *right*.

develops, and older shoot leaves roll toward the upper surface. Increasing deficiency produces marginal necrosis. Symptoms of moderate to severe deficiency on the Italian Prune in Oregon (fig. 152) vary from slightly mottled, light-yellow leaves that roll upward in August to leaves showing marked chlorosis with a slight to moderate marginal necrosis at harvest (fig. 152). Fruits are reduced in size. Deficiency symptoms usually develop on all varieties at or below 1.0 percent potassium in midsummer.

Leaf rolling combined with a light-green color on the older leaves are early symptoms of the deficiency in sour cherry (224, 239). In advanced stages the leaf margins on the lower surface first become bronze, and then scorched. Small leaves and fruits, shortened slender growth, and greatly reduced yields are characteristic of severely affected trees. The symptoms develop at or below 0.75 percent potassium in midsummer leaves.

Potassium deficiency was corrected easily in prune, peach, or cherry orchards growing in coarse- to fine-textured soils of the Coastal Plains, the North Atlantic, and the north central regions. Application of 1 to 6 pounds per tree of potassium chloride, depending on tree age, produced satisfactory recovery (138). Mulches of straw, hay, or manure also increased leaf potassium, often by several times.

In areas of low to nonexistent summer rains, such as the tree fruit areas of the Pacific Coast States, improvement usually requires heavy applications of potassium fertilizers. In California (398, 400, 401), at least 25 pounds of potassium sulfate per tree is necessary, except on certain acid or sandy soils where half this amount produced satisfactory results. Italian Prune trees in Oregon required 12.5 to 15 pounds per tree of potassium chloride to produce recovery. Smaller amounts applied annually were ineffective. The larger amounts were effective for several years. Deep placement of potassium chloride increased the response in California (400). The high fixation of potassium in the residual acid soils of the Willamette Valley has been bypassed effectively by use of potassium nitrate foliage sprays applied 6 to 8 weeks after bloom.

Calcium Deficiency (*Norman F. Childers*)

Peach is the only stone fruit species known to suffer from calcium deficiency in orchards (284).

When peach trees are grown in a nutrient solution lacking calcium, the first symptom of the deficiency is usually decreased terminal growth; the older leaves may be of normal size, but the young leaves are usually smaller than normal (fig. 149). The leaves are dark green, and no chlorosis is evident. Later, a large chlorotic, then necrotic, area develops in the center of some of the younger leaves, particularly on the lateral spurs and shoots (fig. 153). It is a large, characteristic spot involving tissue on either side of the midrib. The older leaves later show marginal chlorosis and breakdown. Finally the leaves drop at the shoot tip; then the ends of the shoots may die back. Under controlled nutrient conditions, calcium deficiency is shown first in peach foliage, where

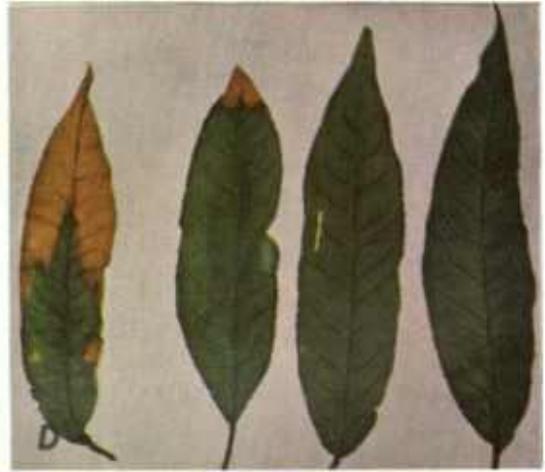
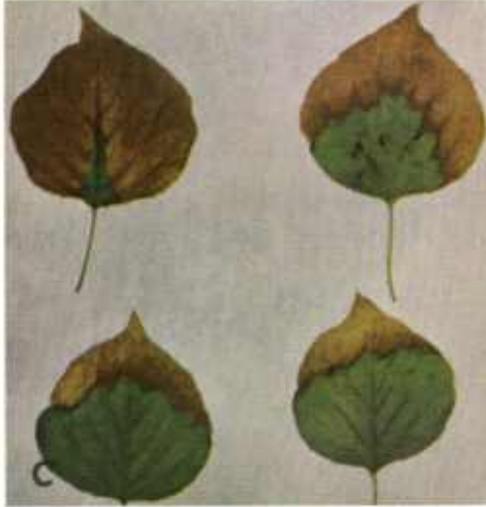
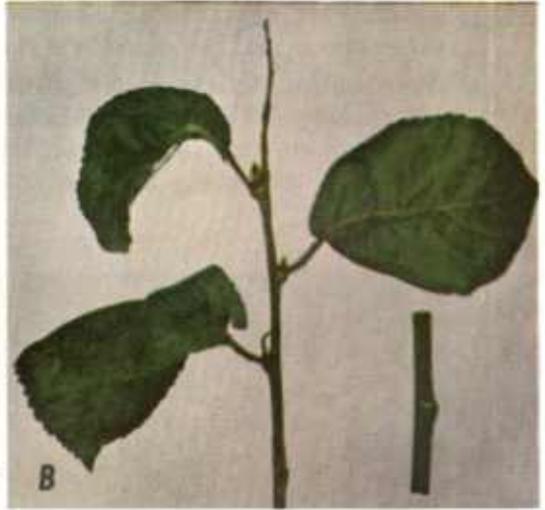
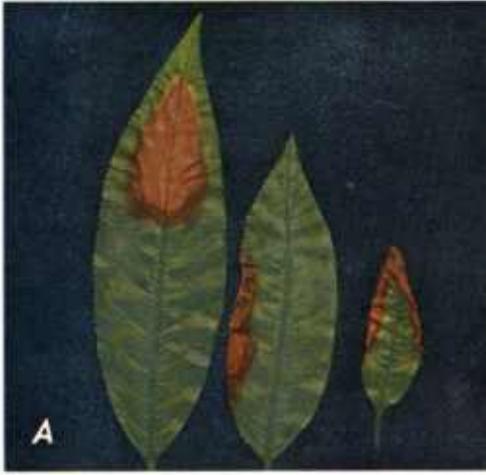
potassium and magnesium are relatively high. In the field, calcium deficiency has greatly curtailed root extension of peach.

Magnesium Deficiency (*W. E. Ballinger*)

Magnesium deficiency on this continent has been noted only on peach. When peach trees are grown in a magnesium-deficient nutrient solution, the older green leaves may develop light-gray or fawn-colored spots in the area between



FIGURE 152.—Italian Prune branch showing fall leaf symptoms of potassium deficiency in Oregon: chlorotic blotches and slight to moderate marginal necrosis.



the veins (fig. 149). In severe cases, the spots may enlarge and take in the edges of the leaves (fig. 154). In early stages, leaves may show a chlorosis somewhat like the early stages of iron chlorosis. The leaves of severely affected trees shed from the base toward the tip, and only a few of the youngest leaves may remain near the growing tip. As the green fades out between the veins, the leaf tissue takes on a papery, gray appearance, and the dead spots may enlarge and envelop the outer margin of the leaves. Under controlled nutrient tests in sand culture, magnesium deficiency appears first where calcium or potassium is relatively high in relation to magnesium. Two types of magnesium deficiency symptoms on leaves in orchards in North Carolina Sandhills may develop: (1) marginal and interveinal necrosis (described above) which develops quickly, followed by leaf drop and (2) marginal chlorosis (fig. 155, *F*) (427). Symptoms are more prevalent on trees bearing large crops of fruit than on trees with small or no crop loads (138).

For long term correction on sandy soils, apply magnesium sulfate up to 2.5 pounds per tree per year. Use dolomitic limestone to correct acid soil conditions. Spraying the leaves with a solution of magnesium sulfate (epsom salts) may give a quicker response. Avoid applications of excessive amounts of potassium fertilizers or low-magnesium limes.

Manganese Deficiency (*Omund Lilleland and K. Uriu*)

Manganese deficiency has been found on almond, apricot, sweet cherry, sour cherry, peach, European plum, and Japanese plum.

A distinct pattern has been observed on the leaves of all stone fruit species in which manganese deficiency has been noted. The midrib and main veins with adjacent bands of tissue of varying width remain green, whereas the interveinal and peripheral areas of the leaf edges may be chlorotic (fig. 149; fig. 155). Generally chlorosis does not occur until the young leaves have attained full size, and it becomes more pronounced on the older leaves as the season progresses. Only in very severe cases is there a dwarfing of shoot growth and the development of a spot necrosis into a shothole condition of the leaf (fig. 156). Non-typical symptoms may occur when both zinc and manganese or iron and manganese are deficient and the correction of the zinc or the iron may reveal the more typical manganese symptoms. Multiple deficiencies of manganese, zinc, iron, and even other elements may infrequently occur, in which cases symptomatology alone is inadequate to diagnose the trouble.

There appears to be an excellent correlation between the results of leaf analyses and the manganese status of fruit trees. Studies with many kinds of

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FIGURE 153.—*A*, Peach leaves showing symptoms of calcium deficiency. *B*, French prune twigs and leaves showing injury due to boron excess. *C*, Apricot leaves and *D*, peach leaves showing injury due to sodium excess. *E*, Peach leaves and *F*, peach branches showing arsenic injury.

fruit trees in many places have shown a fairly constant critical value of 15 to 20 ppm manganese on a dry-leaf basis in midsummer. There are fewer data on toxicity or excess manganese than there are on manganese deficiency, but the value for excess is probably close to 1,000 ppm in the leaf.

Manganese deficiency is easily corrected by foliage sprays in early spring. An earlier recommendation of 5 pounds of manganese sulfate has been reduced

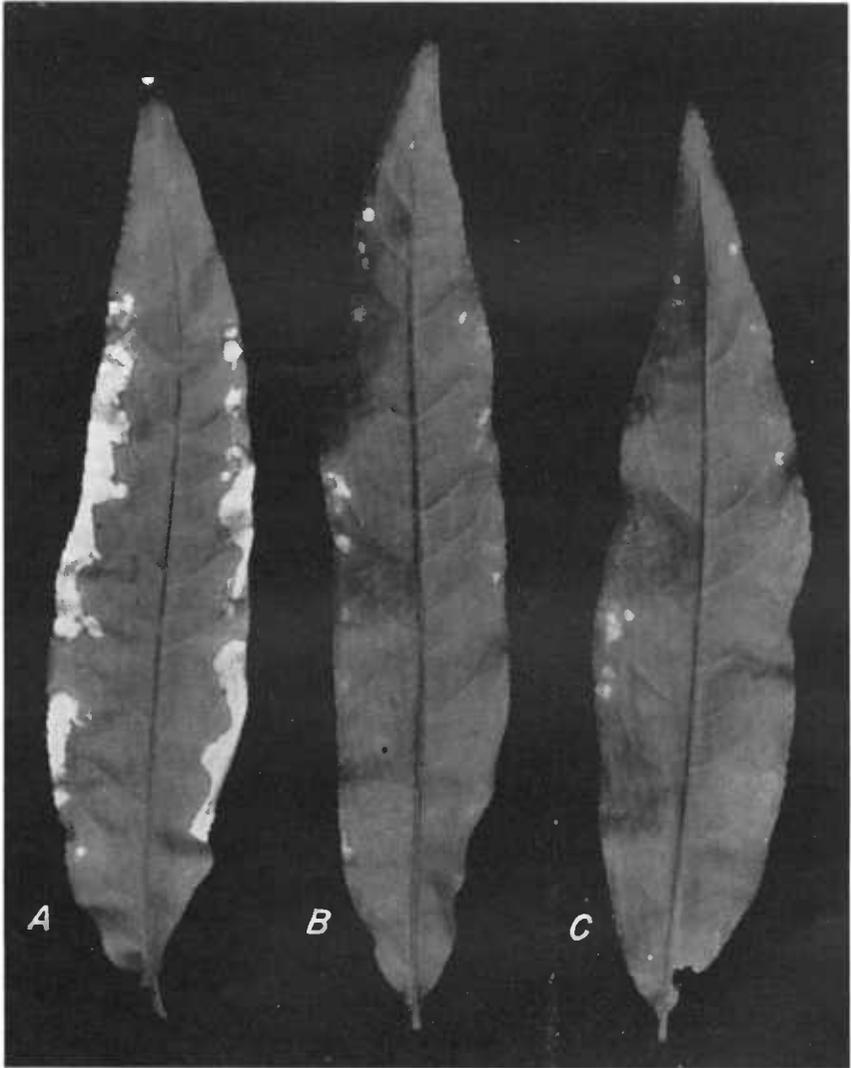


FIGURE 154.—Leaves from a peach tree grown in a magnesium-deficient nutrient solution: *A*, Severe symptoms; *B* and *C*, mild symptoms.

to the present 2 pounds of manganese sulfate in 100 gallons of water for California orchards. MnEDTA at $\frac{1}{2}$ to 1 pound per 100 gallons of water has also given good correction. If annual spraying is inconvenient over a number of seasons, a correction may be obtained in alkaline soil by an application of sulfur.

Iron Chlorosis (*Walter J. Kochan*)

A number of factors influence the amount of iron required to prevent chlorosis, so a precise sufficiency level cannot be given. However, the susceptibility of stone fruits to lime-induced iron chlorosis from most susceptible to least susceptible has been assigned as follows: peach, sweet cherry, plum, apricot, and almond.

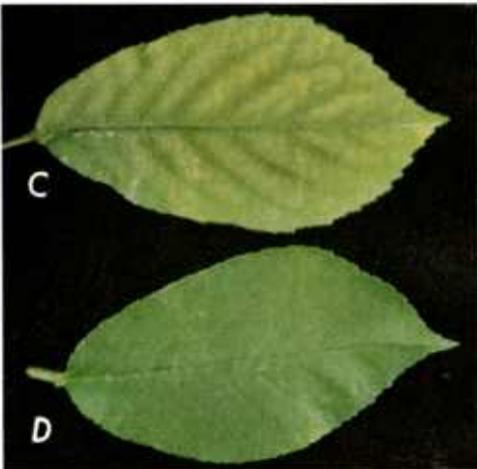
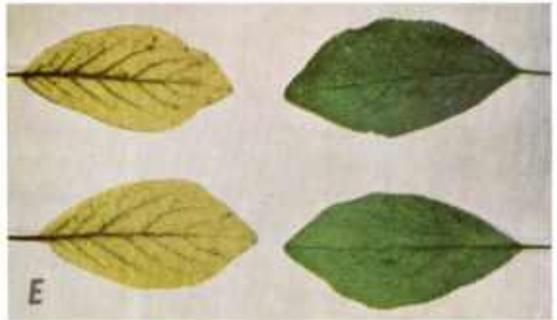
Foliar symptoms of iron chlorosis are a reduction in chlorophyll content and a paling of the green of the leaves (fig. 157). As the disturbance becomes more acute, the areas between the veins become yellow; if not corrected, the deficiency may cause the entire leaf to become white, progressing to dead leaf margins (fig. 149). Severely chlorotic leaves are smaller than nonchlorotic leaves. With very severe deficiencies, the small branches will die, beginning at the tips. The young leaves that emerge are chlorotic; if iron is made available, they will become green. Mature, green leaves do not become chlorotic. Although chlorosis is not specific for any element, this complex of symptoms is such that the deficiency may be identified. Diagnostic techniques (588) used in conjunction with the foliar symptoms aid in confirming the deficiency.

Many methods have been recommended to cure lime-induced iron chlorosis. All, apparently, have some merit, but none is without drawbacks. Dilute sprays of iron sulfate early in the season may correct the deficiency but must be repeated to include developing shoots. Injecting iron salts into bored holes in the trunk or large limbs was probably the most successful treatment before synthetic iron chelates were introduced but were time consuming and destructive of tissue. An iron chelate was first used successfully in culture solution (331); its agricultural potential was demonstrated in citrus (627) and exhaustively studied (703); and its beneficial effects on peach on a calcareous soil were demonstrated (379). The principal function of the chelating complex seems to be that of preventing the formation of insoluble iron salts in the soil.

Zinc Deficiency (*David R. Walker, R. L. Smith, and W. H. Chandler*)

Chandler (133) listed sweet cherry as the most susceptible of the stone fruits to zinc deficiency. Other stone fruits affected, in order of susceptibility, seem to be Japanese plum, European plum, peach, apricot, almond, and sour cherry.

The deficiency is often referred to as "little leaf." Leaf symptoms, more than any other symptoms, have been emphasized in describing the deficiency syndrome (fig. 158). Leaves that develop in early spring at the apex of a long shoot may be much smaller than normal (about 1 inch long and $\frac{1}{4}$ inch or less wide). Shortened internode growth results in leaves crowded very closely into tufts at



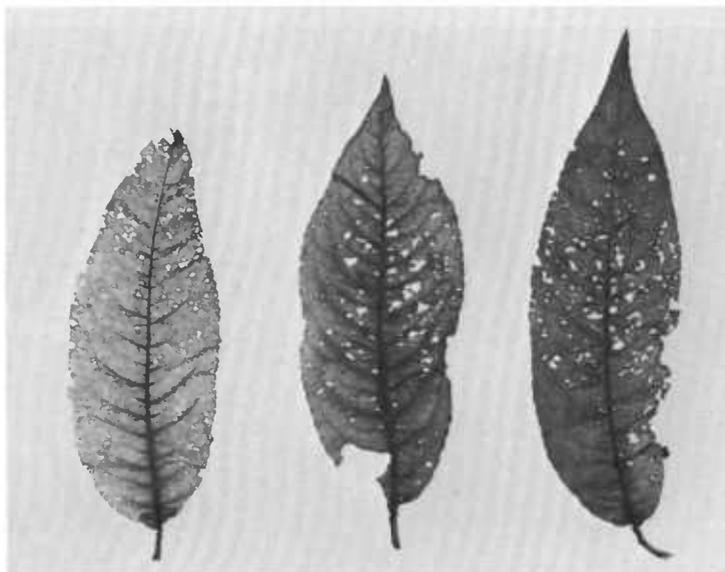


FIGURE 156.—Shothole in peach leaves associated with a severe manganese deficiency.

the terminal end, resulting in the disease sometimes being called rosette (fig. 159).

In the early stages, irregular splashes of yellow or chlorotic areas occur between the veins. These isolated chlorotic areas gradually coalesce until continuous bands of yellow extend from near the midrib to the outer edge where they form a continuous chlorotic leaf margin (663). Most of the leaves have wider green areas along the veins and around the yellow areas than manganese-deficient leaves have, and the yellow tends to be paler and less golden. Leaves at the end of some summer shoots may be creamy yellow, with very little green even along the veins. Red or purple blotches sometimes occur on these chlorotic areas. Tissue in these spots may dry up and drop out, producing a shot hole effect. In some varieties of plums, stippled foliage occurs due to small chlorotic areas scattered over the leaf. Wavy leaf margins and crinkling of midribs occurs especially on the tip leaves. In apricot leaves, the margins curve upward forming a cup-shaped effect. Premature leaf fall may also occur. Short

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FIGURE 155.—*A*, Peach leaves showing a gradient of manganese deficiency symptoms; *B*, Montmorency cherry leaf showing manganese deficiency symptoms; *C*, Montmorency cherry leaf from a manganese-deficient branch; and *D*, a comparable leaf from a branch cured by injection of manganese sulfate; *E*, prune leaves showing symptoms of iron deficiency, *left*, compared with normal leaves, *right*; *F*, peach leaves showing symptoms of magnesium deficiency.

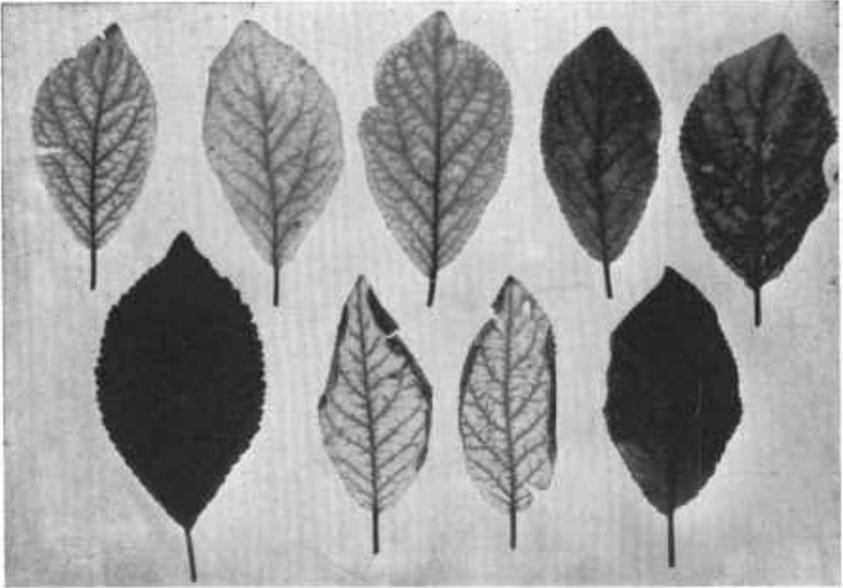


FIGURE 157.—Leaves of French (Agen) prune showing characteristic pattern in reduction of chlorophyll due to lack of available iron; normal leaf, *lower left*.

twigs, undeveloped buds, small leaves, and early leaf fall result in bare limbs and dieback, which is common on affected trees. Weak wood which fails to harden and, in extreme cases, death of the tree occurs as a result of this deficiency (fig. 160).

Zinc-deficient trees often produce small yields of undersized or misshapen fruit. Fruit in the top of the tree and especially toward the end of the branches, tend to be progressively much smaller than normal. Deficient peach and Japanese plum trees produce more flattened fruit than normal. Mature peaches from zinc-deficient trees break down sooner after harvest than those of normal trees. Apricot fruits are less flattened than normal, more nearly round, and greatly reduced in size to that of medium-sized cherries. Flat, pointed peaches have been observed on zinc-deficient trees.

The leaf and fruit symptoms may appear on just one or a few branches with the rest of the tree appearing healthy. The symptoms sometimes resemble X-disease (663). Distinguishing features are that X-disease does not cause a reduction in leaf size, the leaves may turn downward, and they may drop off prematurely.

Many soils have a great capacity to render zinc unavailable to trees. In some of the Pacific Slope soils this fixing capacity is so great that 1,000 to 1,500 pounds of zinc sulfate applied to an acre of soil may not supply the necessary ounce or two of zinc to an acre of trees. In Florida, however, soil applications

of one-half pound to 5 pounds per tree of $ZnSO_4$ were effective in eliminating zinc deficiency symptoms (189).

Dormant sprays of $ZnSO_4$ applied as a separate spray just before the buds start to swell, at the rate of 10 to 50 pounds per 100 gallons, has been the main control measure for peach, almond and Japanese plum. The amount required depends on the severity of the symptoms. Apricot does not respond as well as peach to dormant spraying, and therefore stronger solutions must be used.

Foliar sprays of $ZnSO_4$ have been used successfully. Up to 8 pounds per 100 gallons without lime has been used without injury reported. Hydrated lime, calcium caseinate, and calcium hydroxide have been used with $ZnSO_4$ to reduce plant injury. A solution of 10 pounds $ZnSO_4$, 5 pounds hydrated lime, and 100 gallons of water applied any time during the growing season but preferably in the early spring has been used successfully (87). Spraying three to four consecutive seasons may be necessary and every other year thereafter to control severe deficiencies. Foliar sprays have been applied at the rate of 12 to 25 pounds $ZnSO_4$ and half as much hydrated lime in 100 gallons of water. Foliar injury is more likely to occur under humid conditions. Foliar applications of 5 pounds of $ZnSO_4$ plus 2.5 pounds of calcium hydroxide per 100 gallons was more effective in controlling the deficiency than soil applications of 500 pounds $ZnSO_4$ per acre or 25 pounds $ZnSO_4$ per 100 gallons as a dormant spray (426).

Synthetic chelated zinc materials ($Na_2Zn-EDTA$ or $NaZnH-EDTA$) applied to the soil (1 pound per peach tree in June) was reported as being more effective than dormant $ZnSO_4$ sprays applied at the rate of 12 pounds per 100 gallons (426). Natural chelated zinc has been effective as a foliar spray at the rate of 5 pounds per 100 gallons. This material must be applied early in the spring while the fruit is small in order to avoid a fruit residue problem.

Although trunk injections of $ZnSO_4$ and zinc-coated nails driven into the trunk have been recommended as control measures, they are not being used much at present. Considerable labor is involved and some tree injury has occurred from these practices.

Copper Deficiency (K. Urie and Carl J. Hansen)

In this country, copper deficiency has been observed on plum (*Prunus domestica* and *P. salicina*) and almond. In South Africa, the deficiency has been observed on plum (*P. salicina*), apricot, and peach. In Australia, it has been observed on plum (*P. salicina*) and, in Great Britain, on almond and sweet cherry.

One of the characteristic symptoms of copper deficiency is shoot dieback. In early summer terminal growth ceases, mottling and chlorosis (fig. 161) may develop, and the terminal leaves become brown at the tips and margins, giving the shoots a withered appearance (fig. 162). Eventually, many of the terminal



leaves fall as the tips of the shoots are killed back (fig. 158 pl. 9, *D*). Sometimes in the current season, but mostly in the following season, the lateral buds below the dieback are forced into growth. These shoots die back in the same manner, and the repetition of the dieback over a period of years causes affected trees to have a bushy, stunted appearance.

The bark of affected trees may be rough and corky, but the extent of such injury may depend on the species and variety. In plum, hard, rough, irregular, and dark-colored fissures develop in the bark, and reddish-brown gum often exudes from the ruptured fissures. In almond, severe rough bark also occurs, and considerable gumming of trunks and lower parts of main branches occurs in late winter and early spring in Nonpareil (fig. 163) and Ne Plus Ultra. Trees of Texas (Mission) almond show dark, rough bark but seldom produce gum.

The copper level in the leaves from deficient trees is, in general, about 3 ppm or less.

Plum trees showing symptoms of copper deficiency have been cured by application of copper sulfate to the soil and by spraying the young leaves with Bordeaux mixture. For copper-deficient almond, good correction has been obtained by application of copper sulfate and CuEDTA to the soil or by foliar spray of Bordeaux mixture or CuEDTA. For diagnostic purposes, copper sulfate can be injected into holes bored in the trunk.

Soil applications have generally varied from 1 pound to 5 pounds of copper sulfate placed in the bottom of a circular trench or spaded in around the base of each tree, the exact quantity depending on the size of the tree and the fixing power of the soil. CuEDTA, $\frac{1}{4}$ to 1 pound per tree as a broadcast treatment, has been used on almond with success. Sometimes response from soil treatments may not become evident the first season.

Foliar sprays in early spring with Bordeaux mixtures of 5-5-100 to 10-10-100 have been reported to give good correction on plum and almond. Almond has even responded to Bordeaux as dilute as 1-1-100. Also with almond, CuEDTA at one-quarter pound per 100 gallons of water has been effective. Higher rates of CuEDTA were found to be phytotoxic to the leaves.

The holes used for the injection of dry copper sulfate are spaced about 4 inches apart around the trunk, and excessive injury is avoided by keeping the material away from the cambium and the bark. This is done by inserting the copper sulfate in gelatin capsules or forcing it through a tube. Wooden plugs can then be driven into the holes.

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FIGURE 158.—*A-C*. Plant parts showing symptoms of zinc deficiency: *A*, Peach; *B*, apricot; *C*, sweet cherry. In each illustration deficient parts (*left*) are compared with healthy parts (*right*). *D*, Prune shoots with tip killing due to copper deficiency.



FIGURE 159.—Limb on peach tree affected by zinc deficiency showing typical rosetted growth.

Boron Deficiency (*C. G. Woodbridge*)

Most stone fruit species are affected by boron deficiency. Marked deficiency is characterized by the dying back of new shoots usually in the spring but sometimes later in the season (204, 277, 278, 431). The buds may swell in early spring then die and slough off. Whole trees may die, but this usually takes several years (figs. 164 and 165). Lateral shoots may develop below the dead portion, giving the branch a witches'-broom effect. Dieback due to boron deficiency may be mistaken for winter injury; however, in early spring with boron deficiency the cambium is white, whereas with winter injury the cambium is discolored.

Leaves on shoots affected with dieback may scorch, curl, and die (fig. 166). Sometimes rosettes of leaves may occur and these symptoms may be confused with zinc deficiency. Boron-deficient sweet cherry leaves are narrow and frequently have irregular serrations (fig. 167). Observations and diagnosis can readily be confirmed with chemical analysis of the affected tissue. If the boron



FIGURE 160.—Sweet cherry tree in Utah, showing small chlorotic leaves and severe dieback due to zinc deficiency.

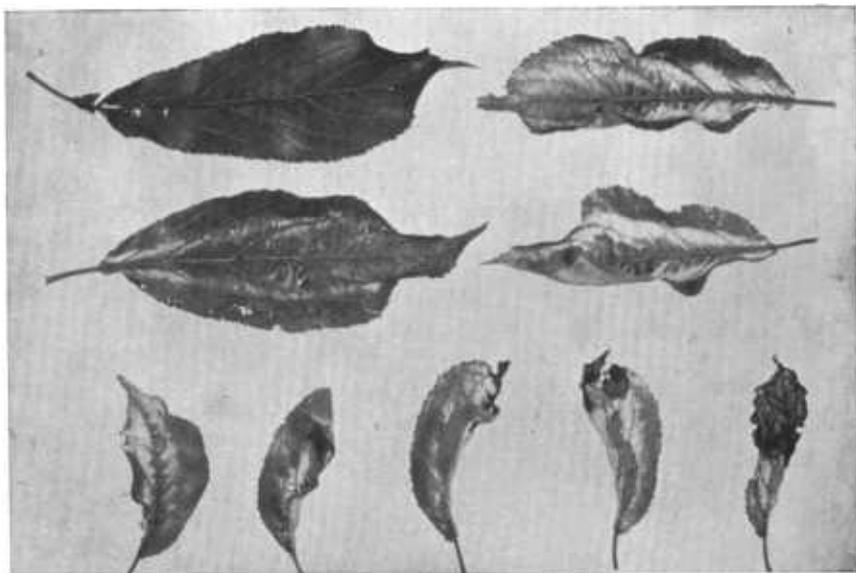


FIGURE 161.—Leaves of Santa Rosa plum showing chlorosis and scorching characteristic of copper deficiency.



FIGURE 162.—Copper-deficient almond shoots showing tip and marginal necrosis of terminal leaves, that give the shoots a withered appearance.

content of leaves is below 20 ppm on a dry-weight basis, boron deficiency can be suspected.

Symptoms of boron deficiency appear first on fruit, but seldom is the entire crop affected. Fruit may show surface cracking, shriveling, and malformation (101). Water-soaked areas in the flesh are common in prunes and plums, but internal browning and corky tissue are found in apricot, and gumming occurs on almond fruit.

Preventive sprays are used in most areas where boron deficiency is known to occur. Soil applications of boron compounds have also successfully prevented boron deficiency.

Boron-Excess Injury (*C. G. Woodbridge*)

Most stone fruit species are affected. The most striking feature associated with boron toxicity in apricot is the greatly enlarged nodes on 1- and 2-year-old twigs (fig. 168). Internodes may be somewhat shorter than usual and gumming may occur. The bark is frequently rough and may slough off. Tips of shoots may die during the summer, and the leaves near the tip may turn black and fall. Small twigs, leaf petioles, and the lower side of leaves may show necrosis. Fruit ripens early. Scablike protuberance and corking of the fruit are associated with high boron.



FIGURE 163.—Scaffold limb of Nonpareil almond showing rough bark and gumming characteristic of copper deficiency.

Dieback of cherry twigs (fig. 169) is also caused by an excessive amount of boron. Gumming is common. Growth is negligible. Leaves affected with boron toxicity may have necrotic areas near the midrib or in the lamina of the leaf (fig. 170). Opening of the flower buds is delayed.



FIGURE 164.—Young peach tree showing moderate dieback in the spring on shoots of the previous year's growth due to boron deficiency.



FIGURE 165.—Terminal dieback on sweet cherry due to boron deficiency. The terminal bud started to grow in the spring but died later, following partial defoliation.

Small necrotic spots on the underside of the main rib of peach, cherry, and almond leaves are common symptoms of boron toxicity (fig. 171). In severe cases leaves turn yellow and drop prematurely. Twigs are frequently cankered and gummied. Fruits are malformed and the kernels develop poorly (fig. 172).

On prune trees affected with boron toxicity the nodes swell, but not as much as on apricot, and the bark cracks and curls (fig. 168 and fig. 153, *B* and fig. 173).

Spurs develop slowly and the leaves are small. Leaves feel coarse, the midrib is thick, and many small necrotic patches appear and fall out. The latter symptom is more striking in peach. Fruit is small and ripens prematurely. Diagnosis should be confirmed by tissue analysis. In leafy tissue, a boron content of over 80 ppm on a dry-weight basis is suspect.

Control measures depend upon the quality of the irrigation water. If the water is low in boron, excess boron may be leached out.



FIGURE 166.—Boron deficiency in apricot leaves. The midrib is thickened, and the leaves show a characteristic boatlike form.

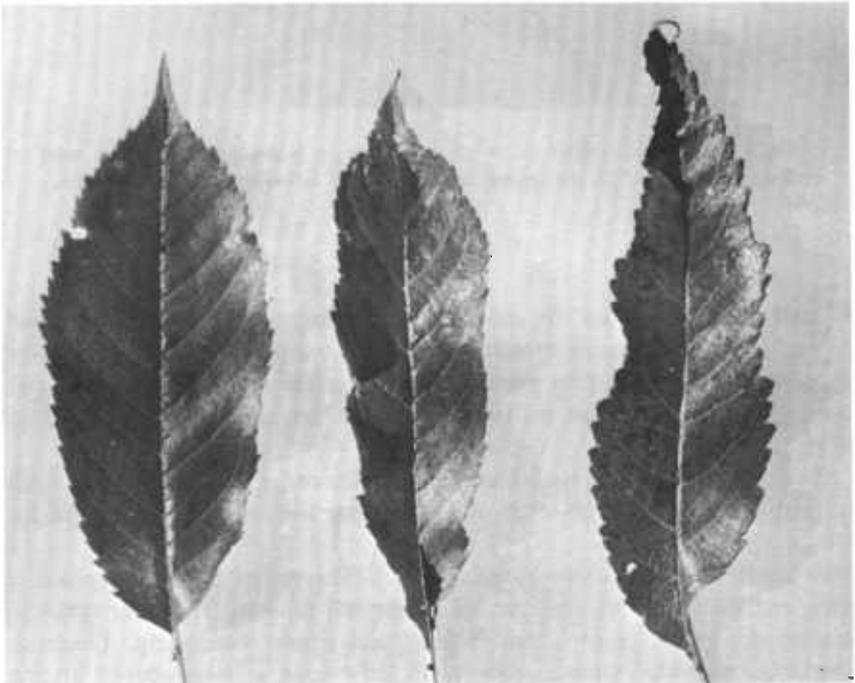


FIGURE 167.—Leaves from sweet cherry tree growing in a low-boron sand culture, compared with normal leaf, *left*. Affected leaves are narrower than normal and serrations are irregular.

Chloride-Excess Injury (*R. M. Carlson and Omund Lilleland*)

Almond, apricot, sweet cherry, peach, Japanese plum, and European plum are known to be injured by excess chloride.

Trees growing in soils with moderate salt concentration may be stunted without showing specific toxicity symptoms (286). This effect can be cumulative, causing a slow, progressive decline. When exposed to high concentrations of chloride, specific symptoms will develop; apricot leaves develop a cup shape caused by upward rolling of the margins and the leaf tips, and leaf margins scorch (40, 41, 397, 681). Almond, plum, and prune show tip and marginal leaf scorch (41, 681). Excess chloride in cherry causes small, pale-green leaves, marginal necrotic scorch, slender stems, and some leaf abscission (186, 511). The symptoms in peach are chlorosis, tip and marginal leaf burn, leaf



FIGURE 168.—Boron toxicity symptoms on apricot branch, showing enlarged nodes and some stunting and killing of twigs.



FIGURE 169.—Dieback of sweet cherry twigs caused by an excessive amount of boron.

abscission, and dieback of branches (41, 191, 285, 286, 397). Symptoms may develop on only one side of a tree (397). Soil analysis is not always a reliable tool for diagnosis of chloride injury. Leaf analysis can be used to confirm diagnosis by visual symptoms. Brown et al. (100) found the lower limit of chloride content in leaves showing injury was about 1.0 percent (dry-weight basis) for apricot and peach, 0.6 percent for prune and plum, 1.2 percent for Nonpareil almond, and 1.8 percent for Texas almond. Deciduous trees generally show some detrimental effect if foliar chloride concentrations exceed 0.3 percent.

The form of chloride in the root media can greatly influence the amount of chloride damage to the tree. Wadleigh et al. (681) showed that excess chloride damage to stone fruits is much more severe when calcium chloride is the source compared with isosmotic solutions of sodium chloride.

Excess chloride injury can also occur through foliar absorption of chloride from sprinkler-applied irrigation water. Ehlig and Bernstein (197) showed that sodium or calcium chloride in water sprinkled on trees caused necrosis of leaf tips and partial defoliation of almond and plum. Twig tips and fruiting spurs of almond, plum, and apricot were partially killed by sodium or calcium chloride in this experiment.

Leaching the soil to remove excess chloride and use of low chloride irrigation water are suggested as control measures.

Sodium-Excess Injury (*Omund Lilleland*)

Almond, apricot, sweet cherry, peach, and Japanese plum are known to be injured by excess sodium.

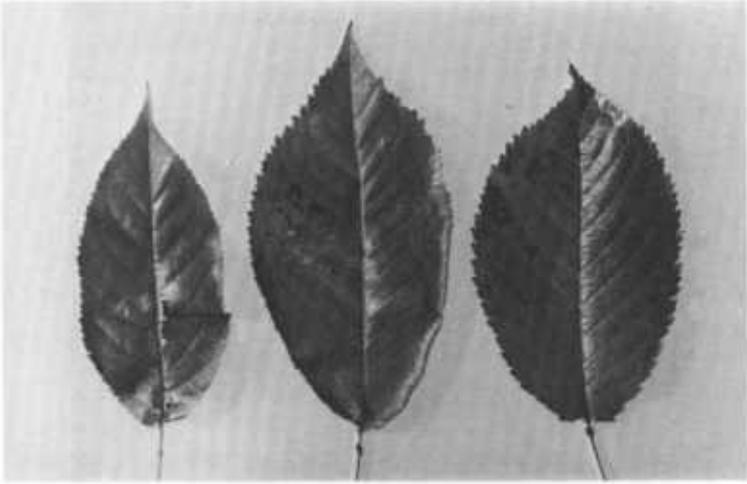


FIGURE 170.—Sweet cherry leaves showing symptoms of boron toxicity. Necrotic areas are commonly near the midrib but may be elsewhere in the leaf lamina.



FIGURE 171.—Severe boron toxicity on young peach shoot showing necrotic spots along the stem and petioles.

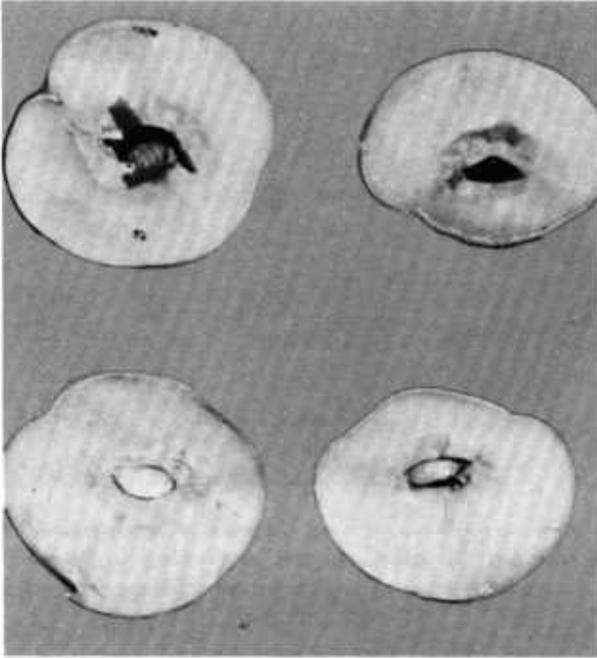


FIGURE 172.—Boron toxicity in peach fruit, showing malformation and poor kernel development.

Several types of leaf scorch (397, 399) are caused by excess sodium. Burning at the tip of the leaf is frequently characteristic of early stages of injury (fig. 154, C and D). Trees may be stunted but remain alive for a number of years, or they may die in one season. Analysis of scorched leaves often shows 0.5 percent or more of sodium in the dry matter. A low potassium content is frequently associated with a high sodium content. Chemical analysis of the roots of injured trees reveals accumulation of sodium in the wood. Injury may occur on soils which contain as low as 250 ppm of total soluble salts.

Leaching the soil with nonsaline irrigation water and additions of gypsum, sulfur, and similar materials, depending upon soil conditions, are suggested as corrective measures.

Arsenic Injury (*N. R. Benson*)

All fruit species show marked reduction in growth when planted on soils containing excess arsenic. The effect is believed to be toxicity to the roots. In addition to growth retardation, some species also exhibit symptoms on the leaves. Peach and apricot are most easily injured; sweet cherry is only moderately damaged; and plum (*Prunus domestica*) shows no foliage symptoms

even when planted on soils that contain so much arsenic that growth is severely restricted.

As a general rule, arsenic toxicity symptoms on peach do not become apparent until midsummer and appear first on the older or basal leaves. The younger, terminal leaves frequently remain normal for a while or throughout the season. The first indication of injury is the development of brown to reddish-brown spots along the leaf margins. These spots also appear in the leaf blade between the veins. Later these necrotic islands fall from the leaf, leaving irregular-shaped holes—a shot hole effect similar to that caused by the X-disease virus (fig. 153, E). In fact, the symptoms are so much alike, the arsenic injury has often been mistaken for X-disease (403, 566). The injured tissue around the leaf margin may drop out, leaving a ragged margin. In severely affected trees, defoliation takes place later in the season, but any fruits on a tree remain attached and ripen somewhat prematurely (fig. 153, F). The fruits do not attain full size and are astringent in flavor. A similar injury results from arsenical sprays applied to peach trees.

Symptoms in apricot resemble those in peach but with less tendency for defoliation. Symptoms on cherry are much less severe and are usually confined to interveinal browning.

At the present time there appears to be no practical method of removing arsenic from the soil. Therefore, emphasis has been placed on reducing arsenic



FIGURE 173.—Boron toxicity on 2-year-old prune twigs on which bark cracked, curled outwards, and in some cases was sloughed off. Normal twig on *left*.

absorption by the tree and of increasing the tolerance of trees for arsenic. This has been accomplished by continued high nitrogen fertilization and occasional applications of zinc sulfate to the soil. The addition of sulfur to the soil in addition to the zinc sulfate on alkaline soils increased the effectiveness of the treatment (659). Also effective, especially on alkaline or calcareous soils when soil application of zinc sulfate was least effective, were sprays of zinc EDTA and other chelates (26). The rate of application of zinc sulfate for soil application was 5 to 8 pounds per tree. Sprays of zinc EDTA consisting of 2.5 pounds of material per 100 gallons were applied in June and July. Applications of zinc sulfate to the soil may be repeated as necessary but should not be required more often than every 3 to 4 years. Sprays may be required annually.

HERBICIDE INJURIES THAT RESEMBLE VIRUS DISEASES

W. V. WELKER

Many of the symptoms of herbicide injury are similar to those caused by virus disorders and mineral deficiencies. In general, these injuries are the result of incorrect use of herbicides. Injury from an herbicide may occur when the desired dosage is exceeded through error, as when the sprayer is improperly calibrated, or when an inaccurate delivery system is used. Although a twofold to threefold tolerance factor to avoid injury to crops is usually present in recommendations for the use of herbicides, foliar symptoms may occur before the critical level is reached at which yields are reduced. Injury seldom occurs when recommendations for herbicide use are carefully followed.

Young trees with small root systems are more susceptible to herbicide injury than older trees with well-developed root systems. Many of the recommendations stipulate that applications of herbicides should be delayed for varying periods from the time the trees are planted. Injury is also more apt to occur on light-textured soils than on heavy-textured soils. The amount of clay and organic matter in a soil influences the availability of an herbicide and its rate of downward movement with water.

The amount and distribution of rainfall can also influence the appearance of injury symptoms. Frequent, light showers alternating with dry periods usually result in less downward movement of herbicides than a heavy, soaking rain.

Deep disking just prior to applying an herbicide can result in injury with compounds that are normally safe. Rains may wash the herbicide into the base of the cut, thus placing the herbicide deeper than normal and also concentrating it. Many of the herbicides used in orchards rely upon depth protection for most of their selectivity. Deep disking can eliminate much of the depth protection.

Injury symptoms vary for a given herbicide depending upon the amount of herbicide available at any one time. At a low availability level an herbicide may show bleaching of the leaf color in a characteristic pattern. At a high availability level it may show none of this; burning or scorching of the leaf may be the only symptom exhibited.

In evaluating plant injury symptoms, all aspects of the situation should be carefully considered. Such aspects include nutritional deficiencies or excesses, root pruning, debarking, and the presence of insects, diseases, and nematodes.

Simazine [2-chloro-4, 6-bis (ethylamino)-s-triazine] injury on peaches is characterized by a mottling of the interveinal area of the leaf. This usually begins with chlorosis that appears in the peripheral area of the leaf and progresses toward the midrib. The midrib and main veins as well as the tissue immediately adjacent to them remain green. The affected area becomes bright

yellow. In cases of severe injury, the mottling may not develop as far as the midrib. A scorching may develop at the margin and envelop most of the leaf; this is followed by leaf drop. Mild symptoms of mottling may not affect the ability of the tree to carry a normal crop (fig. 174).

Terbacil [3-*tert*-butyl-5-chloro-6-methyluracil] injury in peach begins with the loss of green from the main veins. This occurs in mature leaves as well as newly expanding leaves. The veins turn a golden yellow. The tissue immediately adjacent to the veins is next affected, and the yellowing spreads until the entire leaf may become yellow. Exposure to higher dosages may result in little yellowing other than the veins. Leaf scorch may become the dominant injury symptom. Leaf scorch usually begins at the periphery of the lower half of the leaf and may envelop the entire leaf. Abscission of the leaves may follow. Quite often, vegetative buds break following leaf drop. These new shoots have shortened internodes and produce leaves that are much reduced in size (fig. 175).



FIGURE 174.—Peach leaves showing interveinal mottling and chlorosis resulting from simazine toxicity.



FIGURE 175.—Peach leaves showing terbacil injury: *Top*, Vein clearing at low dosage; *bottom*, leaf scorch at high dosage.

Isocil [5-bromo-3-isopropyl-6-methyluracil] injury to peach very closely resembles that of terbacil with vein clearing and yellowing of the adjacent tissue. The injury may occur more quickly over the tree than with terbacil. This may be due to isocil's solubility in water, which is approximately three times as great as terbacil (fig. 176).

Bromacil [5-bromo-3-*sec*-butyl-6-methyluracil], being closely related to isocil and terbacil, affects peaches in much the same manner. The peach seems to have less tolerance to bromacil than to either isocil or terbacil. Injury symptoms appear first as a yellowing of the veins and then leaf scorch. The yellowing of the tissue adjacent to the veins usually does not develop nearly as well as in terbacil or isocil (fig. 177).

Dalapon [2,2-dichloropropionic acid] may induce a number of injury symptoms. Tip burn may occur over any portion of the tree. At higher dosages tip burn may be accompanied by marginal leaf burn. The entire margin may become scorched. Leaf scorch may also appear between the veins. The new growth may appear lighter in color, have a shiny appearance, and show some twisting. The young leaves may fail to elongate (fig. 178).

Amitrole [3-amino-1,2,4-triazole] induces in peach the same basic symptom as it does in so many other species—whitening of the leaves. Amitrole inhibits



FIGURE 176.—Peach leaves showing vein clearing and leaf scorch resulting from isocil toxicity.



FIGURE 177.—Peach leaves showing vein clearing and leaf scorch resulting from bromocil toxicity.

chlorophyll formation, thus the lack of chlorophyll appears in the new growth. At higher dosages, the older leaves may exhibit scorching in blotches beginning at the margins. This scorching does not engulf the entire margin. Leaf abscission may occur shortly after leaf scorch appears (fig. 179).

Dichlobenil [2,6-dichlorobenzonitrile] injury in peach appears as small, necrotic spots along the margins of the fully expanded mature leaf. The dark necrotic area is surrounded by a light chlorotic area. The necrotic spots are quite small when they first appear and tend to enlarge with time (fig. 180).

Dicamba [3, 6-dichloro-*o*-anisic acid] affects terminal leaves of peach by inhibiting their unfolding. A characteristic symptom of dicamba injury in most species is cupping of the leaves. The peach exhibits a similar response as the folded leaves take on a crescent shape with the midrib as the axis. The elongation of the leaves may not be influenced at low dosage. The leaves that are folded may appear to be wilting as they droop; however, examination will show that they are firm and turgid. At higher dosages, necrosis of the growing point may occur. Leaf burn may begin with the apical leaves. It usually starts at the base of the leaf, either along the leaf margin or the midrib, and progresses acropetally. Necrosis may also occur in the stem, starting at the tip and progressing basipetally (fig. 181).



FIGURE 178.—Peach leaves showing dalapon injury: *Top*, marginal necrosis at low dosage; *bottom*, marginal and interveinal necrosis at high dosage.



FIGURE 179.—Peach leaves showing necrotic blotches resulting from amitrole toxicity.



FIGURE 180.—Peach leaves showing marginal necrosis resulting from dichlorobenil toxicity.



FIGURE 181.—Peach shoot showing dicamba injury: Crescent-shaped leaves and necrosis of growing point and leaves.

AIR POLLUTION INJURIES TO STONE FRUIT TREES

H. E. HEGGESTAD

Air pollution injury to vegetation is often confused with injury from other causes, especially virus diseases and nutritional disorders. Plants are very sensitive to air pollution; however, species vary considerably in their sensitivity to specific pollutants. Interest in air pollution has increased in the past decade. Vegetation injury by toxicants in photochemical smog is now recognized to be a national problem (441).

As with vegetation generally, little is known about the economic impact of air pollution on stone fruits. Certain markings resulting from acute injury can be attributed to specific pollutants. Emphasis in this section is on the effects of fluorides (which seem to be the pollutant of most concern to us) on stone fruits. The information on the effects of other air pollutants is reviewed.

Fluoride

Fluoride causes injury to leaves of many species of *Prunus* and to fruits of some species. Fluoride injury to peach fruit is known as soft suture, although other agents may cause similar symptoms.

The problem was first recognized in the United States in the early 1940's (394, 463, 502). Leaf injury was severe near certain industries. Principal sources of fluoride are: industries that manufacture aluminum and steel; phosphate rock processing; brick and pottery plants; chemical industries that use fluorides; and rocket fuel combustion. Although procedures for reducing fluoride pollution at the source have been improved, the number and size of some of the industries emitting fluoride have increased. Fluorides may be toxic to sensitive species when the concentration in air is a half part per billion or less. Sensitive species show leaf necrosis when concentrations in the leaf have accumulated to about 50 parts per million or more.

Prunus species are among the most sensitive plant species to fluoride air pollution. Apricot generally is highly sensitive but, Moorpark and Tilton apricot are less sensitive. Other sensitive plants are Italian Prune (*P. domestica*), peach, sloe plum, and mahaleb cherry. Sweet cherry has been classified as a tolerant species (86). Apparently, oriental flowering cherry (*P. serrulata*) and myrobalan plum (*P. cerasifera*) are also quite tolerant. A black tip condition of peach fruit, described as similar to soft suture, was reported in 1952 (428). Although 25 percent of fruits showed injury in orchards near a steel plant, no leaf scorch was observed, except on apricots near one of the orchards. Later, Benson (33) presented evidence that soft suture of peaches may be caused by fluorides. Bolay and Bovey (86) describe a dark-brown to black lesion of the stylar end of apricot and cherry attributed to fluorides.

Fruit injury results when peach trees are exposed to fluoride fumes of relatively low concentrations, especially during the pit-hardening period. Most foliage necrosis, however, is associated with the accumulation of fluorides. Exposure to very low concentrations for long periods of time, or shorter periods at high concentrations, may be equally injurious. The amount of fluoride accumulated in the tissue is the critical parameter (3, 86, 463).

The first symptom of fluoride injury is wilting at the edges of leaves, usually followed by a brown or tan marginal scorch. Injury induced by hydrogen fluoride to Belle of Georgia peach leaves is shown in figure 182. A sharply defined, reddish-brown band often separates the necrotic tissue from the adjacent healthy tissue. The marginal scorch resembles that attributed to sodium excess and magnesium deficiency. Leaf margins may cup upward. Necrotic areas may dry and fall away. Defoliation may occur. Leaf injury is intensified by lack of rain and unfavorable soil conditions, such as gravel spots or too heavy soil (502). Actually, drought may cause similar injuries in the absence of fluoride. With fluoride, the necrosis is confined more to the leaf margins than with drought. Under some conditions, chlorosis develops following exposure to fluorides, as shown on Belle of Georgia peach in figure 183. Somewhat similar chlorosis may be due to magnesium deficiency and, no doubt, other causes. Cupping and shriveling of leaf tips also occurs, as shown on Moorpark apricot in figure 184. If fluoride is the cause of injury, analysis of leaf tissue should reveal elevated concentrations of fluoride. The more sensitive species may develop leaf necrosis when fluoride in the tissue is less than 50 ppm; however, more than 50 ppm are usually required. Plants in a tolerant class, such as sweet cherry, may store over 500 ppm in leaves without visible injury (86). Controlled fumigation studies by Adams et al. (3) revealed greater injury from daily exposure for 8 hours to 1.5 parts per billion (ppb) than exposure for 8 hours



FIGURE 182.—Necrosis of peach leaves resulting from fluoride toxicity.

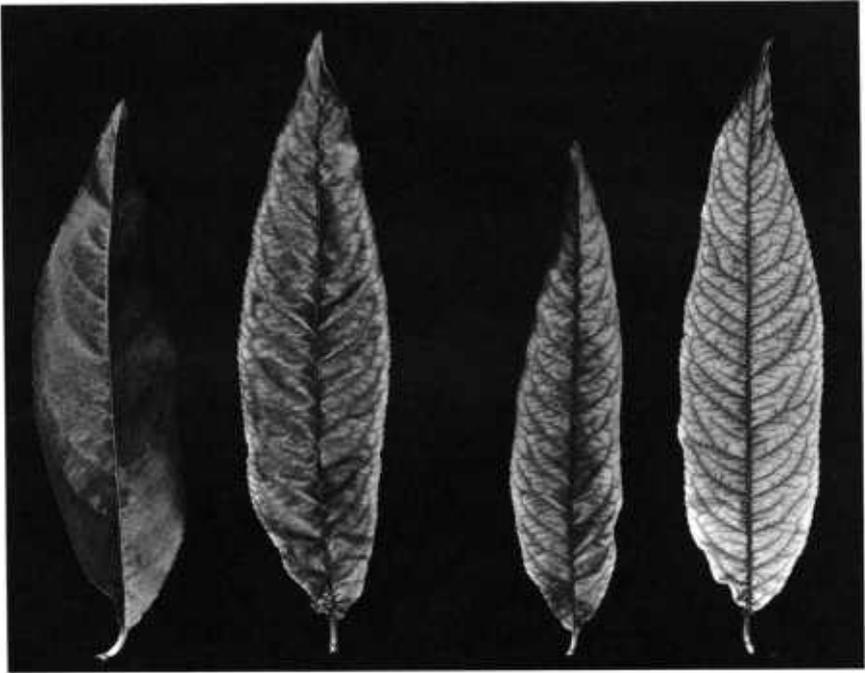


FIGURE 183.—Chlorosis of peach leaves resulting from fluoride toxicity.

every third day at 5 ppb or for 4 hours every third day to 10 ppb hydrogen fluoride. They state that plants exposed at the lowest level every day had less chance for recovery between fumigations. Field studies in Ontario (193) provide additional information on fluoride levels and injury to peach leaves and fruit.

The first symptom of fluoride injury to fruits is reddening of the skin along the suture and usually near the styler end about 2 weeks before fruits are mature. The flesh is also reddish, ripens faster, and becomes soft (figs. 185 and 186). Soft suture is sometimes known as suture red spot. Cracks in the skin may develop, and the affected portion is bruised easily and is subject to decay. Although fluorides cause soft suture, a very similar syndrome is caused by growth regulators and perhaps other agents. Fruit affected by 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) may show growth protuberances as well as soft suture (fig. 187). According to Benson (33), fruit injury induced by fluorides can be distinguished from injury caused by growth regulating compounds, such as 2,4-dichlorophenoxyacetic acid (2,4-D). In addition to uneven growth and ripening, the injury by the growth regulators is usually most severe toward the stem end of the fruit.



FIGURE 184.—Apricot shoot showing cupping and shriveling of tips of leaves resulting from fluoride toxicity, *right*; normal shoot, *left*.

Best control of fluoride injury is obtained with the use of proper scrubbers to reduce fluoride emissions at the source. Soft suture of peach may be controlled 95 percent or more by one to three sprays of lime or calcium chloride. Two pounds of calcium chloride per 100 gallons of water applied at 2- to 3-week intervals, beginning at pit hardening, is preferred. According to N. S. Benson (personal communication), calcium chloride does not leave a fruit residue and does not destroy insecticide effectiveness.

Sulfur Dioxide

Sulfur dioxide is a major air pollutant affecting vegetation. Primary sources are the burning of fossil fuels, smelting of ores, production and refining of petroleum and natural gas, and manufacture and utilization of sulfur and sulfuric acid. Zimmerman and Hitchcock (775) reported the results of controlled fumigations with sulfur dioxide on 49 species of plants, including *Prunus*. The concentration of the gas ranged from 0.2 to 1.1 ppm, and the time from 2 to 8 hours. With an exposure of 0.5 ppm sulfuric dioxide for 4 to 8 hours, Moorpark apricot showed slight markings. Italian Prune was more susceptible; it developed moderate markings. Italian Prune was approximately as susceptible as buckwheat, smartweed, grape, and sweetpotato. On dicotyledonous plants,

the leaf tissue surrounding larger veins remains green; spotting results from death of cells in interveinal areas. Injury to apricot and Sun High peach from exposure to 3 ppm sulfur dioxide for 4 hours is shown in figure 188. Chronic injury appears as chlorosis; death of a few isolated cells is indicated by white or brownish areas.

Field studies at Biersdorf, Germany, included observations on sweet cherry, sour cherry, and plum. Levels of sulfur dioxide varied with distance from the primary source, an iron sintering plant. The 24-hour average concentration at the station nearest the source was about 0.15 ppm; and taking into account only emission periods, the average was about 0.6 ppm. The pollutant caused a marked reduction in growth of trees and in quality of fruit (268).

Ozone

Ozone is the major phytotoxicant in photochemical smog. Symptoms on most dicotyledonous plants are either a fleck or stipple on the upper surface of

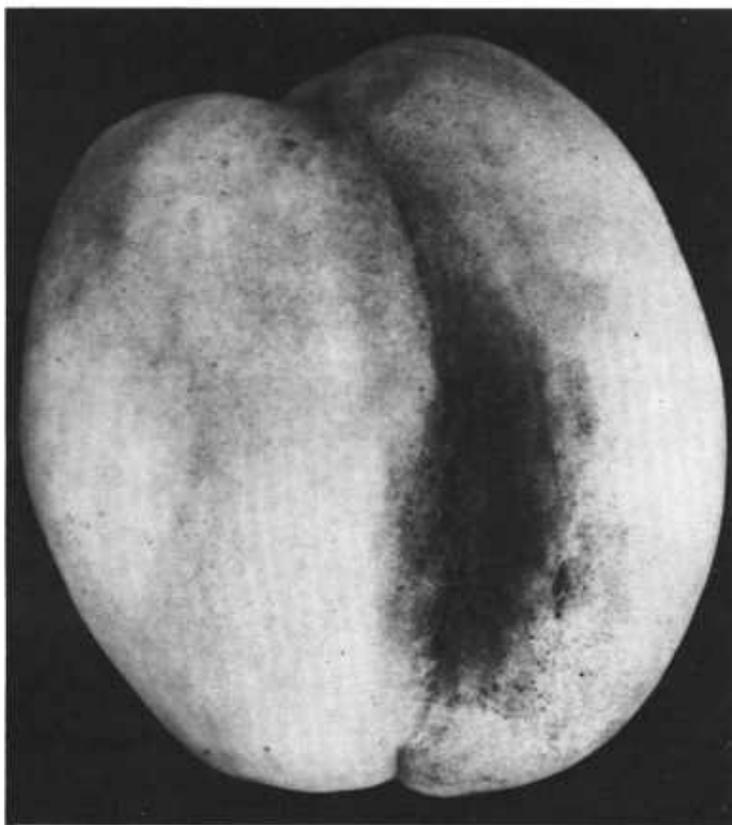


FIGURE 185.—Soft suture of peach fruit resulting from fluoride toxicity.

fully expanded leaves. Chlorosis and loss of leaves is usually a part of the syndrome. There is very little information about effects on stone fruits. Injury by mites resembles fleck caused by ozone.

Elberta peach was one of 37 plant species fumigated with ozone by Hill et al.

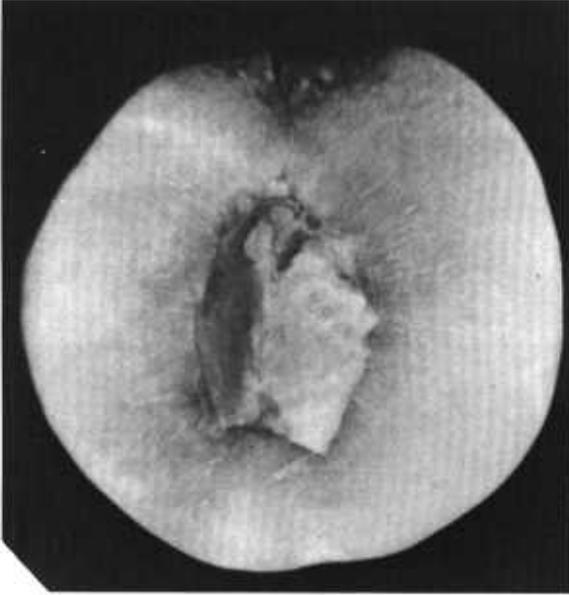


FIGURE 186.—Cross section of peach fruit with soft suture resulting from fluoride toxicity, showing, at *top*, a darkened area in the flesh.



FIGURE 187.—Peach fruit showing growth protuberances, predominantly at the stem end, resulting from 2,4,5-T toxicity.

(314). Plants were exposed for 2 hours to ozone treatments, ranging from 0.13 to 0.41 ppm. Peach was intermediate in sensitivity. The injury was described as "a mild chlorotic upper-surface mottling along veins." In addition, dark lesions and shot-holing may develop, as shown in figure 189. The injury resulted from exposure to 0.6 ppm ozone for 2 hours. The dark lesions and shot-holing developed more rapidly than the mottling along veins, which was



FIGURE 188.—Leaves of peach, *left*, and apricot, *right*, injured by exposure to sulfur dioxide 3 ppm for 4 hours.

more pronounced on leaves a few days after the photograph (fig. 189) was taken.

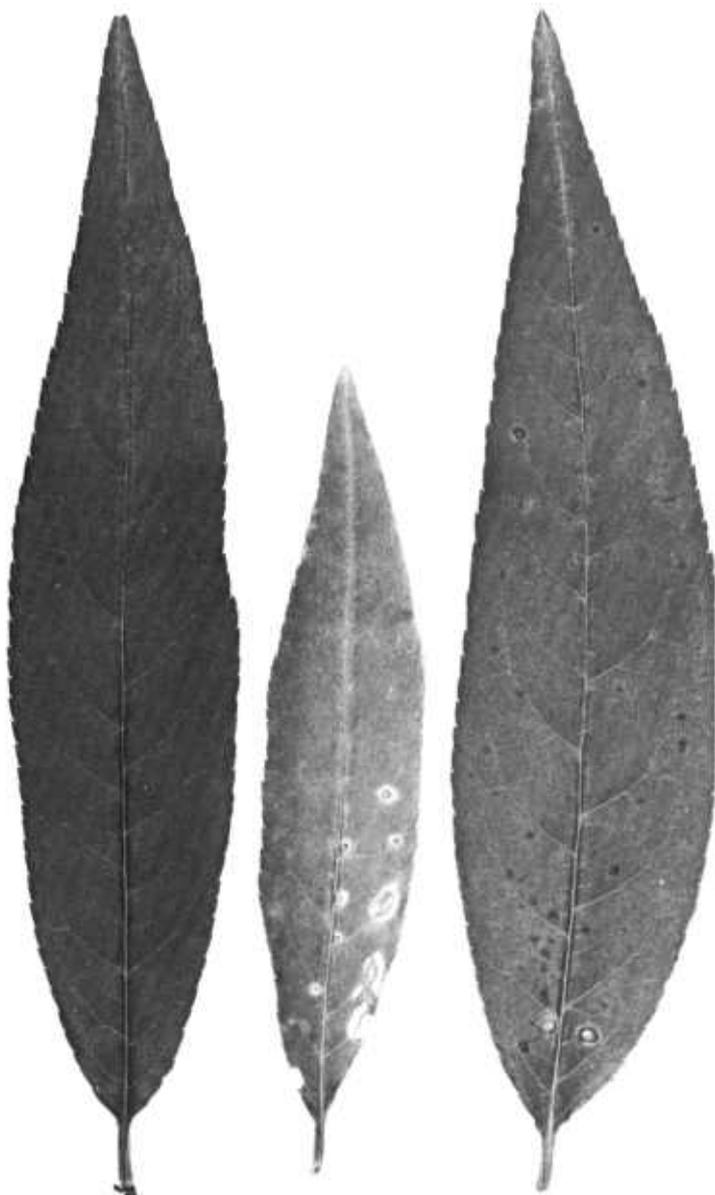


FIGURE 189.—Peach leaves showing chlorosis and dark lesions with shotholing resulting from ozone toxicity; normal leaf on *left*.

Plum trees were injured in Switzerland by ozone in the vicinity of a water reservoir where ozone treatment of drinking water was underway (596). Apparently, sufficient ozone escaped to the air to reach phytotoxic levels in the valley. The leaves of plums were chlorotic, especially between the larger veins. There was considerable tip and border necrosis. Italian Prune trees were more sensitive to ozone than cherry trees tested under the same conditions.

Researchers in California (356) listed about 115 plant species as either susceptible or resistant to photochemical smog. Perhaps the primary toxicant in the smog was ozone. Apricot was rated susceptible, and cherry, peach, and plum resistant.

The use of ozone to preserve peach and other fruits was investigated by Spalding (623). Except for inhibiting surface mycelium, no significant effect was found on the fungi that were causing rhizopus and brown rots of peaches, when ozone concentrations of up to 10 ppm were maintained for 5 days. This dosage of ozone, however, resulted in injury to peaches described as sunken and browned tissue in the region of the stomata. Ozone caused death of tissue and loss of moisture. The fruit had a pebbly appearance (fig. 190). Little or no injury to fruit occurred when the concentration of ozone was reduced to 0.5 ppm and lower.

Other Pollutants

Peroxyacetyl nitrate was identified about 10 years ago as a primary phytotoxicant in photochemical smog (441). Typically, the injury is a glazing or bronzing of the undersurface of leaves. No reports are available on the sensitivity of *Prunus* species.

Ethylene is a common air pollutant, with motor vehicle exhaust and the manufacture of certain plastics as primary sources. Symptoms on plant species include epinasty, chlorosis, necrosis, leaf and bud abscission, and failure of

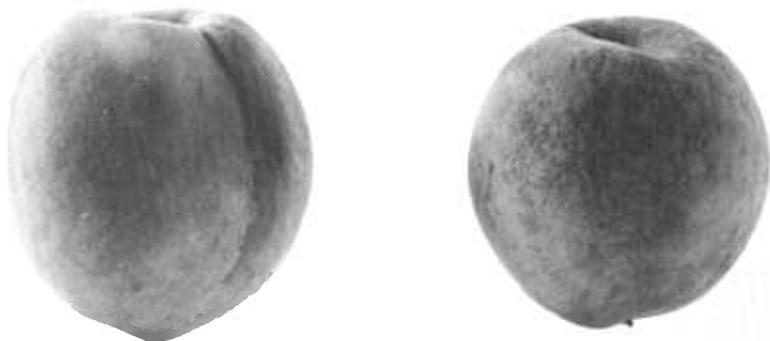


FIGURE 190.—Injury to peach fruit, *right*, resulting from exposure to high concentration of ozone during storage. Normal fruit, *left*.

flowers to open. Heck et al. (288) determined the response of 89 species, including peaches, to relatively high concentrations. Tests with peach seedlings revealed them to be one of the more sensitive of plant species.

Nitrogen dioxide, chlorine, aldehydes, and ammonia are other pollutants receiving attention by researchers, but no specific information on the sensitivity of *Prunus* species seems to be available. The possibility of effects of combinations of pollutants also must be considered. Literature references and more detail on air pollution injury to vegetation are found in recent reviews (95, 289, 752).

Contact type chemicals, such as arsenite, used for defoliant on such crops as potatoes prior to harvest, have caused varying types of injury to nearby fruit orchards as a result of drift of the chemicals. Trees nearby may lose nearly all leaves. At greater distances, the side of the trees nearest the sprayed area will show most injury. Increased injury is usually associated with improper formulations and application techniques.

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APPENDIX

COMMON AND SCIENTIFIC NAMES OF PLANTS MENTIONED IN THE TEXT⁴

ALMOND	<i>Prunus dulcis</i> (Mill.) D. A. Webb
ALMOND, DESERT	<i>P. fasciculata</i> (Torr.) Gray
ALMOND, FENZL	<i>P. fenzliana</i> Fritsch
ALMOND, FLOWERING	<i>P. triloba</i> Lindl.
ALMOND, DWARF FLOWERING	<i>P. glandulosa</i> Thunb.
ALMOND, RUSSIAN	<i>P. tenella</i> Batsch
ALMOND, TANGUT	<i>P. tangutica</i> (Batal.) Koehne
APPLE	<i>Malus sylvestris</i> Mill.
APRICOT	<i>Prunus armeniaca</i> L.
APRICOT, ANSU	<i>P. armeniaca</i> var. <i>ansu</i> Maxim.
APRICOT, DESERT	<i>P. fremontii</i> S. Wats.
APRICOT, JAPANESE	<i>P. mume</i> Sieb. + Zucc.
BALSAMAPPLE	<i>Momordica balsamina</i> L.
BEAN, COMMON	<i>Phaseolus vulgaris</i> L.
BEAN, PINTO	<i>P. vulgaris</i> L.
BLACKBERRY, HIMALAYA	<i>Rubus procerus</i> P. J. Muell.
BRISTLY OXTONGUE	<i>Picris echioides</i> L.
BUSHCHERRY, CHINESE (KOREAN)	<i>Prunus japonica</i> Thunb.
CARROT	<i>Daucus carota</i> L.
CASTOR BEAN	<i>Ricinus communis</i> L.
CATJANG	<i>Vigna unguiculata</i> <i>supp. cylindrica</i> (L.) Van Eseltine
CELERY	<i>Apium graveolens</i> var. <i>dulce</i> (Mill.) Pers.
CHEESEWEED	<i>Malva parviflora</i> L.
CHERRY, ALMOND	<i>Prunus glandulosa</i> Thunb.
CHERRY, AMUR	<i>P. maackii</i> Rupr.
CHERRY, BITTER	<i>P. emarginata</i> (Hook.) Walp.
CHERRY, BITTER (VAR. MOLLIS)	<i>P. emarginata</i> var. <i>mollis</i> (Doug. ex Hook.) Brew. & Wats.
CHERRY, BLACK	<i>P. serotina</i> Ehrh.
CHERRY, CAPOLLIN	<i>P. serotina</i> var. <i>salicifolia</i> (HBK) Koehne (syn. <i>P. capuli</i> Cav.)
CHERRY CATALINE	<i>P. lyonii</i> Sarg.
CHERRY, CHINESE FLOWERING	<i>P. pseudocerasus</i> Lindl.
CHERRY, DESFONTAINES	<i>P. xfontanesiana</i> (Spach) Schneid. (<i>P. avium</i> L. x <i>P. mabaleb</i> L.)
CHERRY, DUKE	<i>P. avium</i> L. x <i>P. cerasus</i> L.
CHERRY, GROUND	<i>P. fruticosa</i> Pall.
CHERRY, HOLLYLEAF	<i>P. ilicifolia</i> (Nutt.) Walp.

⁴Names checked by the Plant Taxonomy Laboratory, Agricultural Research Service, U.S. Department of Agriculture.

- CHERRY, JAPANESE FLOWERING
 FUJI CHERRY
 SIEBOLD CHERRY
 TAKANE CHERRY
- CHERRY, MAHALEB
 CHERRY, MANCHU
 CHERRY, MAZZARD
 CHERRY, ORIENTAL FLOWERING
- HIGAN CHERRY
 SARGENT CHERRY
 YOSHINA CHERRY
- CHERRY, PIN
 CHERRY, SAND
 CHERRY, SAND, PURPLE LEAF
 CHERRY, SOUR
 CHERRY, SOUR, DOUBLE
 FLOWERING
 CHERRY, SWEET
 CHERRY, WESTERN SAND (BESSEY)
 CHOCKECHERRY, AMUR
 CHOCKECHERRY, EASTERN
 CHOCKECHERRY, GREAT PLAINS
- CHOCKECHERRY, WESTERN
- COWPEA
 CRAB APPLE, BIGFOOT
 CUCUMBER
 CURRANT, COMMON RED
 DODDER
- DOLICHOS
 FULLER'S TEASEL
 GLOBE AMARANTH
 GOOSEFOOT
 GRAPE
 GROUNDCHERRY
 GUAR
 HEDGENETTLE
 HOP
 JIMSON-WEED
 KERRIA, JAPANESE
 LAURELCHERRY, COMMON
 LAURELCHERRY, ZABEL
 LAURELCHERRY, PORTUGUESE
 MAPLE, RED
 MILKWEED, COMMON
 MOUNTAIN ASH
 NECTARINE
 PARSLEY
- P. incisa* Thunb.
P. sieboldii (Carr.) Wittm.
P. nipponica Matsum.
P. ssiiori F. Schmidt
P. mahaleb L.
P. tomentosa Thunb.
P. avium L.
P. serrulata Lindl.
P. serrulata var. *lannesiana* (Carr.) Rehd.
P. subhirtella Miq.
P. sargentii Rehd.
P. yedoensis Matsum.
P. pennsylvanica L.
P. pumila L.
P. cistena N. E. Hanson
P. cerasus L.
P. cerasus f. *rbexi* (Kirchn.) Voss.
P. avium L.
P. besseyi Bailey
P. maackii Rupr.
P. virginiana L.
P. virginiana var. *melanocarpa* (A. Nels.)
 Sarg.
P. virginiana var. *demissa* (Nutt. ex
 Torr. & Gray) Torr.
Vigna unguiculata (L.) Walp.
Malus platycarpa Rehd.
Cucumis sativus L.
Ribes sativum Syme
Cuscuta campestris Yunck.
C. subinclusa Dur. & Hilg.
Dolichos biflorus L.
Dipsacus fullonum L.
Gomphrena globosa L.
Chenopodium amaranticolor Coste & Reyn.
Vitis spp.
Physalis floridana L.
Cyamopsis tetragonoloba (L.) Taub.
Stachys bullata Benth.
Humulus americanus Nutt.
Datura stramonium L.
Kerria japonica (L.) DC.
Prunus laurocerasus L.
Prunus laurocerasus f. *zabeliana* Spaeth
P. lusitanica L.
Acer rubrum L.
Asclepias syriaca L.
Sorbus spp. L.
Prunus persica var. *nectarina* (Ait.) Maxim.
Petroselinum crispum (Mill.) Nym.

PEACH	<i>Prunus persica</i> (L.) Batsch
PEACH, DAVID	<i>P. davidiana</i> (Carr.) Franch.
PEACH, DESERT	<i>P. andersonii</i> Gray
PEACH, FLAT	<i>P. persica</i> f. <i>compressa</i> (Loud.) Rehder
PEACH, KANSU	<i>P. kansuensis</i> Rehd.
PEACH, PURPLELEAF	<i>P. persica</i> f. <i>atropurpurea</i> Schneid.
PEACH, SMOOTHPIT	<i>P. mira</i> Koehne.
PEAR	<i>Pyrus communis</i> L.
PERIWINKLE	<i>Vinca rosea</i> L.
PETUNIA	<i>Petunia hybrida</i> Vilm.
PLANTAIN	<i>Plantago</i> spp.
PLUM, AMERICAN	<i>Prunus americana</i> Marsh.
PLUM, APRICOT	<i>P. simoni</i> Carr.
PLUM, BOKAR	<i>P. bokhariensis</i> Royle ex Schneid.
PLUM, BEACH	<i>P. maritima</i> Marsh.
PLUM, CHICKASAW	<i>P. angustifolia</i> Marsh.
PLUM, DAMSON (BULLACE)	<i>P. insititia</i> L.
PLUM, EUROPEAN (PRUNE)	<i>P. domestica</i> L.
PLUM, FLATWOODS	<i>P. umbellata</i> Ell.
	<i>P. umbellata</i> var. <i>injucunda</i> (Small.) Sarg.
PLUM, HOG	<i>P. reverchonii</i> Sarg.
PLUM, HORTULAN	<i>P. hortulana</i> Bailey
PLUM, HYBRIDS	<i>P. besseyi</i> Bailey x <i>P. salicina</i> Lindl.
	<i>P. salicina</i> Lindl. x <i>P. simonii</i> Carr.
PLUM, JAPANESE	<i>P. salicina</i> Lindl.
PLUM, KLAMATH	<i>P. subcordata</i> Benth.
PLUM, MARIANNA	<i>P. cerasifera</i> Ehrh. x <i>P. munsoniana</i> Wight & Hedr.
PLUM, MEXICAN	<i>P. mexicana</i> S. Wats.
PLUM, MYROBALAN	<i>P. cerasifera</i> Ehrh.
PLUM, PURPLELEAF	<i>P. cerasifera</i> cu. <i>Atropurpurea</i> syn. <i>P. pissardi</i> Carr.
PLUM, SLOE	<i>P. spinosa</i> L.
PLUM, WILDGOOSE	<i>P. munsoniana</i> Wight & Hedr.
PORTUGAL LAUREL	<i>P. lusitanica</i> L.
QUINCE	<i>Cydonia oblonga</i> Mill.
QUINOA	<i>Chenopodium quinoa</i> Willd.
RASPBERRY, RED	<i>Rubus idaeus</i> L.
SEASIDE DAISY	<i>Erigeron glaucus</i> Ker
SERVICEBERRY	<i>Amelanchior</i> spp. Med.
SESBANIA	<i>Sesbania exaltata</i> (Raf.) Cory
SNAPDRAGON	<i>Antirrhinum majus</i> L.
SQUASH	<i>Cucurbita maxima</i> Duchesne, + 3 other spp. called squash
CULTIVATED STRAWBERRY	<i>Fragaria x ananassa</i> Duch.
STRAWBERRY, SAND	<i>F. chiloensis</i> (L.) Duchesne
SWEET BASIL	<i>Ocimum basilicum</i> L.
TITHONIA	<i>Tithonia speciosa</i> (Hook.) Griseb.
TOBACCO	<i>Nicotiana tabacum</i> L.
	<i>N. glutinosa</i> L.
	<i>N. rustica</i> L.
	<i>N. megalosiphon</i> Heurck & Muell.-Arg.
TOMATO	<i>Lycopersicon esculentum</i> Mill.
ZINNIA	<i>Zinnia elegans</i> Jacq.

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