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Important Diseases of *Prunus* Caused by Viruses and Other Graft-Transmissible Pathogens in California and South Carolina

The genus *Prunus* comprises more than 400 species that include stone fruits, almonds, and many ornamentals. The diversity of the genus is reflected in the large number of virus and viruslike diseases that are known to infect these species. Nemeth (27) lists more than 100 diseases worldwide. This reference source and the USDA's *Agriculture Handbook No. 437* (28) provide details of individual diseases and should be consulted for more information on diseases of interest.

The agents inducing many of these diseases are unknown but have been considered "viruslike" because they are graft-transmitted and because the diseases share symptoms associated with virus infection, such as mosaic, mottle, shot-hole, leaf distortion, stunting, gummosis, canker and other bark abnormalities, precocious flowering, delayed flower and leaf development, alterations in ripening date, fruit abnormalities, and tree death. The symptoms in fruit trees, herbaceous test hosts, and woody indicator species may vary markedly, depending on the strain or isolate of the virus. For example, some isolates of Prunus necrotic ringspot virus cause chlorotic spots and blotches, enations on the underside of leaves, and decline of trees, whereas others cause only shot-holes in leaves (shock reaction). Some isolates cause shot-hole symptoms annually and are referred to as recurrent ringspot strains.

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Chemicals or pathogens unrelated to viruses can cause viruslike symptoms. An example is injury to peach caused by the herbicide norflurazon (Solicam), including shortened internodes, precocious flowering, and a vivid green and white mosaic of the leaves. These symptoms, singly or collectively, are commonly attributed to a viral pathogen. Thus, while some virus diseases may be diagnosed by the symptoms, it is prudent to test representative samples by serological, biochemical, or biological assays for verification. A number of diseases that were classified as viral in nature in the past are now known to be caused by viroids, mycoplasmalike organisms (MLOs), or fastidious vascular bacteria. Undoubtedly, when causal agents of other currently intransigent diseases are identified, diagnosis and control will be improved.

In California, more than 275,000 ha were devoted to the cultivation of *Prunus* fruit and nut crops during 1989 (3), including 173,342 ha planted to almonds (*P. dulcis* (Mill.) D. Webb), 33,190 ha to prunes (*P. domestica* L.), 15,208 ha to clingstone and 12,239 ha to freestone peaches (*P. persica* (L.) Batsch), 17,821 ha to plums (*P. salicina* Lindl.), and 4,988 ha to cherries (*P. avium* (L.) L.). The collective production value was in excess of $1 billion (2).

Stone fruit production in South Carolina is limited primarily to peaches and nectarines. In 1988, 13,292 ha were planted to the two crops, with production valued at $53 million (1). South Carolina ranks second in peach production in the United States, with most of its crop entering the fresh fruit market. Some peach growers maintain small plantings of plums for local markets.

This article focuses on the diseases of current economic importance in California and South Carolina and considers in detail their epidemiology and control.

Diseases Caused by Ilarviruses

Two ilarviruses (*i*sometric *la*bile *r*ingspot *viruses*) (9), Prunus necrotic ringspot (NRSV) and prune dwarf (PDV), are routinely found in commercially cultivated almond and stone fruit trees. Although not related serologically, NRSV and PDV share biological properties. They feature quasi-isometric particles (Fig. 1A), and both infect a wide range of *Prunus* species and plant families other than the Rosaceae. They are borne in pollen and seed, are readily graft-transmitted, and during pollination can cause infection of healthy mother trees. These properties have contributed to their worldwide distribution.

Prunus necrotic ringspot was described in 1941 on peach (6). The initial (shock) symptoms consist of chlorotic spots and rings that turn necrotic on recently developed leaves. The necrotic tissues later fall out, leaving a tatter or shot-hole appearance of the leaves (Fig. 1B). After the shock phase, the trees may appear relatively normal, even though the virus continues to replicate and reach a high concentration annually. The recurrent ringspot strain of NRSV induces shock-type symptoms every year on certain hosts. In contrast, the rugose mosaic strain causes chlorotic blotches (Fig. 1C), leaf enations, and decline of cherry trees, and the calico strain causes a yellow mosaic pattern, as seen in almond. Fruit ripening is uneven on cherry trees with rugose mosaic disease.

The name "prune dwarf" was derived from symptoms of stunting and leaf malformation observed on Fellenberg prune (*P. domestica*) (Fig. 2) (38). The virus also causes chlorotic spots and rings in sweet cherry, yellows in sour cherry (7), and stunting in Muir peach (22).

Peach stunt disease (PSD), caused by a combination of PDV and NRSV, has reached epidemic proportions in Califor-

nia. Shoot growth of young trees is retarded, and although infected trees are always smaller than healthy trees (Fig. 3), the growth pattern is more normal with increased ambient temperatures. Fruit yields on PSD trees are greatly reduced (J. K. Uyemoto, *unpublished*).

Surveys of young orchards in California showed a high incidence of NRSV and PDV. One in four peach trees was infected by one or both viruses (41). Similarly, almond and prune orchards averaged 20% infected trees and sweet cherry orchards, 4% infected trees (40). Because field spread of NRSV and PDV is thought to occur during pollination by infected pollen, their presence in nonbearing trees indicates that infected materials were used in propagation. To minimize introduction of disease into orchards by this means, all scion and seed-source trees should be tested for virus either by serological means, e.g., enzyme-linked immunosorbent assay (ELISA), or by grafting of Shirofugen flowering cherry (*P. serrulata* Lindl.).

A survey of South Carolina peach orchards (33) showed that an average of 30% of the trees sampled were infected with NRSV, which was not causing symptoms. No infected trees were detected in some orchards, whereas in others, all the trees sampled were infected. The incidence of infection varied among the cultivars and ranged from 7% in Harvester to 74% in Redhaven. Because all trees in some young orchards were infected, the trees were probably propagated with infected scion buds or rootstocks, or both. PDV was found in ornamental flowering cherry and in peach, but no instances of peach stunt were found.

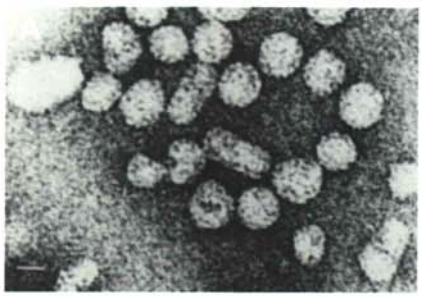

Fig. 1. (A) Electron micrograph of apple mosaic ilarvirus. Spherical to bacilliform quasi-isometric particles are 25–30 nm in diameter. Particles were stained with 1% sodium phosphotungstate. Scale bar = 17.6 nm. (Courtesy Patrick Shiel) (B) Tatter leaf (shock) symptoms in sweet cherry caused by Prunus necrotic ringspot virus (NRSV). (C) Chlorotic blotch in sweet cherry caused by the rugose mosaic strain of NRSV.

Fig. 2. (Right) Small, narrow leaves on prune caused by prune dwarf virus. (Left) Healthy plant.

Fig. 3. (Right) Peach trees with stunt disease caused by dual infection with prune dwarf and Prunus necrotic ringspot viruses. (Left) Healthy peach trees planted a year later.

Diseases Caused by Nepoviruses

Nepoviruses (*ne*matode *po*lyhedral *viruses*) share such attributes as being: 1) sap-transmissible to a wide variety of herbaceous hosts, 2) frequently borne in weed seeds, 3) vectored by nematodes, and 4) polyhedral in particle outline (Fig. 4A). Two members of the nepovirus group occur in California: tomato ringspot virus (TmRSV) on several *Prunus* species and cherry raspleaf virus (CRLV) on sweet cherry trees.

A disease caused by TmRSV was first described in 1936 (29,34). The virus is endemic in North America, and although it does not cause a disease of cultivated tomato, it does cause severe losses in a number of perennial plants, e.g., *Prunus* species, apple (*Malus domestica* Borkh.), grapevine (*Vitis vinifera* L. and interspecific hybrids), and red raspberry (*Rubus idaeus* L. var. *strigosus* (Michx.) Maxim.). In the eastern and western United States, TmRSV infects many *Prunus* species, causing thickened, necrotic bark tissues and pits and grooves in the woody cylinder; affected trees die.

The yellow bud mosaic strain (31) of TmRSV incites primary leaf symptoms

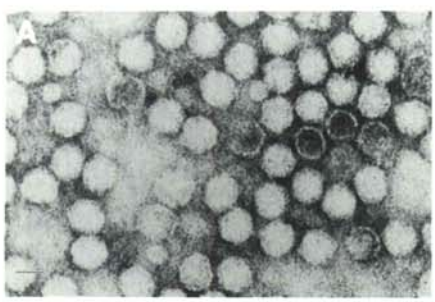

Fig. 4. (A) Electron micrograph of negatively stained particles (about 28 nm in diameter) of tobacco ringspot nepovirus. A few particles are stained internally. Scale bar = 21.5 nm. (Courtesy O. W. Barnett) (B) Peach leaf with ringspot symptoms caused by the yellow bud mosaic strain of tomato ringspot virus (TmRSV). (C) Small yellow-leaf clusters associated with peach yellow bud mosaic disease. (D) Brownline symptoms caused by TmRSV at the scion/rootstock junction of President plum on peach root.

consisting of chlorotic rings and mosaic patterns (Fig. 4B). On chronically infected trees, budbreak is delayed and the affected leaf buds produce small, yellowed leaves (Fig. 4C) that subsequently die. Fruit-set and yield are reduced. On affected Mission almond trees, the fruit hull is thickened and wrinkled.

Strains of TmRSV, including yellow bud mosaic strain, also cause the brownline disease of prunes (*P. domestica*) (23). A dark brown line, representing necrotic phloem tissues, forms at the scion/rootstock junction and extends into pits and grooves in the woody (xylem) cylinder (Fig. 4D). This girdling causes interveinal chlorosis of the leaves of the scion, and the tree dies. These symptoms are seen on prune trees grown on susceptible rootstocks, i.e., peach or Myrobalan plum (*P. cerasifera* Ehrh.), but not on those grown on Marianna 2624 plum (*P. cerasifera* × *munsoniana* W. Wight & Hedr.). Graft inoculation trials revealed that the latter rootstock is immune to the virus (12).

Another strain of TmRSV causes cherry leaf mottle, the symptoms of which include a light green leaf mottle, poor shoot growth, and decline (11). Although stem pitting was not observed in natural infections, affected trees nonetheless declined. Hoy (11) reported that graft inoculations of different *Prunus* species with several TmRSV strains, including the leaf mottle strain, caused stem pitting in some, but not all, virus-host combinations.

The virus is transmitted by the dagger nematode, *Xiphinema americanum* Cobb sensu lato, the species taxonomy of which is uncertain (5,37). TmRSV infects and becomes seedborne in several species of weeds commonly found in orchards. For example, the dagger nematode can acquire TmRSV from dandelion (*Taraxacum officinale* Wigg.) and transmit it to dandelion seedlings (25).

TmRSV occurs in South Carolina in dogwood (*Cornus florida* L.) (30) and *Trifolium ambiguum* M. Bieb. (32). Although trees in several peach and nectarine orchards in the Piedmont region of the state show classical symptoms of stem pitting, the presence of TmRSV in these *Prunus* hosts has not been demonstrated thus far. Nematodes collected in soil samples taken from around tree bases have been used in transmission tests to cucumber. Veinclearing of some plants resulted, and the symptom could be sap-transmitted to other cucumbers. Results of ELISA of cucumber extracts for TmRSV and tobacco ringspot virus were negative. However, dsRNA analysis of infected cucumber tissues revealed two faint bands with molecular weights similar to those found for dsRNAs of TmRSV (S. W. Scott, *unpublished*).

TmRSV is difficult to control. The orchard site must be thoroughly fumigated before planting to greatly reduce or eliminate the nematode vector population. Only stocks that test free of the virus should be planted. Once an orchard is established, proper weed control is essential to minimize reintroduction of the virus through infected seed. When prune is being replanted in known infested sites, trees on the resistant Marianna 2624 rootstock should be used.

CRLV was reported to cause a disease of cherry in 1942 (4) and is currently confined to areas of the United States and Canada west of the Rocky Mountains. Symptoms on sweet cherry consist of enations on the underside of affected leaves (Fig. 5) on the lowest branches of the tree. Sometimes the entire canopy is affected. Symptomatic trees eventually decline and die.

This nepovirus is also vectored by *X. americanum* and infects a number of weed hosts (35). It is seedborne in dandelion, infects peaches, and causes a flat-fruit symptom in apple. The disease is difficult to control. Soil should be fumigated and the weed host suppressed. Natural resistance among rootstocks used for cherry and peach is not known.

Another Soil-Associated Disease

Recently, a stem-pitting disease of sweet cherry trees has occurred in epidemic proportions in California (42). Attempts to associate TmRSV with the disease have failed (11; J. K. Uyemoto, *unpublished*). Affected trees produce small leaves and small fruits borne on short pedicels. Severe stem pitting develops on the scions and rootstocks of *P. avium* (Fig. 6). When cherry trees on *P. mahaleb* L. rootstocks are infected, the stem pitting is confined to the scion (*P. avium*) portion of the trees; no stem pitting has been observed on *P. mahaleb*. Affected trees die within a year or so after onset of symptoms. A soilborne cause is suggested because cherry replants develop symptoms after 1 year and pitting symptoms are most severe near or below the soil line when mazzard roots are involved.

Diseases Caused by Xylem-Limited Bacteria

Phony peach, plum leaf scald, and almond leaf scorch are three currently recognized diseases of *Prunus* species caused by the xylem-limited bacterium *Xylella fastidiosa* Wells et al (43,44).

Phony peach was first observed in Georgia in 1885 (27) and over the next 30 years reached epidemic proportions throughout the southeastern United States. Trees are dwarfed and have a lush green, bushy appearance in midsummer (Fig. 7); fruit production is reduced. The pathogen is transmitted when scions containing diseased xylem are grafted onto the xylem of a host tree. Natural spread from tree to tree is by leafhoppers of the subfamily Tettigellinae: *Draeculacephala* sp., *Homalodisca coagulata* (Say), *H. insolita* (Walker), *Oncometopia orbona* (Fabricius), *Graphocephala versuta* (Say),

Fig. 5. Enations on the underside of a sweet cherry leaf caused by cherry raspleaf virus.

Fig. 6. Stem pitting caused by a graft-transmissible agent in the xylem of mazzard rootstock. This symptom is also induced on Bing scions.

Fig. 7. (Right) Tree with phony peach disease is stunted and dark green compared with (left) healthy tree. (Courtesy J. H. Aldrich)

Fig. 8. Symptoms of plum leaf scald. (Courtesy J. H. Aldrich)

and *Cuerna costalis* (Fabricius). Because severe disease outbreaks continued in the endemic areas, a cooperative eradication program was implemented in 1929 and continued until 1950. During that period, some 2 million trees were destroyed in the southeastern states. Federal and local state inspectors visited peach orchards and marked diseased trees, which growers later removed. Concomitantly, participating growers were encouraged to eliminate wild plum trees and Johnson grass (*Sorghum halepense* (L.) Pers.), which serve as natural hosts of the pathogen.

The state of South Carolina has continued the eradication program since its inception. All symptomatic trees are destroyed. Consequently, phony disease occurs only in certain areas and orchards where it is endemic. The overall disease incidence is about 0.03% in a population of nearly 3 million peach trees.

With plum leaf scald, chlorosis of the leaf margin begins early in the summer and gradually becomes more pronounced, and the leaf margin turns brown and dry (Fig. 8). Scald may affect over one-half of the leaf before abscission occurs. In the early stages of disease, symptoms may affect only a few twigs or larger branches, but as the season progresses, almost all of the foliage is affected. The disease is a limiting factor in plum production in some parts of the southeastern United States (21).

In California, almond leaf scorch was first observed in the 1930s (24). The disease is an economic problem of almonds in localized regions of the state. The causal organism also incites Pierce's disease in grapes and a dwarf condition in alfalfa. Leaf scorch develops late in the growing season, beginning in early July with yellowed leaf tips and margins, followed by necrosis of the affected areas and a yellow zone separating the green and necrotic areas (Fig. 9A). Diseased trees show reduced vegetative growth and develop dead spurs and limb dieback. The symptoms of bacterial leaf scorch can be confused with those of salt damage. Salt-associated leaf scorch lacks the yellow zone, however, and a simple colorimetric test confirms almond leaf scorch. The test involves soaking exposed cross or longitudinal sections of young symptomatic branches in an acidified methanol solution (5 ml concd HCl in 500 ml methanol) for 30 minutes or more. Diseased xylem shows deep red spots or streaks (Fig. 9B); healthy tissues lack similar areas of concentrated stain. Alternatively, the pathogen may be cultured or tested by serology (8,10).

The causal bacterium invades and is confined to the xylem (Fig. 9C and D). Hence, it is vectored only by xylem-feeding insects such as spittlebugs and certain species of leafhoppers known as sharpshooters, e.g., *Draeculacephala minerva* Ball (Fig. 9E).

Disease control involves annual tree inspections during late July, when leaf scorch symptoms are well developed. Infected trees should probably be sprayed (to control insect vectors) before they are removed. A recently infected tree with symptoms confined to the outer limbs occasionally is rescued by severe pruning of the symptomatic limbs or scaffolds. In endemic areas, the most tolerant almond cultivars, e.g., Harvey, Ruby, Padre, Solano, Carmel, Butte, and Fritz, should be planted. Also, orchards should not be planted near riparian areas where the vectors and perennial hosts of the pathogen are likely to occur.

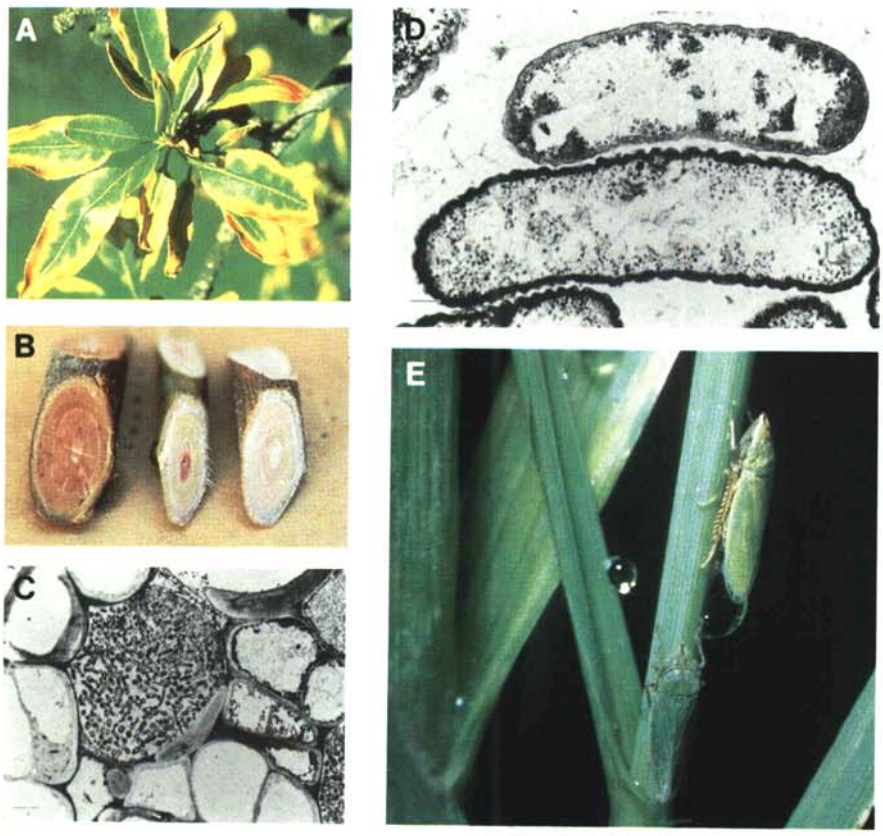

Fig. 9. Almond leaf scorch: (A) Almond leaves showing chlorotic area between necrotic and green tissues. (Courtesy D. A. Golino) (B) Cross sections of (left) diseased and (middle and right) healthy shoots stained with acidified methanol. Diseased xylem shows dark red spots. (C) Electron micrograph of the causal agent in a grape xylem vessel and (D) enlargement of the bacterium showing the rippled, solid wall. Scale bars = 4.0 μm and 100 nm, respectively. (Courtesy A. H. Purcell) (E) Male (top) and female (bottom) green sharpshooters (*Draeculacephala minerva* Ball), a vector of the pathogen. (Courtesy A. H. Purcell)

Fig. 10. (A) Symptoms of little peach confined to a single scaffold branch. (B) Peach rosette, with tufts of leaves borne on shoot terminals. (C) Electron micrograph of mycoplasmalike organisms associated with western X-disease in a phloem sieve cell.

Diseases Caused by MLOs

Three yellows-type diseases of peach are recognized in South Carolina: peach yellows, little peach, and peach rosette. Peach yellows was first observed in Pennsylvania in 1791 (19); about a century later little peach was observed in Michigan and peach rosette, in Georgia. All three diseases cause shoot proliferation, greatly reduced growth, and leaf yellowing.

Peach yellows is characterized by yellowed leaves with the margins rolled upward. On severely affected trees, chlorotic leaves are small, narrow, and borne on slender shoots. Fruits are of low quality and lack flavor. Affected trees may survive for 2–3 years.

With little peach disease, first described in 1896 (27), the canopy is darker green than normal during the early season. Affected trees appear bushy because of small leaves borne on short shoots and spurs that have proliferated along the main limbs (Fig. 10A). Affected leaves are chlorotic, drooped, and rolled downward. Fruits are deformed and undersized, and affected trees decline.

Peach rosette was described in 1881 (27). Affected trees may collapse suddenly and die or may produce tufts of leaves near the apex of otherwise bare limbs (Fig. 10B). Shoot growth is greatly reduced. The trees do not produce fruit and usually die within months.

Early work by Kunkel (19,20) on these diseases indicated that peach yellows and little peach were related but distinct from peach rosette. A hot water treatment of budwood affected with peach yellows or little peach showed that the pathogens survived 50 C for 2 minutes but not for 4 minutes. In contrast, heat-inactivation of peach rosette required more than 8 minutes. Also, graft-inoculation of peach with the agent of peach yellows protected against symptoms caused by the agent of little peach, and vice versa, but neither agent protected against peach rosette. Recent investigations employing 24 probes against genome fragments of the western X-disease MLO revealed that 18 of the DNA probes hybridized with DNA prepared from plants with peach yellows (16). In contrast, only two of 24 DNA probes hybridized with preparations made from tissues affected by peach rosette. These results suggest a close relationship between peach yellows and western X-disease and a distant one with peach rosette.

The MLOs associated with all three diseases (13,14,18) (Fig. 10C) are known to spread naturally. Peach yellows and little peach are vectored by the leafhopper *Macropsis trimaculata* (Fitch). No vector has been identified for peach rosette. Disease control consists of roguing diseased trees in the orchard and wild plum trees in the adjacent area. Use of insecticides for vector control is advised.

In California, two MLO-associated diseases are known in peach and sweet cherry trees: peach yellow leafroll (PYLR-MLO) (15) and western X-disease (WX-MLO) (26). The predominant symptoms of PYLR are yellowing and upward rolling of the leaves, accompanied by abnormally enlarged midribs and veins (Fig. 11A). Affected leaves drop prematurely. Fruits, if set occurs, are small and unpalatable. Trees gradually decline and usually die within 2–3 years. Several attempts to infect Bing sweet cherry trees with PYLR-MLO

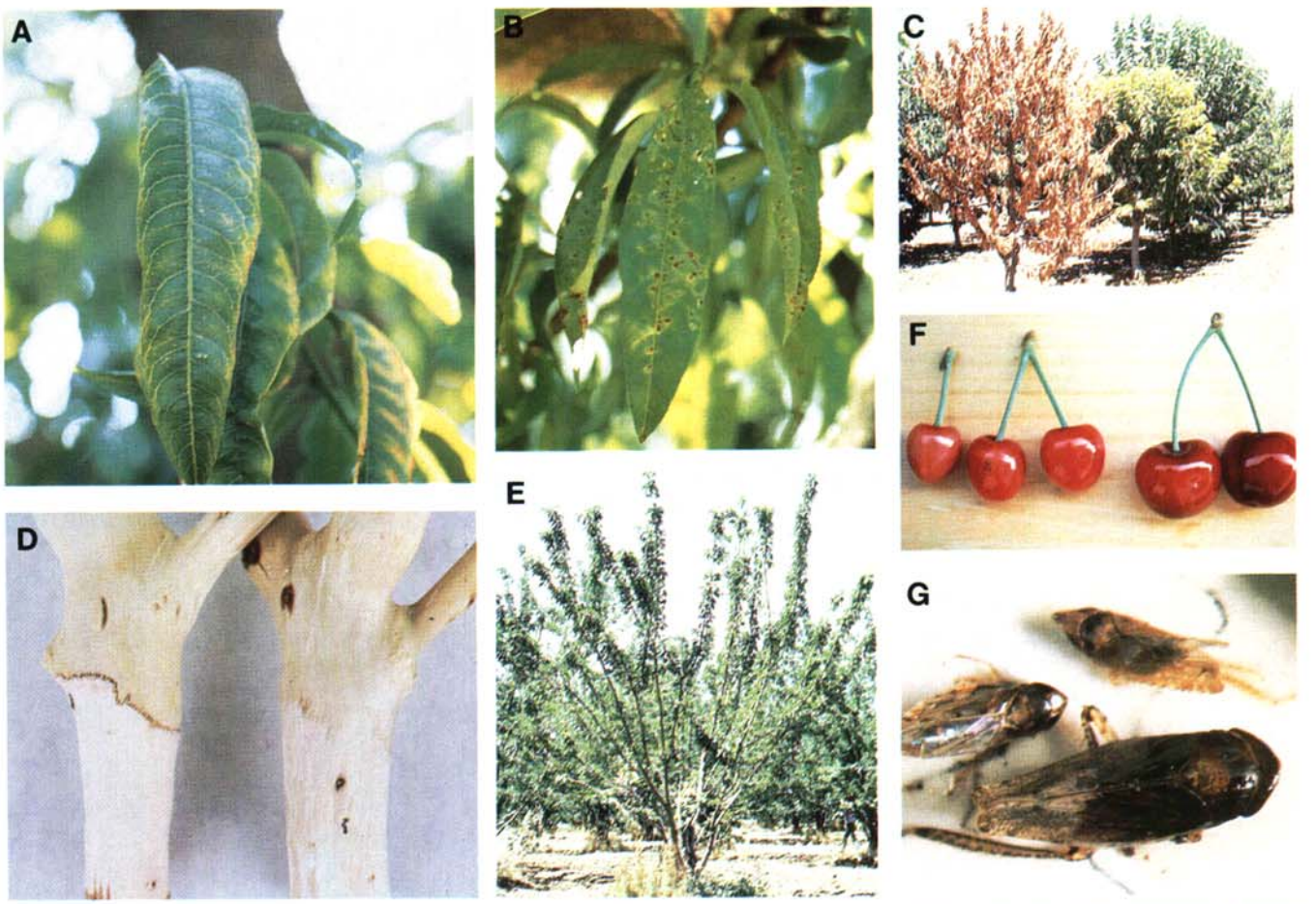

Fig. 11. (A) Enlarged midrib and primary veins associated with peach yellow leafroll. (B) Irregular chlorotic and necrotic blotches symptomatic of western X-disease on peach leaves. (C) Bing cherry trees on mahaleb rootstock dead or declining rapidly because of western X-disease. Symptomatic trees were graft-inoculated 10 months earlier. (D) Union areas of Bing cherry/mazzard trees (left) with and (right) without western X-disease. (E) Typical sparse canopy of a Bing cherry/mazzard tree with western X-disease. (F) Size of fruit from Bing cherry trees (left) with and (right) without western X-disease. (G) Vectors of X-disease mycoplasmalike organism include (top) *Scaphytopius acutus*, (middle) *Osbornellus* sp., and (bottom) *Paraphlepsius* sp.

have failed (J. K. Uyemoto, *unpublished*).

Symptoms of western X-disease in peach trees are distinct from those of PYLR. The leaf veins are never enlarged. Instead, the leaves are initially light green with irregular red and yellow blotches that turn necrotic and fall out, leaving a tatter-leaf appearance (Fig. 11B). In sweet cherry, the symptoms are influenced by the understock. Affected trees on mahaleb (*P. mahaleb*) roots decline rapidly and die after harvest (Fig. 11C). Although the sudden collapse of mahaleb-rooted trees is also associated with Phytophthora root and crown rot or other biotic or abiotic factors, X-diseased trees can be properly identified by removing the bark and examining the scion/rootstock junction. The union area of X-diseased trees shows numerous pits and grooves, i.e., union aberration (Fig. 11D) (39). In contrast, sweet cherry cultivars on mazzard (*P. avium*), Stockton morello (*P. cerasus* L. var. *austera* L.), or Colt (*P. avium* × *P. pseudocerasus*) slowly decline. The tree canopy is thin (Fig. 11E), reflecting poor shoot growth and small leaves. Also, small, pointed fruits are produced on short pedicels (Fig. 11F). No obvious symptoms have been observed in the woody cylinder.

Recent insect trapping in California has identified several potential leafhopper vectors of WX-MLO. Leafhopper species captured on yellow sticky traps included *Colladonus montanus montanus* (Van Duzee), *Fieberiella florii* (Stal), *Paraphlepsius* sp., *Osbornellus* sp., and *Scaphytopius acutus* (Say) (Fig. 11G) (J. K. Uyemoto, *unpublished*). These species and species within these genera are known to transmit WX-MLO. When captured leafhoppers were individually extracted, dot-blotted, and hybridized with a nucleic acid probe specific for the WX-MLO (17), one or more leafhoppers in each genera were dot-blot positive, indicating their infective potentials (J. K. Uyemoto, *unpublished*). The dot-blot-positive leafhoppers were captured in diseased cherry orchards.

Disease control measures may differ with the host. The results of a study by Suslow and Purcell (36) implied that peaches may be a dead-end host for PYLR-MLO. Repeated caging of *C. montanus* (involving some 4,500 leafhoppers) on peach trees during two seasons resulted in no apparent acquisition of the PYLR-MLO by the leafhoppers. However, because a single leafhopper species was used in that study (36), the implication that peach trees cannot serve as MLO hosts must be viewed with caution, and removal of symptomatic trees is prudent.

The results differed considerably when a similar access feeding trial on X-diseased sweet cherry trees produced infective leafhoppers. After a week's exposure on the host tree, the leafhoppers were returned to the laboratory, allowed to recuperate on healthy celery plants for a week, and then transferred at regular intervals onto fresh celery plants. Infected plants developed characteristic symptoms following incubation for 6–9 weeks. Access feedings in April resulted in zero acquisitions from diseased cherry and zero transmissions to healthy celery, but those in August–September from cherry produced over 20% infective leafhoppers that transmitted the pathogen to celery and mazzard seedlings.

In California, outbreaks of PYLR in peach orchards correlate closely with proximity to pear orchards, implying that pears may be a PYLR-MLO reservoir. There is no evidence of spread from peach to peach. With sweet cherry, however, the situation differs in that the primary MLO reservoir is the cultivated tree itself. The major cherry-growing region, San Joaquin County, does not have such alternate hosts as chokecherry (*P. virginiana* L.) and bitter cherry (*P. emarginata* (Douglas ex Hook.) Walp.), which are sources of MLOs in Michigan, New York, and Connecticut. Currently, the control strategy used in sweet cherry orchards of California involves the use of an insecticide on diseased trees or orchards before their removal. The practice of removing trees without spraying has commonly resulted in a more severe disease situation than before trees were removed (J. K. Uyemoto, *unpublished*). Presumably, infective leafhoppers were moving from the removed infected trees to nearby healthy ones.

Advice for the Grower

Overall, viruses and other graft-transmitted pathogens are common in stone fruits and almonds. Their wide range of effects can lead to severe reductions in yields or tree mortality, or both. Symptoms vary from the obvious to the subtle and can be confused with abnormalities that are caused by abiotic or other biotic factors.

To be aware of the risks caused by pathogens present in a particular area, a grower first should consult with the local cooperative extension fruit tree specialist and with a plant pathologist. Appropriate measures can then be taken, for example, soil fumigation for a soilborne virus disease known to be endemic there or removal of wild *Prunus* hosts that may be disease reservoirs. Second, and very important, the most effective and practical way to control many of these graft-transmitted pathogens is to establish orchards with stocks that arise from a virus-testing program. Many states have rules and regulations promulgated for clean stock programs. Caution is advised, however, as nursery participation in such programs is often on a voluntary basis. The grower should contact the state's tree nursery inspectors to learn of program participants.

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