Relationship Between Stem Pitting in Peach and in Other Prunus Species

Srecko M. Mircetich and E. L. Civerolo

Research Plant Pathologists, Plant Science Research Division, ARS, USDA, Beltsville, Maryland 20705.
Mention of trade names does not imply endorsement by the USDA over similar materials not so named.
Accepted for publication 23 May 1972.

ABSTRACT

Differential expression of stem pitting symptoms in various naturally infected species of stone fruits was observed. However, the stem pitting (SP) causal agent was readily graft-transmitted by cross inoculation of different *Prunus* spp. Stem pitting symptoms in inoculated indicator plants are similar to those in the same *Prunus* sp. naturally pitted regardless of the donor *Prunus* spp. Apparently, stem pitting in different *Prunus* is caused by the same, or related strains of the same, causal agent. Natural spread of stem pitting was demonstrated by annual surveys in densely planted apricot and sour cherry seedling orchards. Spread in the field may occur from an infected tree to trees of the same or different *Prunus* sp. The disease usually spreads from infected to adjacent trees with no random occurrence of newly infected trees in the orchards.

A virus recovered from soil of peach, nectarine, and apricot orchards infested with stem pitting was serologically related to the type culture of tomato ring spot virus and to two isolates of peach yellow bud mosaic virus. The same virus was also recovered from apricot, European plum, and Nanking cherry seedlings inoculated with root chips from naturally infected peach and apricot orchard trees. However, we found no correlation between the presence of this virus in soil and stem pitting in orchard trees, nor consistent association of this virus with experimentally infected-pitted indicator plants. Apricot seedlings planted to sites of naturally pitted peach orchard trees developed stem pitting within 5 months.

Phytopathology 62:1294-1302.

Additional key words: stone fruit trees, soil-borne virus disease.

Peach stem pitting is caused by a graft-transmissible agent (SP-agent), possibly a virus or viruses (15). Recently, a disorder of wide geographical distribution and unknown etiology resembling peach stem pitting has been described in several other *Prunus* species (1, 5, 8, 11, 13, 16, 19). The most characteristic symptom of stem-pitting disease in all naturally affected *Prunus* is the presence of grooves and pits in the woody cylinder of the lower trunk. However, differences in symptoms have been observed among *Prunus* spp., resulting in speculation that stem pitting in different *Prunus* spp. may not be caused by the same agent(s) (8, 13, 16). There is no conclusive experimental evidence on the nature of stem pitting in *Prunus* spp. other than peach, or on the relationship between stem pitting in peach and stem pitting in other stone fruit trees.

Mircetich et al. (12, 15) suggested a viral cause of peach stem pitting, observed spread of the disease in the field which appears to be effected by a soil-borne vector(s), and showed that the causal agent is not uniformly distributed in infected peach trees. SP-agent is readily transmitted from naturally infected peach, nectarine, and Chinese wild peach trees to peach seedlings (12). However, several attempts to transmit the SP-agent from naturally infected peach trees to apricot seedlings failed, although the agent was readily transmitted from experimentally infected peach seedlings to apricot seedlings (12). Lott (7) failed to transmit by bud inoculation the causal agent of xylem aberration (a disease with symptoms similar to those of peach stem pitting) from apricot to Elberta peach, Montmorency sour cherry, Italian Prune, and Shirofugen flowering cherry trees. However, this author reported only one case of transmission of stem pitting causal agent from apricot to chokecherry (*Prunus virginiana* var. *demissa*) trees. Mircetich et al. (12) reported recovery of a virus from small young shoots developing from the pitted trunk of apricot seedlings inoculated with root chips from naturally pitted trees. Attempts to transmit a virus mechanically from naturally pitted stone fruit species to herbaceous plants were unsuccessful (12, 15).

Elucidation of the nature of stem pitting disease in various *Prunus* spp. and its relationship to peach stem pitting should contribute to developing more effective control measures for this economically important disease.

The present study was undertaken to determine possible relationships between stem pitting disease in peach and other *Prunus* spp. We also investigated the occurrence of tomato ringspot virus in soil around naturally pitted and symptomless orchard trees, and its presence in naturally and experimentally pitted stone fruit trees.

MATERIALS AND METHODS.—*Prunus persica* L. Batsch, peach; *P. armeniaca* L., apricot; *P. domestica* L., European plum; *P. mahaleb* L., St. Lucie cherry; *P. cerasus* L., sour cherry; and *P. tomentosa* Thumb., Nanking cherry seedlings were grown in steam-pasteurized soil in 25-cm clay pots in the greenhouse. Ten seedlings of each *Prunus* sp. were inoculated as described (12) with buds or root chips from naturally or artificially infected trees of the following stone fruit species: peach, apricot, European plum, sour cherry, and Nanking cherry. The inoculated indicator plants were grown in the greenhouse for 8 to 14 months (12).

Soil samples collected around pitted and nonpitted orchard trees were assayed for the presence of virus using as bait cucumber (*Cucumis sativus* L. 'National Pickling') seedlings (17). We attempted also to transmit mechanically the causal agent(s) from

different naturally infected and experimentally inoculated *Prunus* spp. to cucumber and cowpea [*Vigna unguiculata* (L.) Walp. 'Early Ramshorn'] plants (12). Viruses recovered from soil and *Prunus* trees were serologically compared with each other and with known strains of peach yellow bud mosaic virus (PYBMV), tomato ring spot virus (TomRSV), and tobacco ringspot virus (TobRSV) in agar gel double-diffusion tests using 0.7% Ionagar (No. 2) with 0.2% NaN_3. Antigen sources were expressed sap from infected cucumber plants.

RESULTS.—*Variation of symptoms in naturally pitted Prunus spp.*—In general, all *Prunus* species affected by stem pitting show lack of terminal growth, various degrees of leaf chlorosis and discoloration, pitting and grooving associated with enations, and disorganization of xylem tissue in the lower trunk. Severity of these symptoms usually varies with the stage of the disease and with the species and cultivar. However, growth habit, thickness and necrosis of the bark, type and extent of pitting, and disorganization of the xylem tissue may differ among *Prunus* spp. Affected European plum, Japanese plum (*Prunus salicina* L.), and sour cherry trees usually have a pronounced weeping growth habit, whereas this type of growth is not observed in affected apricot or peach trees. Naturally pitted apricot trees show more pronounced enlargement at the lower trunk and thicker bark with cracks (Fig. 1-A, B) than do other stone fruit species affected by stem pitting. Type of pitting and amount of enations in the woody cylinder of affected apricot, however, may differ among cultivars (Fig. 2-A, B). This is similar to the difference reported in peach cultivars (14). Wood pitting in naturally infected apricot

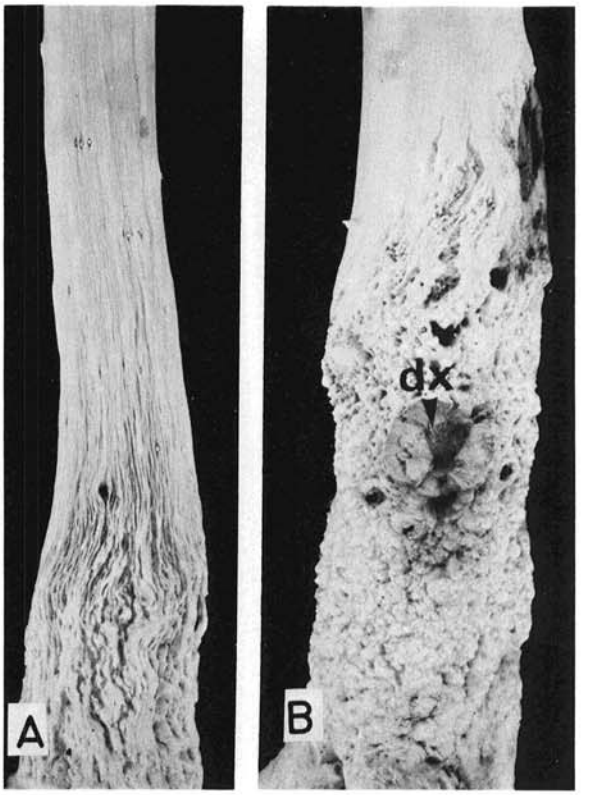

Fig. 2. Lower trunks of **A)** Perfection and **B)** Riland open-pollinated apricot seedlings naturally infected by stem pitting agent. Note difference in the type of stem pitting between A and B and disorganized xylem tissue (dx) at the point where lateral root broke off.

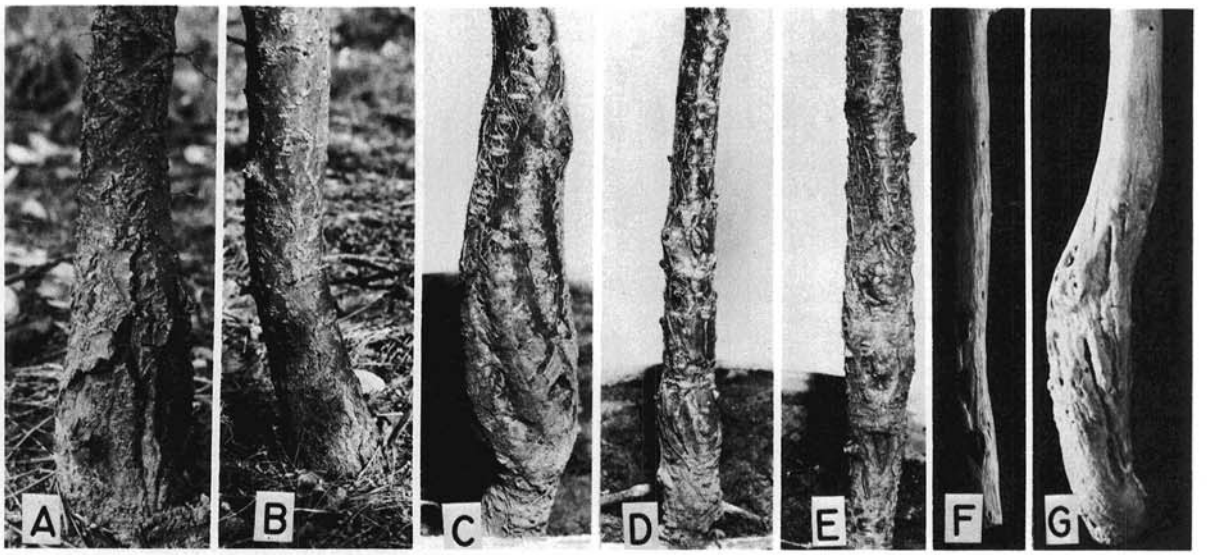

Fig. 1. A,B) Lower trunk of naturally pitted and apparently healthy apricot seedlings, respectively. Note deep cracks in the bark at the enlarged section of the trunk of A. **C,D)** Stem of apricot seedlings inoculated with root chips from naturally pitted and healthy peach seedlings, respectively. Note enlargement and crack in the bark of C similar to that in A. **E,F)** Stem and woody cylinder, respectively, of peach seedling inoculated with root chips from naturally pitted apricot seedlings. **G)** Woody cylinder of C showing stem pitting.

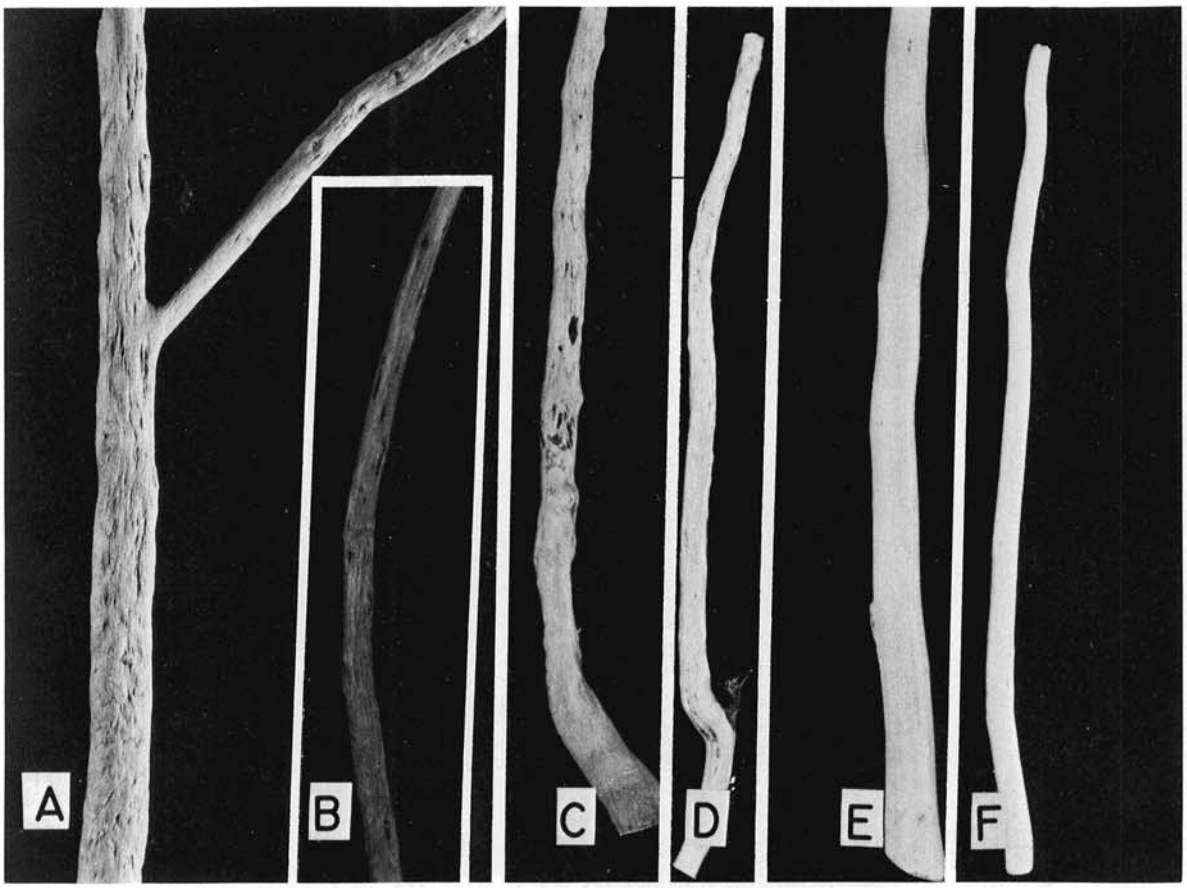

Fig. 3. A) Wood pitting in the leader and lateral branch of Montmorency sour cherry seedling naturally infected with stem pitting agent. **B)** Terminal shoot of A showing wood pitting. **C)** Lower stem of Montmorency sour cherry seedling inoculated with root chips from naturally pitted peach seedling. Note uniform pitting throughout the stem. **D)** Terminal shoot of C with wood pitting. **E,F)** Nonpitted stem and terminal shoot of Montmorency sour cherry received root chips from healthy peach seedling.

(Fig. 2-A, B) and peach trees (14) is restricted to the lower trunk, whereas in Montmorency seedlings and European plum (Smith Late, Edwards, Stanley cultivars, and Geneva No. 981 selection), wood pitting may occur throughout the trees, including the terminal growth (Fig. 3-A, B).

Interspecific graft-transmission of causal agents of stem pitting.—Since there is no conclusive experimental data on the nature of stem pitting in *Prunus* spp. other than peach, we investigated interrelation of stem pitting in different *Prunus* spp. by cross-inoculating various *Prunus* spp. (Table 1). The stem-pitting causal agent was graft-transmitted from naturally infected apricot trees to apricot and peach seedlings; from naturally infected peach to peach, apricot, European plum, Nanking cherry, and sour cherry seedlings; from naturally infected sour cherry to peach, apricot, Nanking cherry, European plum, and St. Lucie cherry seedlings; and from experimentally infected Nanking cherry to peach seedlings. Transmission of the causal agent was effected by both buds and root chips (Table 1). Root chips from naturally pitted apricot and peach trees that develop pitting only in the lower trunk and roots were more efficient in transmitting SP-agent than were buds. Buds from naturally infected sour cherry seedlings that develop pitting throughout the tree, including the terminal growth, were more efficient than buds of apricot and peach trees (Table 1). Apparently, SP-agent is more uniformly distributed in sour cherry than in apricot and peach trees. Stem-pitting symptoms in the greenhouse appeared within 3-6 months after inoculation, depending on the indicator species. Symptoms in indicator plants of the same species were similar regardless of the source of inoculum, and resembled those in the same naturally infected *Prunus* sp. Apricot seedlings, graft-inoculated with SP-agent from various sources, usually developed thick bark with deep cracks and wood pitting regardless of the source of inoculum (Fig. 1-C, D, G). There was no difference in symptoms in peach seedlings inoculated with the SP-agent from different *Prunus* spp. (Fig. 1-E, F, Fig. 4-E). Initial wood pitting in all graft-inoculated

TABLE 1. Interspecific graft-transmission of prunus stem pitting causal agent and recovery of tomato ringspot virus (TomRSV)[a] from indicator plants in the greenhouse tests

Source of inoculum	Recovery of TomRSV[a] from soil around inoculum source	Type of inoculum	Indicator (seedlings)	Indicators with stem pitting (%)	Pitted indicators from which TomRSV[a] was recovered (%)
Apricot seedling No. 66114, pitted[b]	-	Buds	F59-62 apricot	20	0
		Root chips	F59-62 apricot	100	60
		Buds	Halford peach	0	0
		Root chips	Halford peach	100	0
Apricot seedling No. 66300, pitted[b]	+	Buds	F59-62 apricot	0	0
		Root chips	F59-62 apricot	60	33
		Buds	Halford peach	20	0
		Root chips	Halford peach	100	0
Peach seedling No. 65200, pitted[b]	+	Buds	F59-62 apricot	20	0
		Root chips	F59-62 apricot	40	0
		Buds	Halford peach	40	0
		Root chips	Halford peach	60	0
Peach seedling No. 65243, pitted[b]	-	Buds	Perfection apricot	0	0
		Root chips	Perfection apricot	100	0
		Buds	Sunhigh peach	0	0
		Root chips	Sunhigh peach	100	0
		Buds	Stanley prune	80	0
		Root chips	Stanley prune	80	25
		Buds	Nanking cherry	20	0
		Root chips	Nanking cherry	80	0[f]
		Buds	Montmorency cherry	0	0
		Root chips	Montmorency cherry	100	0
Sour cherry seedling, pitted[b,c]	-	Buds	Perfection apricot	50	0
		Buds	Sunhigh peach	75	0
		Buds	Stanley prune	66	0
		Buds	Nanking cherry	100	0
		Buds	St. Lucie cherry	66	0
Nanking cherry artificially infected, nonpitted[d]		Buds	Sunhigh peach	20	0
Controls, nonpitted:[e]					
Apricot seedling		Root chips	Perfection apricot	0	0
Peach seedling		Root chips	Sunhigh peach	0	0
Sour cherry		Buds	Montmorency cherry	0	0
Nanking cherry		Buds	Nanking cherry	0	0

[a] Serologically related to TomRSV and to peach yellow bud mosaic virus; - = not recovered; + = recovered.
[b] Inoculum from a Plant Industry Station, Beltsville, Md., orchard tree, pitting restricted to the lower trunk.
[c] Pitting present throughout the tree, including the terminal shoots.
[d] Inoculated with root chips from naturally pitted peach seedling No. 65243. The buds from shoots developing near the original inocula and from terminal shoots that contained TomRSV were used as the inoculum.
[e] Grown in pasteurized soil in greenhouse.
[f] TomRSV was not recovered from any of the pitted plants, but was recovered from 20% of nonpitted plants that received the same inoculum.

Prunus occurred near the site of inoculation (Fig. 4-A, B, C, D, E), but never at or below ground level as observed in naturally infected trees (Fig. 2-A, B). Wood pitting in graft-inoculated seedlings of apricot, peach, St. Lucie cherry, and Nanking cherry was restricted to the inoculated trunks. However, 35% of Stanley prune and 75% of Montmorency sour cherry seedlings developed pitting throughout the plant, including the terminal growth, 1 year after inoculation (Fig. 3-C, D, E, F), similar to that observed in naturally infected trees (Fig. 3-A, B). There were no leaf symptoms in any artificially inoculated peach, apricot, European plum, St. Lucie cherry, and sour cherry seedlings, whereas 20% of Nanking cherry seedlings inoculated with root chips from pitted peach No. 65243 developed symptoms in leaves of terminal growth 6 weeks after inoculation. These symptoms consisted of leaf mottling, chlorotic rings, and slight distortion of very young leaves (Fig. 5). Since the SP-agent was readily transmitted by cross graft-inoculating different stone fruit species, and since the symptoms in experimentally infected

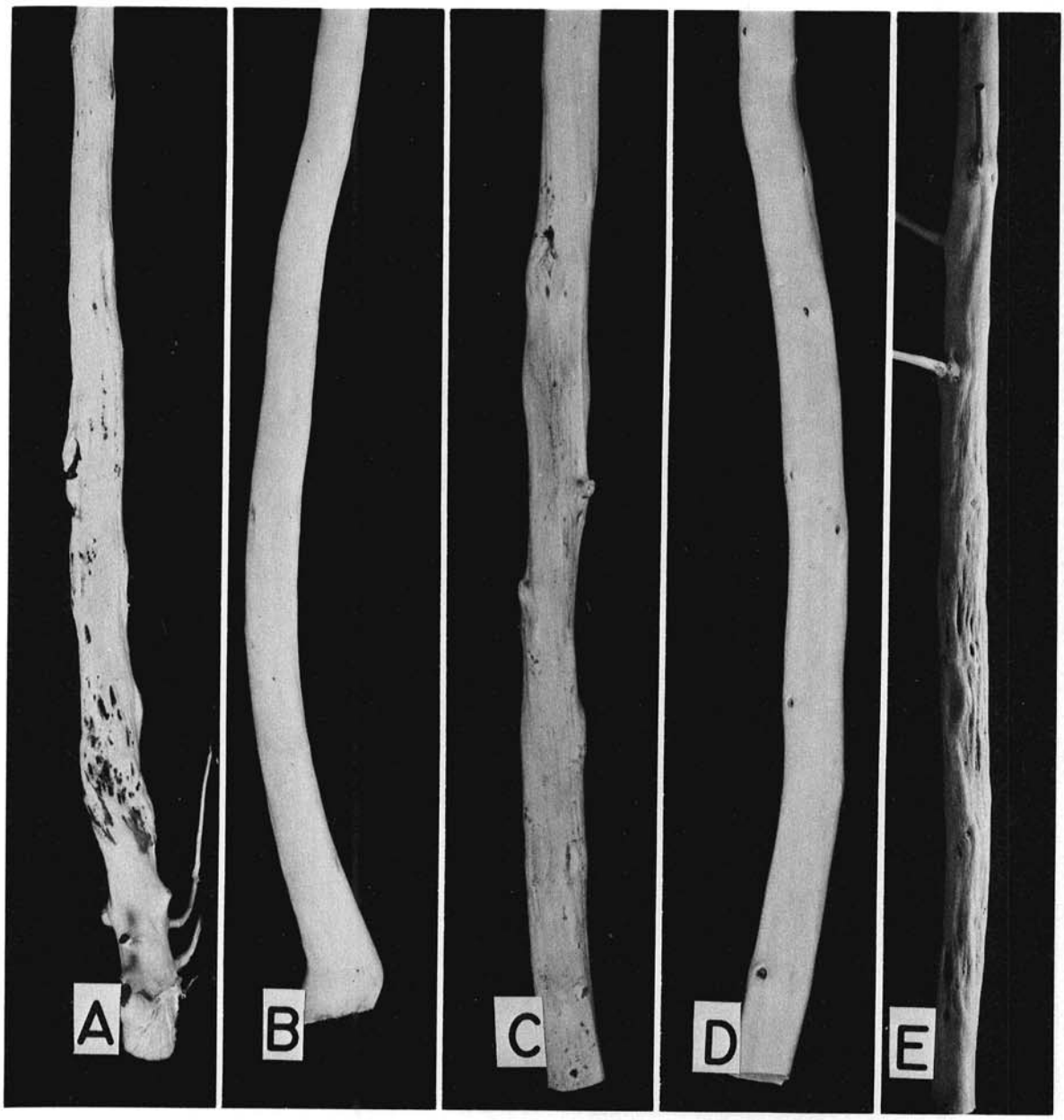

Fig. 4. Lower stem of different *Prunus* spp. seedlings inoculated with root chips from the same orchard peach trees in the greenhouse. **A, C, E)** Stanley prune, Nanking cherry, and Sunhigh peach seedlings, respectively, showing stem pitting that received inoculum from the naturally infected-pitted peach tree. Note the absence of stem pitting at the lower portion of the stems which was near the ground level. **B, D)** Stanley prune and Nanking cherry seedlings, respectively, that received the inoculum from healthy peach tree. Note the absence of pitting.

plants resembled those observed in naturally infected trees of the respective species regardless of the source of inoculum used, apparently stem pitting in all *Prunus* spp. is caused by the same, or related strains of the same, agent.

Incidence of soil-borne viruses in orchard soil.—To determine possible correlation of soil-borne viruses with naturally pitted stone fruit trees, soil samples were collected around 64 orchard trees that included nectarine [*P. persica* (L.) Batsch var. *nectarina* (Ait.) Maxim.], peach, apricot, and European plum. Thirty-nine and 25 soil samples from diseased and symptomless trees, respectively, were assayed for the presence of TomRSV and TobRSV (17). No virus was recovered from soil samples collected around eight European plum trees. Viruses serologically related to TomRSV or TobRSV were recovered from soil collected around both symptomless and pitted

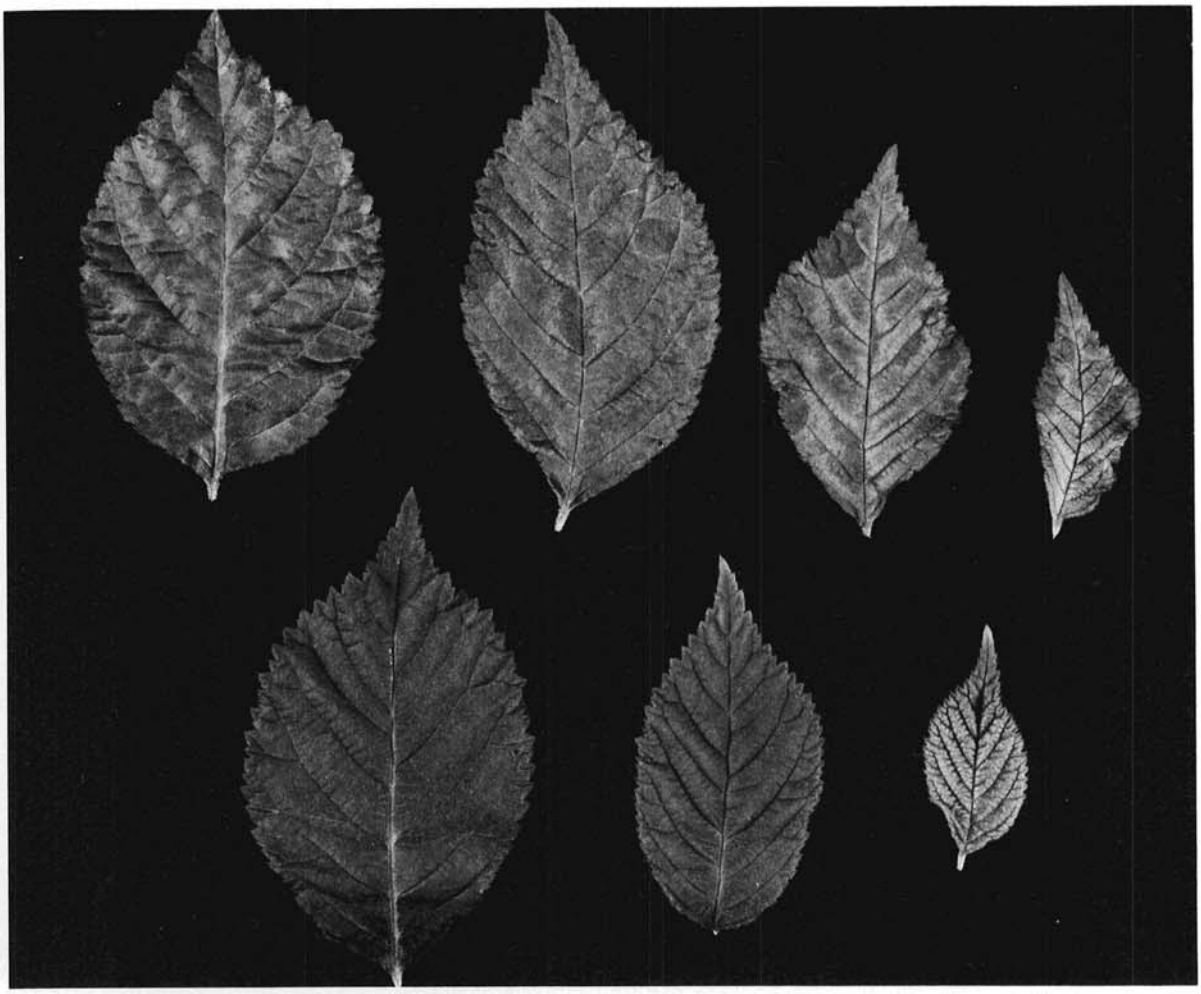

Fig. 5. Leaf symptoms in Nanking cherry seedling inoculated with root chips from naturally infected-pitted orchard peach tree (upper) and comparable leaves from the seedlings that received root chips from a healthy peach tree (lower).

nectarine, peach, and apricot orchard trees. A virus serologically related to TomRSV was recovered from 23% and 28% of soil samples collected around pitted and apparently health trees, respectively. A virus serologically related to TobRSV was recovered from 19% and 32% of soil samples collected around pitted and symptomless trees, respectively. Both TomRSV and TobRSV were occasionally recovered from soil around the same tree. Symptomless trees growing in soil containing these viruses remained symptomless for 3 years. Thus, these investigations failed to show any correlation between the presence of these viruses in soil and stem pitting in orchard trees. Repeated attempts to transmit a virus mechanically from leaves of terminal growth of naturally pitted and symptomless trees to cucumber and cowpea plants failed.

Incidence and distribution of TomRSV in pitted trees.—Wood pitting in naturally infected and experimentally inoculated apricot and peach trees is restricted to the lower trunk, and attempts to recover a virus from terminal growth of naturally infected trees were unsuccessful. We therefore attempted to recover common virus or viruses that may have limited distribution in *Prunus* spp. experimentally inoculated with buds or root chips from naturally pitted, stone fruit trees. Dormant buds located near the inoculum from various sources on stems of different indicator plants (Table 1) were induced to develop into shoots by a treatment with 1,000 μg/ml of gibberellic acid (GA_3) in Viscolan (American Cholesterol Products Co., Edison, N. Y.) or by spraying these buds with 400 μg/ml of GA_3 in water. Shoots developing from treated stem buds were observed for leaf symptoms. When these shoots were 10 to 15 cm long, we attempted to mechanically transmit virus from leaves of these stems, and the terminal shoots from each indicator plant to cucumber and cowpea plants.

No leaf symptoms developed on either stem or terminal shoots of inoculated apricot, peach, sour cherry, St. Lucie cherry, and European plum

regardless of type or source of inoculum used. However, 20% of Nanking cherry seedlings inoculated with root chips from naturally infected peach (No. 65243, Table 1) developed leaf symptoms (Fig. 5) on both the stem and terminal shoots. No virus was recovered from any indicator plant inoculated with buds from any naturally infected *Prunus* spp. However, a virus serologically related to TomRSV and to the virus present in soil of some orchard stone fruit trees was recovered repeatedly from stem shoots, but not from terminal shoots of pitted apricot and European plum seedlings that received root chips from three out of five inoculum sources. No virus was recovered from any of pitted Nanking cherry seedlings inoculated with root chips from naturally infected peach seedling No. 65243. However, 20% of nonpitted Nanking cherry seedlings inoculated with the same inoculum developed leaf symptoms from which the virus was readily recovered. When peach seedlings were subsequently inoculated with buds from stem and terminal shoots of nonpitted Nanking cherry seedlings that contained the virus, and had been originally inoculated with root chips from naturally pitted peach, 20% of inoculated peach seedlings developed stem pitting, although we repeatedly failed to recover TomRSV from any of the inoculated peach seedlings (Table 1).

Distribution of SP-agent in experimentally infected apricot seedlings.—To determine the distribution of SP-agent in experimentally infected apricot seedlings, buds and stem and root chips collected at various distances from the original graft-inoculum were used to inoculate Halford peach and apricot (F69-52) seedlings as described (12).

The source of inoculum was an apricot (F69-52) seedling with severe stem pitting that was originally inoculated with root chips from a naturally infected orchard apricot tree and from which we failed repeatedly to isolate any virus. The donor apricot seedling was grown for 14 months in the greenhouse after inoculation. Buds for inoculation were collected from the terminal growth 100 to 120 cm above the nearest pitted area in the stem. Stem chips were collected 10 to 30 cm from the original graft-inocula within the pitted area in the stem. Root chips were collected from nonpitted roots, but 15 to 20 cm distant from the pitting in the stem. Each of five Halford peach and apricot (F69-52) seedlings received three buds, stem chips, or root chips. Stem chips readily transmitted the apricot SP-agent to both peach and apricot seedlings. Root chips induced stem pitting only in apricot, whereas the buds failed to induce stem pitting in either apricot or peach seedlings (Table 2). Apparently SP-agent is not uniformly distributed in apricot as was the case with peach trees (12). Attempts to recover virus from stem shoots developing near the site of inoculation or from the terminal shoots of the indicator plants repeatedly failed.

Natural spread in the field.—Spread of the disease was determined by annual surveys in densely planted (5 X 15 ft) apricot and sour cherry seedling orchards. The orchards were surveyed each spring and fall for 3 years. Typically, spread in both orchards was from infected to adjacent trees. The orchards were planted in 1966 with ca. 500 trees each on sites that were previously planted to peach trees. In the spring of 1968, about 5% of the trees in each orchard were pitted, whereas 30% of the trees were pitted by November 1971. In 1969 and 1970, the spread was predominantly within the row. However, in 1971 the disease, in many instances, spread between the rows. Stem pitting also spread from peach to adjacent apricot seedlings, from apricot to adjacent sour cherry seedlings, and from Japanese plums to adjacent European plum trees. Newly pitted trees were not randomly scattered in the orchards, but they always occurred in clusters, suggesting underground spread and a soil-borne vector.

TABLE 2. Distribution of stem pitting causal agent in artificially infected apricot seedling[a] in a greenhouse test

Type and location of inoculum	Indicator (seedling)	Fraction[b] with stem pitting
Buds – 100 to 100-cm above pitted area on the stem	Blenril apricot	0/5
	Halford peach	0/5
Stem chips[c] – 10 to 30-cm from original inoculation site	Blenril apricot	5/5
	Halford peach	5/5
Root chips – 15 to 20-cm below pitted area on stem	Blenril apricot	2/5
	Halford peach	0/5
Control, noninoculated	Blenril apricot	0/5
	Halford peach	0/5

[a] Apricot seedling inoculated with root chips from naturally pitted apricot seedlings and grown for 14 months after inoculation in the greenhouse.

[b] Number of plants with stem pitting per number of plants inoculated.

[c] Woody portion was removed from chips before they were inserted in T-cuts of the indicators.

Soil transmission.—Surveys of numerous commercial peach orchards with naturally pitted trees often revealed the presence of younger replant trees with stem pitting. Stem pitting occurred in *Prunus* spp. seedlings planted on sites that were previously planted to peach trees. Cross sections of the lower trunk of pitted replant trees revealed disorganized xylem tissue only in the outer annual growth rings that developed after the trees were planted at the orchard site. Since these trees invariably develop initial pitting at the underground portion of the trunk but those experimentally graft-inoculated develop initial pitting at the point of inoculation (12), it appears that infection of the replants occurs through roots.

To determine possible transmission of prunus stem pitting through soil, 20 tree sites of pitted and nonpitted trees in a peach orchard were randomly selected and replanted to Perfection apricot seedlings. In order to determine the occurrence of soil-borne viruses and vectors in this orchard, soil samples were

collected at random around 22 pitted and nonpitted peach trees and assayed for the presence of nematodes and TomRSV. American dagger nematode (*Xiphinema americanum* Cobb) was detected in all soil samples. A virus serologically related to TomRSV and PYBMV was detected also in 23% and 33% of soil samples collected around pitted and nonpitted trees, respectively. Apricot seedlings from seed of a tree free of stem pitting were grown in steam-pasteurized soil in the greenhouse until planted in the field. Ten sites each of pitted and nonpitted trees were replanted to the apricot seedlings 3 months after removal of peach trees. Four of 10 seedlings planted in May at the sites of pitted peach trees developed typical symptoms within 5 months (Fig. 6-A). Seedlings planted at the sites of nonpitted trees remained symptomless (Fig. 6-B). Hence SP-agent is soil-borne, and can be transmitted through soil from peach to apricot.

DISCUSSION.—Although some differences in symptoms were observed among *Prunus* spp. infected with SP-agent, these investigations showed that stem pitting in all *Prunus* spp. is caused by the same, or related, strains of the same agent, possibly virus or viruses (15). We also showed that a virus serologically related to TomRSV may be associated with stone fruit trees affected by stem pitting. However, we found no consistent association of this virus with naturally or experimentally infected-pitted stone fruit trees (Table 1). Although the same virus may be present in soil and in pitted trees, we failed to establish positive correlation between its presence in soil and in the trees and the prunus stem-pitting disease.

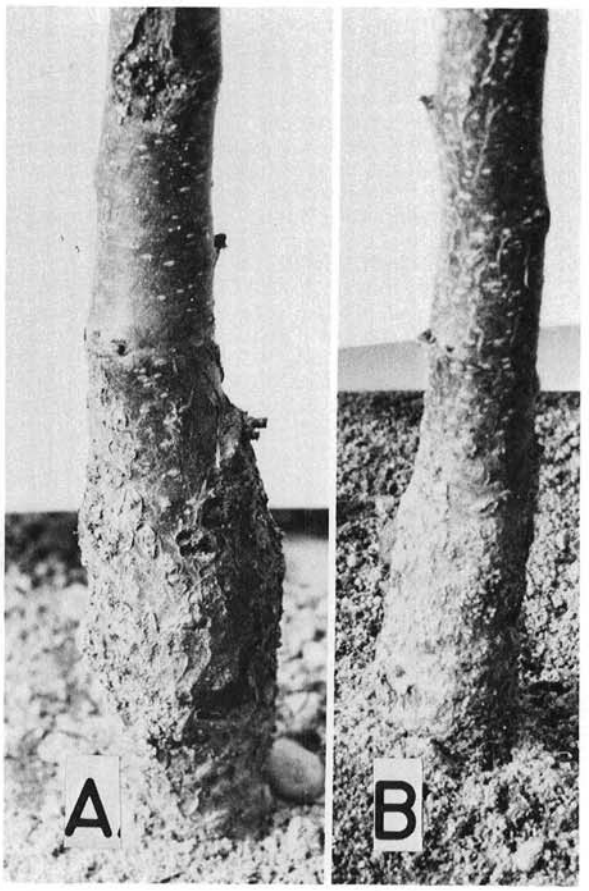

Fig. 6. A) Lower trunk of Perfection apricot seedling grown for 5 months at the site of pitted orchard peach tree. Note enlarged trunk at the ground level typical of naturally infected apricot trees. Compare this with Fig. 1-A and C. **B)** Control seedling at the site of nonpitted orchard peach tree.

Failure to demonstrate a consistent association between TomRSV and pitting in different *Prunus* spp. may be the result of either the absence of the virus in all pitted trees or the use of relatively inefficient methods for detecting this virus in trees. However, the virus recovered from some pitted trees, as with some known nematode-transmitted (NEPO) viruses, is soil-borne, polyhedral in shape (E. L. Civerolo, *unpublished data*), serologically related to TomRSV, and nonuniformly distributed through infected plants. NEPO viruses are usually readily transmitted mechanically to herbaceous plants (3, 9, 10, 20, 22). Since the virus associated with some pitted trees was repeatedly transmitted from leaves and bark of the same plant to cowpea and cucumber plants for 3 years, it appears that the techniques used in these studies are relatively reliable. However, repeated attempts to recover TomRSV from some pitted plants that received the same inoculum failed, suggesting that all pitted plants inoculated with the same inoculum may not contain this virus. Thus, any causal relationship of this virus and stem pitting remains to be elucidated either through satisfying Koch's postulate or by demonstrating the consistent association of this virus and pitting in different stone fruit species.

The occurrence of several different viruses in naturally pitted trees has been reported. Lott (7) reported stem pitting in ring pox and twisted leaf virus-infected stone fruit trees, but he concluded that these viruses are not the causal agents of stem pitting disease. Mircetich et al. (12) reported high incidence of necrotic ringspot virus (NRSV) in pitted peach trees in commercial orchards, but found no causal relationship between NRSV and stem pitting. Smith & Traylor (18) reported an association of stem pitting with peach trees exhibiting severe symptoms of peach yellow bud mosaic, and no stem pitting in trees with mild symptoms of this disease, but they did not establish the presence of PYBMV or other viruses in any of these trees. Cadman & Lister (4) concluded that PYBMV is a strain of TomRSV. Milbrath & Reynolds (9) isolated TomRSV from cherry affected by cherry rasp leaf virus (CRLV), but no wood pitting has been associated with this disease. We observed no symptoms resembling those of PYBMV (21,) in naturally or experimentally inoculated stone fruit trees that have been infected with SP-agent for over 3 years, whereas symptoms of PYBMV in experimentally inoculated peach seedlings through roots develop within 5-10 months (2). Furthermore,

apricot and European plum trees are severely affected with stem pitting and often die within 2-3 years after infection. However, PYBMV has not been observed in European plum, whereas older apricot trees are reported to be symptomless carriers of PYBMV (21). Thus, these investigations indicated that prunus stem pitting in the East and peach yellow bud mosaic may not be caused by the common strains of PYBMV (6).

Our investigations revealed that the prunus stem pitting agent is soil-borne; that *X. americanum* and a virus serologically related to TomRSV and PYBMV may be present in soil around some stone fruit trees affected by stem pitting disease; and that a virus serologically related to the soil isolates and to TomRSV and to PYBMV may be present in some pitted trees of certain *Prunus* spp. *X. americanum* is a vector of CRLV, TomRSV, and PYBMV (2, 3, 10, 20). However, the possible role of *X. americanum* as a vector of SP-agent remains unknown.

Since stem pitting is caused by the same graft-transmissible and soil-borne causal agent in different stone fruit species, and since it spreads relatively slowly in the field, the use of propagation materials from healthy trees is essential. Rogueing of pitted trees in nurseries and orchards should be practiced. Replanting of infested stone fruit orchard tree sites or the repeated use of infested nursery sites for stone fruit nurseries should be avoided. Eradication of both SP-agent and its vector from infested orchard soil may be technically difficult and economically prohibitive. However, differential pitting or tolerance has been observed in peach varieties and selections and among different *Prunus* spp. (13, 14). The SP-agent causes severe damage only in the lower trunks of certain stone fruit species. Therefore, we are testing various *Prunus* spp. for tolerance or immunity to stem pitting agents to be used as rootstocks for stone fruits.

LITERATURE CITED

1. AGRIOS, G. H. 1971. Premature foliation, cambial zone discoloration, and stem pitting of peach and apricot in Greece. Plant Dis. Reptr. 55:1049-1053.
2. BREECE, J. R., & W. H. HART. 1959. A possible association of nematodes with the spread of peach yellow bud mosaic virus. Plant Dis. Reptr. 43:989-990.
3. CADMAN, C. H. 1963. Biology of soil-borne viruses. Annu. Rev. Phytopathol. 1:143-172.
4. CADMAN, C. H., & R. M. LISTER. 1961. Relationship between tomato ringspot and peach yellow bud mosaic viruses. Phytopathology 51:29-31.
5. CAMERON, H. R. 1969. Stem pitting in Italian Prune. Plant Dis. Reptr. 53:4-6.
6. KARLE, H. P. 1960. Studies on yellow bud mosaic virus. Phytopathology 50:466-472.
7. LOTT, T. B. 1967. Xylem aberration, a transmissible disease of stone fruits. Can. Plant Dis. Surv. 47:74-75.
8. LOTT, T. B., F. W. L. KEANE, & J. MAY. 1962. Gumming, distortion and pitting in cherry and apricot. Can. Plant Dis. Surv. 42:229-232.
9. MILBRATH, J. A., & J. E. REYNOLDS. 1961. Tomato ringspot virus isolated from Eola rasp leaf of cherry in Oregon. Plant Dis. Reptr. 45:520-521.
10. NYLAND, G., B. F. LOWNSBERY, S. K. LOWE, & J. F. MITCHELL. 1969. The transmission of cherry rasp leaf virus by Xiphinema americanum. Phytopathology 59:1111-1112.
11. MIRCETICH, S. M. 1971. Peach stem pitting:history, distribution, economic importance, nature and natural spread. Nat. Peach Council Proc. 30:45-49.
12. MIRCETICH, S. M., E. L. CIVEROLO, & H. W. FOGLE. 1971. Relative differential efficiency of buds and root chips in transmitting the causal agent of peach stem pitting and incidence of necrotic ringspot virus in pitted trees. Phytopathology 61:1270-1276.
13. MIRCETICH, S. M., & H. W. FOGLE. 1969. Stem pitting in Prunus spp. other than peach. Plant Dis. Reptr. 53:7-11.
14. MIRCETICH, S. M., H. W. FOGLE, & J. G. BARRAT. 1968. Further observations on stem pitting in Prunus. Plant Dis. Reptr. 52:287-291.
15. MIRCETICH, S. M., H. W. FOGLE, & E. L. CIVEROLO. 1970. Peach stem pitting:transmission and natural spread. Phytopathology 60:1329-1334.
16. RAGOZZINO, A., & R. CAIA. 1968. Procssi cancrossi e gommosi, e butteratura del legno (stem pitting) associati a fenomeni di deperimento del Ciliegio dolce, Albicocco e Susino, in Campania. "Annali" della Facolta di Scienze Agrarie dell' Universita di Napoli, in Portici, Serie IV 3:3-8.
17. SMITH, S. H., & R. F. STOUFFER. 1970. Isolation of tomato and tobacco ringspot viruses from soil around stem-pitted Prunus trees. Phytopathology 60:1017-1018 (Abstr.).
18. SMITH, S. H., & J. A. TRAYLOR. 1969. Stem pitting of yellow bud mosaic virus-infected peaches. Plant Dis. Reptr. 53:666-667.
19. STOUFFER, R. F., F. H. LEWIS, & D. M. SOULEN. 1969. Stem pitting in commercial cherry and plum orchards in Pennsylvania. Plant Dis. Reptr. 53:434-438.
20. TELIZ, D., B. F. LOWNSBERY, R. G. GROGAN, & K. A. KIMBLE. 1967. Transmission of peach yellow bud mosaic virus to peach, apricot and plum by Xiphinema americanum. Plant Dis. Reptr. 51:841-843.
21. THOMAS, H. EARL, & T. E. RAWLINGS. 1951. Yellow bud mosaic. *In* Virus diseases and other disorders with viruslike symptoms of stone fruits in North America. U.S. Dep. Agr. Handbook 10:53-55.
22. YARWOOD, C. E. 1956. Mechanical transmission of peach yellow bud mosaic virus. Plant Dis. Reptr. 40:299.