

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

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ABSTRACT

Causal agent(s) of stem pitting was transmitted readily by root chips and erratically by buds from naturally infected peach, nectarine, and *Prunus davidiana* trees into peach seedlings. Likewise, graft-transmission of the stem pitting causal agent(s) from experimentally infected peach seedlings was effected readily by root and stem chips collected within 30 cm from the original graft inoculum. However, buds 96-113 cm distant from the pitted area from the same source failed to induce stem pitting in the indicator plants. Apparently, peach stem pitting causal agent(s) may not be distributed uniformly in the infected trees. All symptoms of

the disease were reproduced in the graft-inoculated indicator plants in the greenhouse within 5 months after inoculation. These investigations revealed a high incidence of necrotic ringspot virus (NRSV) in pitted peach trees in commercial orchards. However, there was no correlation between the presence of NRSV and stem pitting or the severity of stem pitting symptoms in naturally or experimentally infected peach trees. NRSV failed to induce stem pitting in artificially inoculated peach seedlings. Therefore, the results suggest that NRSV apparently is not the primary cause of peach stem pitting. *Phytopathology* 61:1270-1276.

Additional key words: *Prunus persica*, *P. armeniaca*, virus disease.

Peach stem pitting is caused by graft-transmissible agents (8). Both buds and root chips transmitted the agent from naturally infected trees into peach seedlings. However, the pattern of occurrence of stem pitting in budded trees in the nursery row, as well as in our numerous transmission experiments in the greenhouse, suggests that the causal agent(s) may not be distributed uniformly throughout an infected tree. A better knowledge of the distribution of the causal agent(s) in infected trees, and of relative efficiency of various tissues of these trees in transmittance of the agent(s), should contribute to development of more reliable indexing, early detection methods, and control measures for peach stem pitting.

Mircetich et al. (8) occasionally observed a variety of leaf symptoms in Sunhigh peach seedlings inoculated with buds or root chips from peach trees naturally infected by stem pitting in commercial orchards. There was no correlation, however, between these leaf symptoms and stem pitting, either in naturally infected peach trees or in the indicator plants. Stouffer & Lewis (10) reported that three of 16 pitted peach trees and none of 11 symptomless peach trees reacted positively to the Shirofugen test for necrotic ringspot virus. We failed consistently to associate any plant-pathogenic microorganism with peach stem pitting. Graft-transmissibility and natural spread of the disease suggest that peach stem pitting may be of viral nature. Therefore, association of any plant virus with pitted trees needs elucidation of its possible role in peach stem pitting.

We present data on relative efficacy of buds and root chips in transmitting peach stem pitting, and evidence that the causal agent(s) may not be distributed uniformly in infected trees. Also reported are

high frequency of association of necrotic ringspot virus with pitted trees in commercial orchards, and the evidence that this virus is not the primary cause of peach stem pitting.

MATERIALS AND METHODS.—*Prunus persica* (L.) Batsch 'Halford' peach seedlings were grown in steam-pasteurized soil in 18.9-liter cans. Ten peach seedlings/inoculum source were inoculated in the greenhouse with buds or root chips from stem-pitted or symptomless orchard trees of the following species: peach, *P. persica*; nectarine, *P. persica* (L.) Batsch var. *nectarina* (Ait.) Maxim.; Chinese wild peach, *P. davidiana* (Carr.) Franch; and apricot, *P. armeniaca* L. The inoculum consisted of three buds or root chips/peach seedling, inserted into T-cuts on the seedling trunks (1.5-2.0 cm diam) within 24 hr after inoculum collection. Five Halford peach seedlings were inoculated with buds (three buds/seedling) from each sour cherry, *P. cerasus* L., infected with necrotic ringspot virus (NRSV-A) or (NRSV-G) (6) or prune, *P. domestica* L., infected with prune dwarf virus (PDV), apricot gum strain (PDVA), or PDV. Controls consisted of unbudded seedlings or seedlings that receiver buds or root chips from healthy peach seedlings raised in steam-pasteurized soil in the greenhouse. The indicator plants were pruned when inoculated, and for every 6 months thereafter. The seedlings were fertilized weekly with 20-20-20, soluble greenhouse fertilizer and Nutra-min element concentrate (E. C. Geiger, Box 285, Route 63, Harleysville, Pa.). Inoculated peach seedlings were sprayed with insecticides every 10 days to control insects, and received supplemental light to secure continuous vegetative growth throughout the experimental period of 22 months.

We attempted to mechanically transmit stem pit-

ting causal agents and/or viruses present in naturally infected and experimentally inoculated peach seedlings. Leaves, roots, or gall-like tissues were triturated in 0.03 M potassium-phosphate buffer, pH 7.5, with or without 0.01 M sodium diethyldithiocarbamate. The homogenate was rubbed onto Carborundum-dusted (400 mesh) cucumber (*Cucumis sativus*) L. 'National Pickling' cotyledons.

RESULTS.—Preliminary observations and experiments.—Affected peach trees in the nursery often occur in groups of several adjacent trees in the row. These groups of pitted trees usually are separated within the row by a much larger number of apparently healthy ones, even though the budwood used in propagating both diseased and symptomless trees originated from the same peach tree. Since it is common practice in nursery propagation to use all suitable buds from a single budstick in succession, and since numerous budsticks from a single tree may be used to propagate a large number of trees, this observation suggests that only buds from certain budsticks of a single tree may contain and be effective in transmitting stem pitting causal agent(s).

To determine uniformity of stem pitting transmission in the nursery by using buds from different budsticks of the same naturally infected peach tree, 20 budsticks were collected at random. Inoculated Halford seedlings planted 1.5 m apart were grown for 2 years at a site which has never been planted to peaches. Each seedling received three buds from a single budstick. Inoculated seedlings were in groups of five separated by five noninoculated seedlings in the same row. Two of 20 inoculated plants developed stem pitting. No stem pitting was observed in non-inoculated controls. Therefore, buds from only two of 20 budsticks were effective in transmitting the causal agent(s).

In 1967, we failed in several experiments to induce stem pitting in Sunhigh peach seedlings inoculated in the greenhouse with buds from naturally infected peach trees. However, in 1968, when we used root chips from the same sources as inoculum, four or five of five inoculated Sunhigh seedlings developed severe pitting within 6 months after inoculation. These experiments suggest that root chips are more effective than buds in transmitting the causal agent(s) of the disease.

Efficacy of buds and root chips in transmitting causal agent of stem pitting.—To determine a possible differential transmission efficacy of root chips and buds, we carried out experiments in which five Halford seedlings were inoculated by root chips or buds from the same naturally infected tree. Fifteen budsticks or root pieces (ca. 10 cm long and 0.5-1.0 cm diam) collected from each tree in April served as source of buds or root chips for the inoculation. Budsticks were selected from terminal growth at random. Root pieces were collected at random within 60 cm from the tree trunk. Each indicator plant received three buds or root chips, but only one bud or root chip from a single budstick or root piece. Thus, every inoculated plant contained buds or root chips from

TABLE 1. Relative efficacy of buds and root chips in transmitting the causal agent(s) of peach stem pitting, incidence of necrotic ringspot virus (NRSV) in peach, and failure of NRSV strains to cause stem pitting

Source of inoculum	Type of inoculum	Fraction ^a with	
		Stem pitting	Symptoms of NRSV
Peach cultivar or seedling:			
Control	None	0/10	0/10
Sunhigh seedling nonpitted ^b	Buds	0/5	0/5
	Root chips	0/5	0/5
Seedling 53624, pitted ^c	Buds	0/5	0/5
	Root chips	0/5	0/5
Seedling H-79, nonpitted ^{c,d}	Buds	0/5	0/5
	Root chips	1/5	0/5
Seedling 65113, pitted ^c	Buds	1/5	0/5
	Root chips	5/5	0/5
Seedling 65100, nonpitted ^c	Buds	0/5	0/5
	Root chips	0/5	0/5
Seedling 65116, pitted ^c	Buds	1/5	0/5
	Root chips	5/5	0/5
Seedling 65110, nonpitted ^c	Buds	0/5	0/5
	Root chips	0/5	0/5
Sunhigh, pitted ^e	Buds	0/5	5/5
	Root chips	4/5	3/5
Sunhigh, non-pitted ^e	Buds	0/5	5/5
	Root chips	4/5	3/5
Sunhigh, pitted ^e	Buds	0/5	5/5
	Root chips	5/5	5/5
Blake, pitted ^f	Buds	0/5	5/5
	Root chips	4/5	4/5
Blake, pitted ^f	Buds	0/5	5/5
	Root chips	5/5	5/5
Blake, pitted ^f	Buds	1/5	5/5
	Root chips	5/5	5/5
Blake, non-pitted ^f	Buds	0/5	2/5
	Root chips	0/5	1/5
Sour cherry cultivar: ^g			
Montmorency, nonpitted, NRS, virus A	Buds	0/5	5/5
Montmorency, nonpitted, NRS, Virus G	Buds	0/5	5/5
Plum: ^g			
Plum, nonpitted, PDVA, apricot gum strain	Buds	0/5	0/5 ^h
Plum, nonpitted, PDV	Buds	0/5	0/5 ^h

^a Number of plants with symptoms per number of plants inoculated.

^b Grown in pasteurized soil in greenhouse.

^c Inoculum from Plant Industry Station, Beltsville, Md., orchard tree.

^d Nonpitted and apparently healthy when inoculum collected (April 1968). The tree showed pitting 1 year later.

^e Inoculum from Arden, W. Va., commercial orchard tree.

^f Inoculum from Ellicott City, Md., commercial orchard tree.

^g Inoculum supplied by R. P. Kahn, Plant Quarantine Division, ARS, USDA, Plant Introduction Station, Glenn Dale, Md. 20769.

^h Two of five inoculated plants developed leaf symptoms of PDV (prune dwarf virus).

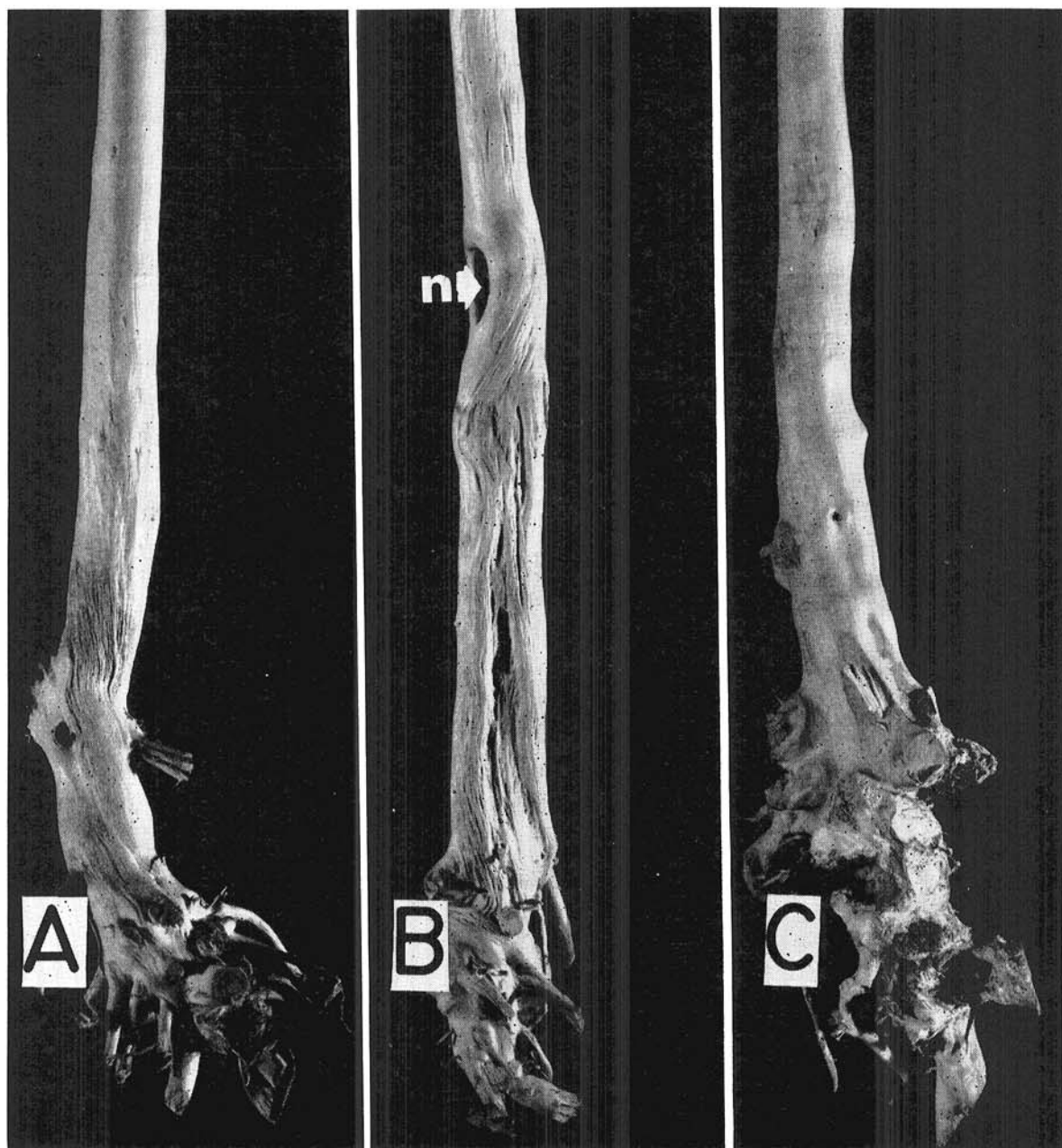


Fig. 1. Lower stem of Halford peach seedlings inoculated in greenhouse. **A, B)** With buds and root chips from naturally infected orchard peach tree, respectively. Note pitting and necrotic area (n) in B developed under inoculum. **C)** Control, inoculated with root chips from healthy peach tree.

three different budsticks or root pieces originating from the same tree.

Causal agents of peach stem pitting were transmitted by both root chips and buds from naturally infected peach trees into peach seedlings (Fig. 1). However, there was a marked difference in efficiency of transmission between these two types of inoculum (Table 1). This difference was reflected in both the number of transmissions per the number of sources indexed, and the number of indicators with pitting

per single tree indexed. Root chips from seven of eight naturally infected, pitted trees induced stem pitting in the indicator plants. However, buds from only three of the same eight inoculum sources induced pitting in the indicator plants. In the majority of cases where buds failed to transmit the causal agent(s), the root chips from the same source induced pitting in four or five of five inoculated plants (Tables 1, 2). Five of 13 inoculum sources were symptomless when the inoculum was collected (Tables 1, 2). However,

TABLE 2. Transmission of stem pitting agent(s) from *Prunus* spp. other than peach to Halford peach seedlings in greenhouse tests

Source of inoculum	Type of inoculum	Fraction ^a with	
		Stem pitting	Symptoms of necrotic ringspot virus
Nectarine cultivar: ^b			
September Grand, pitted	Buds	0/5	5/5
	Root chips	5/5	5/5
La Grand, pitted	Buds	0/5	5/5
	Root chips	4/5	5/5
September Grand, nonpitted	Buds	0/5	5/5
	Root chips	0/5	5/5
Chinese wild peach: ^c			
Seedling, pitted	Buds	1/5	0/5
	Root chips	3/5	0/5
Seedling, nonpitted	Buds	0/5	0/5
	Root chips	0/5	0/5
Apricot: ^c			
Seedling, pitted	Buds	0/5	0/5
	Root chips	0/5	0/5
Seedling, nonpitted	Buds	0/5	0/5
	Root chips	0/5	0/5
Control	Noninoculated	0/5	0/5

^a Number of plants with symptoms per number of plants inoculated.

^b Inoculum from Welch Run, Pa., commercial orchard trees.

^c Inoculum from Plant Industry Station, Beltsville, Md., orchard trees.

root chips of two of five symptomless sources induced stem pitting in the indicators. Stem pitting was observed in these trees 15 months after the inoculum was collected. Therefore, the causal agent(s) was present in some peach trees even though the trees appeared symptomless.

Extent and severity of pitting, cambial and bark necrosis, enlargement of trunk, and disorganization of xylem tissue within the woody cylinder of the trunk increased with the time after inoculation. The first indication of possible positive transmission was manifested by suppressed growth of cambial tissue under and adjacent to the inoculum. This usually was followed by necrosis of cambial tissue under the inoculum. The inoculum may die eventually, but may remain attached to the stem. Definite symptoms of stem pitting appeared in the indicators 5 months after inoculation in the greenhouse. Five months after inoculation, the peach seedlings developed conspicuous sunken areas on the trunk at the point of inoculations. The enlarged trunk becomes flattened and ridged (Fig. 2-A), and pitting and grooving develops in the woody cylinder. Initial pitting in naturally infected orchard trees always occurs at or below ground level, and extends for a short distance into the lower trunk and roots (Fig. 2-B). The initial wood pitting of inoculated peach seedlings occurs usually at the opposite side of the inoculum, but never at or below the

ground level (Fig. 2-C). Pitting in some indicator plants advanced 65 cm above the highest inoculation points, and 12 cm below the lowest inoculation point on the stems within 22 months. Similarly, necrosis of the cambial tissue in the inoculated peach seedlings occurs first under the inoculum (Fig. 1-B), then may gradually spread outward in any direction from the inoculum (Fig. 2-D, E). Occasionally, small areas of necrotic cambium and bark may occur at any point within the pitted area on the trunk of inoculated peach seedlings. These particular symptoms often are observed in naturally infected trees of Halford and several other peach cultivars. Longitudinal sections through pitted woody cylinder of indicator plants revealed disorganized xylem tissue within annual growth rings that developed after inoculation (Fig. 2-F). This particular symptom is strikingly similar to that observed in naturally infected peach trees that remained healthy for a certain period in the orchard before they became infected (Fig. 2-G).

Transmission from other Prunus spp. to peach seedlings.—The stem pitting causal agent was graft-transmitted from naturally infected nectarine and Chinese wild peach to peach seedlings in the greenhouse (Table 2). Root chips were more effective than buds in transmission (Table 2), as was the case in the transmission from peach to peach (Table 1). In this, as in several previously conducted experiments, we failed to transmit the stem pitting agent from naturally infected apricot to peach seedlings (Table 2). However, the stem pitting agent from experimentally infected peach seedlings was graft-transmitted to apricot seedlings (Table 3). Furthermore, in three experiments which are still in progress, the stem pitting agent was successfully graft-transmitted from naturally and artificially infected apricot to peach and from peach back to apricot seedlings.

Incidence of NRSV in peach trees affected with stem pitting.—All 10 inoculum sources from commercial peach orchards induced in inoculated Halford seedlings a variety of symptoms similar to those described for NRSV (4) (Tables 1, 2). These symptoms were vein clearing, leaf mottling, shot hole, chlorotic rings, and occasionally oak leaf pattern (Fig. 3), necrosis and dieback of young new growth, and cankering associated with gumming in the woody terminal growth. Similar symptoms were developed in Halford seedlings graft-inoculated with NRSV-A and -G. In several experiments, none of these symptoms was observed in Halford seedlings inoculated with inoculum sources from 6- to 14-year-old peach trees grown in orchards at Plant Industry Station, Beltsville, Md. However, none of the indicators inoculated with either isolate of NRSV, or with PDV isolates, developed stem pitting, whereas 8 of 10 inoculum sources from the commercial orchards induced stem pitting in the indicator plants (Tables 1, 2). NRSV, A and G, and a virus from inoculated peach seedlings with NRSV-like leaf symptoms were mechanically transmitted to cucumber plants; repeated attempts to mechanically transmit virus or viruses from artificially

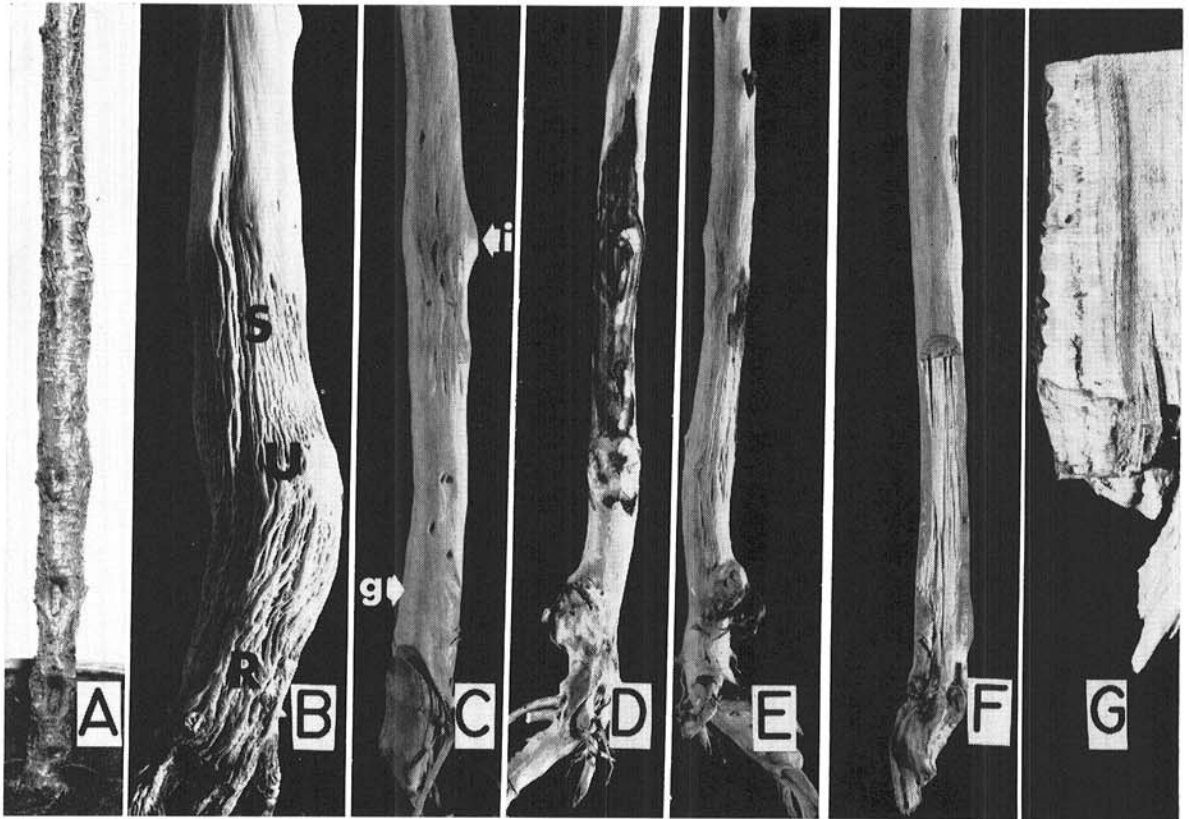


Fig. 2. A) Root chip-inoculated Halford peach seedling with early symptoms of positive transmission of stem pitting. Note depression and sunken areas under inocula, and slightly enlarged stem with ridges. B) Young, naturally infected peach tree showing severe stem pitting which initially developed in underground rootstock (R), then advanced across the union (U) into the lower portion of the scion (S). C) Experimentally inoculated Halford peach seedling with initial pitting, opposite inoculum (i). Note the absence of pitting on the portion of the stem below the ground (g) level. D) Necrotic area around inocula associated often with positive transmission of stem pitting. E) Same as D showing pitting opposite the inocula. F, G) Longitudinal sections through lower trunk of experimentally and naturally infected peach trees, respectively, showing disorganized xylem in the outer annual growth rings.

or naturally pitted trees from Beltsville orchards that had no leaf symptoms failed. Similarity of the leaf symptoms in inoculated peach seedlings induced by NRSV, strains A and G, and the virus associated with peach trees from the commercial orchards indicated that inoculum sources from commercial orchards contained NRSV regardless of stem pitting. NRSV strains A and G and the virus mechanically transmitted from indicator plants with leaf symptoms induced symptoms in cucumber plants similar to those described for NRSV (1). Also, the virus recovered from indicator plants inoculated with root chips from Arden inoculum sources was serologically related to NRSV. This isolate has physical and biological properties similar to those of other NRSV isolates (*unpublished data*). There was no correlation between NRSV and stem pitting, either in naturally or experimentally infected indicator plants. Transmission of stem pitting causal agents was more efficient by root chips than by buds, while both types of inoculum transmitted NRSV equally well (Tables 1, 2, 3). Apparently, NRSV is frequently associated with pitted and symptomless

peach trees in commercial orchards, but this virus is not the primary cause of peach stem pitting.

Distribution of peach stem pitting agent in experimentally infected peach seedling.—To determine distribution of the peach stem pitting causal agent(s) in experimentally infected peach seedlings, buds, stem, and root chips collected at various distances from the original graft inoculum were used to inoculate Halford peach and apricot seedlings. Inoculated indicators were grown in 25-cm clay pots in steam-pasteurized soil for 10 months in the greenhouse.

The source of inoculum was Halford seedling with pronounced stem pitting and NRSV symptoms, originally inoculated with root chips from naturally infected orchard peach tree and grown for 2 years in the greenhouse after inoculation. Buds for the inoculation were collected 96-113 cm above the nearest pitting area in the stem. Stem chips were collected 10-30 cm from the original graft inoculum within the pitted area in the stem. Root chips were collected from non-pitted roots, but 13-17 cm distant from the nearest pitting in the stem. Each of five Halford peach and

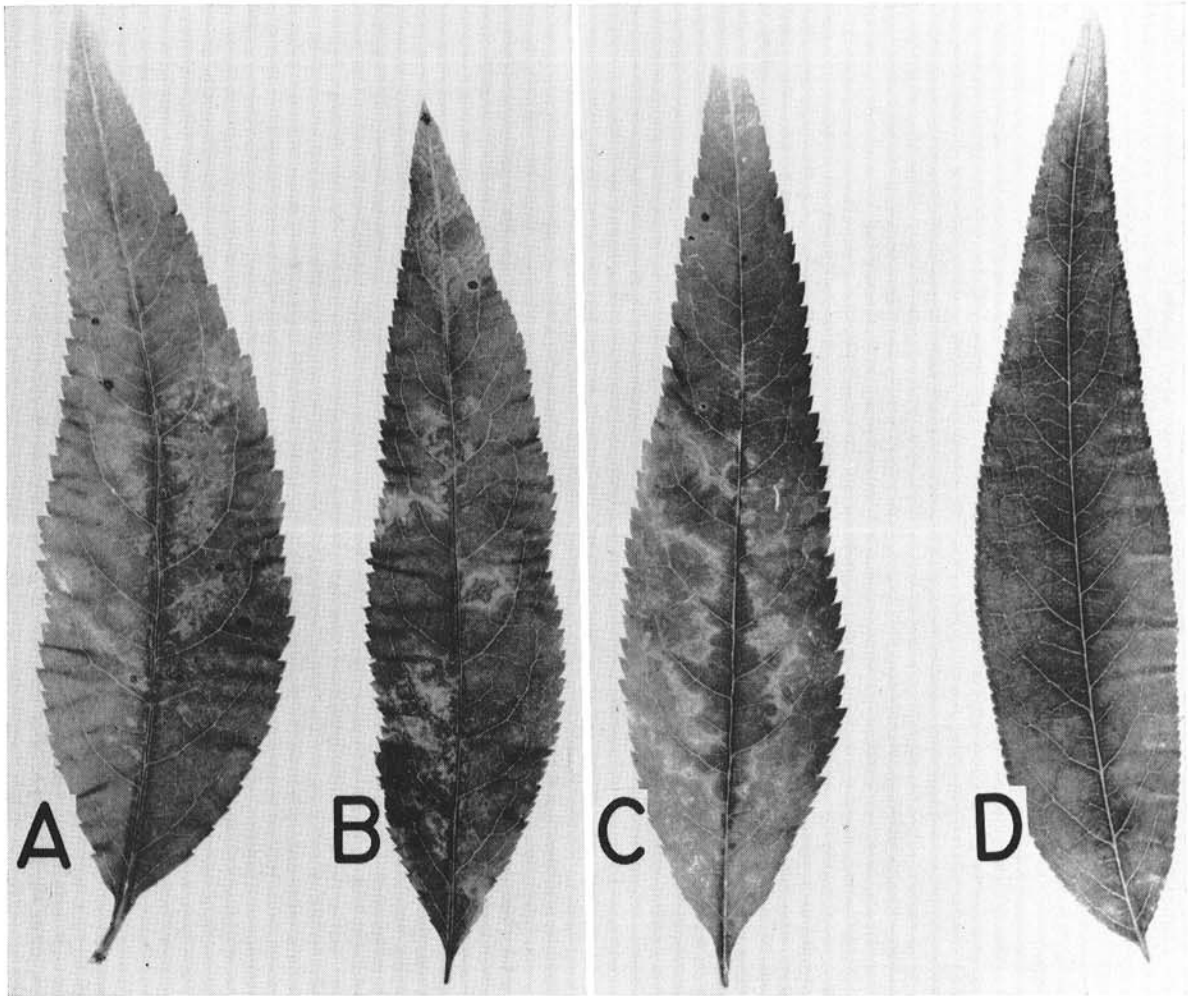


Fig. 3. A, B, C) Various leaf symptoms in Halford seedlings inoculated by buds or root chips from commercial orchard peach trees. These symptoms are not indicative of stem pitting either in naturally or experimentally infected trees. D) Symptomless leaf from peach seedling with severe stem pitting shown in Fig. 1-B.

apricot seedlings received three buds, stem chips, or root chips. Controls consisted of noninoculated seedlings. Inoculated Halford seedlings developed NRSV symptoms regardless of the inoculum used, while those symptoms were not observed in the inoculated apricot seedlings (Table 3). Attempts to mechanically transmit NRSV to cucumber plants from the apricot seedlings failed, but this virus was easily recovered from the peach indicator plants. Stem and root chips readily transmitted the peach stem pitting agent(s) to both peach and apricot seedlings; but buds from the same inoculum source failed to induce pitting in the indicator plants (Table 3). Apparently, the stem pitting causal agent(s) was not distributed uniformly throughout the infected peach while NRSV was.

DISCUSSION.—Mircetich et al. (8) reported recently that peach stem pitting is caused by infectious and graft-transmissible agent(s). These investigations showed that the causal agent(s) can be transmitted

readily by root chips and erratically with buds from naturally infected-pitted trees into peach seedlings (Tables 1, 2). Likewise, graft transmission of the stem pitting causal agent(s) from experimentally infected peach seedlings to peach and apricot seedlings was affected only by root and stem chips collected within 30 cm from the original graft-inoculum. Apparently, the causal agent(s) had limited distribution in the infected peach seedling even 2 years after inoculation; or the concentration of the agent(s) was not sufficient in the buds collected 96-113 cm from the inoculation point to induce stem pitting in the indicator plants. Thus, these results indicate that the causal agent(s) may not be distributed uniformly throughout affected trees. The situation with peach stem pitting resembles that demonstrated for phony virus disease of peach and other stone fruits (7). "Virus" of phony disease is transmitted uniformly with root and erratically with scion inocula. Several plant-pathogenic graft-trans-

TABLE 3. Transmission of peach stem pitting agent(s) from experimentally infected peach seedlings^a to Halford peach and apricot seedlings in greenhouse tests

Type of inoculum	Indicator (seedling)	Fraction ^b with	
		Stem pitting	Symptoms of necrotic ringspot virus ^c
Buds ^d	Halford peach	0/5	5/5
Stem chips ^e	Halford peach	5/5	5/5
	Apricot	5/5	0/5
Root chips ^f	Halford peach	5/5	5/5
	Apricot	2/5	0/5
Control, non-inoculated	Halford peach	0/5	0/5
	Apricot	0/5	0/5

^a Inoculated with root chips from naturally infected, pitted peach tree (inoculum from Ellicott City, Md., commercial orchard tree), and grown for 2 years after the inoculation in the greenhouse.

^b Number of plants with symptoms per number of plants inoculated.

^c See text.

^d Buds collected 96-113 cm above the highest pitting area on the stem.

^e Stem chips collected from the pitted stem 10-30 cm above the original graft inoculum.

^f Root chips collected from nonpitted roots 13-17 cm below the nearest pitting at the stem.

missible viruses are known to have localized distribution in the infected fruit trees (5, 9) or herbaceous plants (2). The viral nature of prunus stem pitting has been suggested (8), but our numerous attempts to mechanically transmit and characterize causal agents(s) of stem pitting failed. Progress in characterization and identification of the causal agent or agents seems unlikely, unless a method for detection and for obtaining mechanical transmission can be developed. However, recently (*unpublished data*) we have mechanically transmitted a virus, but only from small young shoots developing from the pitted trunk of apricot seedlings inoculated with root chips from naturally infected-pitted trees. Characterization and elucidation of the role of this virus in Prunus stem pitting is in progress. Prunus stem pitting resembles, and has the attributes of, several virus diseases in other fruit trees, although we can conclude nothing on the nature and identity of the infectious causal agent of this disease. Natural spread of the disease occurs in orchards (8). The pattern of spread is suggestive of underground dissemination and a soil-borne vector. These investigations revealed that the occurrence of initial pitting in the wood of artificially inoculated plants is always related to the inoculation point, and not to the underground portion of the plants as it occurs in naturally infected peach trees. This observation, in addition to the pattern of the

natural spread, suggests that the infection of naturally affected trees in commercial orchards may originate at the underground portion.

PDV viruses and strains of NRSV are very commonly found infecting stone fruit trees in commercial orchards. These viruses are common contaminants of stone fruit virus cultures (3). Our investigations showed a high incidence of NRSV in commercial orchard trees. However, NRSV failed to induce stem pitting in experimentally inoculated peach seedlings. Since there was no correlation between the presence of NRSV and stem pitting or the severity of stem pitting symptoms in naturally or experimentally infected peach trees, we concluded that NRSV is not the primary cause of peach stem pitting.

The stem pitting causal agent(s) was graft-transmitted readily from nectarine and Chinese wild peach to peach seedlings (Table 2), and from artificially inoculated peach seedling to apricot seedling (Table 3), indicating a common causal agent(s) for stem pitting in these *Prunus* spp.

Since stem pitting is caused by graft-transmissible agent(s), the use of propagation materials from healthy trees is imperative. Root chips as inoculum appear to be very reliable in indexing peach trees for stem pitting and selecting trees for propagation that are free of the stem pitting inducing agent(s).

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