Occurrence and Control of Cherry Stem Pitting Disease

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ABSTRACT

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Cherry stem pitting (CSP) is a debilitating disease of sweet cherry trees (*Prunus avium* (L.) L. 'Bing'). Symptoms on the tree trunks include thickened bark and prominent pits in the woody cylinder. Eventually, affected trees decline. Surveys of commercial orchards, some of which were established with a mixture of cv. Bing on Colt (*P. avium* × *P. pseudocerasus*), mahaleb (*P. mahaleb* L.), or mazzard (*P. avium*) rootstocks, revealed that disease incidence ranged up to 44% but only trees on mahaleb and mazzard rootstocks were symptomatic. All Bing/Colt trees appeared healthy, which indicated that Colt rootstock is likely resistant to the CSP agent. These observations were confirmed in test plots replicated with trees of Bing/Colt and Bing/mahaleb. Although circumstantial evidence suggests a soilborne origin and symptoms resemble those of Prunus stem pitting (PSP), all attempts to detect tomato ringspot virus (the causal agent of PSP) in affected trees by graft-inoculations onto *P. tomentosa* Thunb. have failed.

Prunus species are reportedly afflicted with, perhaps, four "stem pitting" disorders. One disease, called Prunus stem pitting (PSP), is induced by tomato ringspot virus (TmRSV) (11), which is vectored by the dagger nematode, *Xiphinema americanum sensu lato*. The second, xylem aberration, exhibits similar symptoms in apricot (*P. armenicaca* L.), sour (*P. cerasus* L.), and sweet (*P. avium*) cherry trees (7,8), but is not associated with TmRSV (3). In Washington, another widely distributed stem-pitting condition of sweet cherry trees was found not to be associated with a graft- or sap-transmissible agent (1,9). In California, Mircetich et al (10) confirmed PSP in apricot, European plum (*P. domestica* L.), peach (*P. persica* (L.) Batsch), and the cherry rootstocks *P. mahaleb*, Stockton Morello (*P. cerasus* L. var. *austera* L.), but not mazzard (*P. avium*). They also transmitted by grafting a different stem-pitting disorder of sweet cherry (designated herein as CSP). Later, Hoy (4) reexamined the nature of CSP and concluded that an agent other than TmRSV or cherry leaf roll virus was probably involved.

In 1986, we initiated orchard surveys for CSP and conducted replant trials to evaluate Colt and mahaleb rootstocks for field resistance to CSP.

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MATERIALS AND METHODS

Disease symptoms. Affected Bing cherry trees are delayed in spring budbreak and later exhibit small leaves, sparse canopies, and dead scaffold branches and interior limbs. Although fruit develop on diseased trees, they are small and unmarketable and have short pedicels. In the early stages of infection, the lower half of affected trees defoliate prematurely. Occasionally, small leaves and fruit may

be confined to a single scaffold branch. On all trees, the trunks develop thick bark and pit symptoms that are readily evident on the xylem surface of the Bing scion (Fig. 1). Similarly, stem-pitting symptoms were observed on mazzard rootstock. In other cases, more subtle symptoms consisting of shallow, longitudinal grooves may develop on the woody cylinder of *P. mahaleb* rootstocks with symptomatic Bing cherry scions. However, irrespective of rootstock species, mature trees decline and die within 2 to 3 yr following onset of canopy symptoms.

Orchard surveys and trials. Confirmatory disease diagnosis of symptomatic trees was made using a hammer and an arch punch of 3.8 cm diameter to remove a circular bark patch, followed by examination of the underlying xylem surface for pits. A total of 12 orchards were surveyed around Lodi, Stockton, and Linden, CA, five of which were inspected a second year.

In 1988 and 1989, three orchard trials were established with healthy trees of Bing/mahaleb and Bing/Colt replanted in sites previously with diseased trees. One trial contained 15 pairs of trees, another 14 pairs, and a third 10 pairs. In each pair, the rootstocks were planted in a randomized design. All trees were inspected annually for canopy symptoms and all symptomatic and suspect trees were examined for pitting of the woody cylinder. In 1994, all remaining trees were examined for pitted trunks.

Nematode analysis and virus transmission tests. Soil samples were collected at various times during two growing seasons from diseased orchards and screened for nematode species and populations (2). Also, nematodes of *X. americanum* were collected from diseased orchard soils and from greenhouse-maintained colonies (virus-free populations). These were introduced in groups of 60 onto roots of potted mahaleb seedlings that were later grafted with Bing cherry buds supplied by Foundation Plant Materials Service (University of California, Davis). A third treatment, as a check for other possible soil-inhabiting vectors, consisted of saturating each mahaleb root system with 500 ml of effluent (after filtration through a 400-mesh screen) obtained during the extractions of soils taken beneath CSP-trees. Another control included trees with no treatments. All four treatments utilized three potted mahaleb seedlings.

Virus assays. Sap transmissions to herbaceous hosts were attempted directly on orchard sites using diseased and healthy cherry trees by grinding fresh cambial (bark) scrapings in nicotine-phosphate buffer (6) and applying the extracts onto carborundum-dusted leaves of *Chenopodium quinoa* Willd., tobacco (*Nicotiana tabacum* L. 'Turkish' and *N. megalosiphone*) and cucumber (*Cucumis sativus* L. 'National Pickling'). The indicator plants were returned to a greenhouse and observed for symptoms. In addition, cambial scrapings were extracted in enzyme-linked immunosorbent assay (ELISA) buffer (12) and tested in microtiter plates with antiserum of TmRSV. Also, root bark patches from diseased and healthy trees were grafted onto three trees each of *P. tomentosa,* a sensitive indicator for TmRSV (13).

Also, similar assays by herbaceous and woody indicator plants and TmRSV-ELISA were performed using peach root tissues of plum scion trees (orchard located in Placerville, CA) previously diagnosed with (prune) brown line disease, which is incited by TmRSV (5).

RESULTS

The incidence of CSP in seven orchards ranged from 2 to 10% while five other orchards developed 14, 20, 28, 34, and 44% diseased trees. Table 1 shows data from selected orchards surveyed from 1986 to 1991. Repeat surveys in three orchards (i.e., orchards C, L, and R-East) showed that the disease incidence remained unchanged over 2 or 3 yr. However, one (orchard G) had an increase from 8 to 23% after 2 yr, and another (orchard E) had a decline to 24% due to diseased tree removal and replanting following the 1986 harvest; all cherry replants were propagated on Colt rootstocks. In these commercial orchards, none of the original Bing/Colt trees developed disease symptoms.

In the rootstock trials, only Bing/mahaleb trees became infected, with disease incidences ranging from 7 to 87% (Table 2). All replants of Bing/Colt trees appeared healthy.

Natural nematode populations per 200

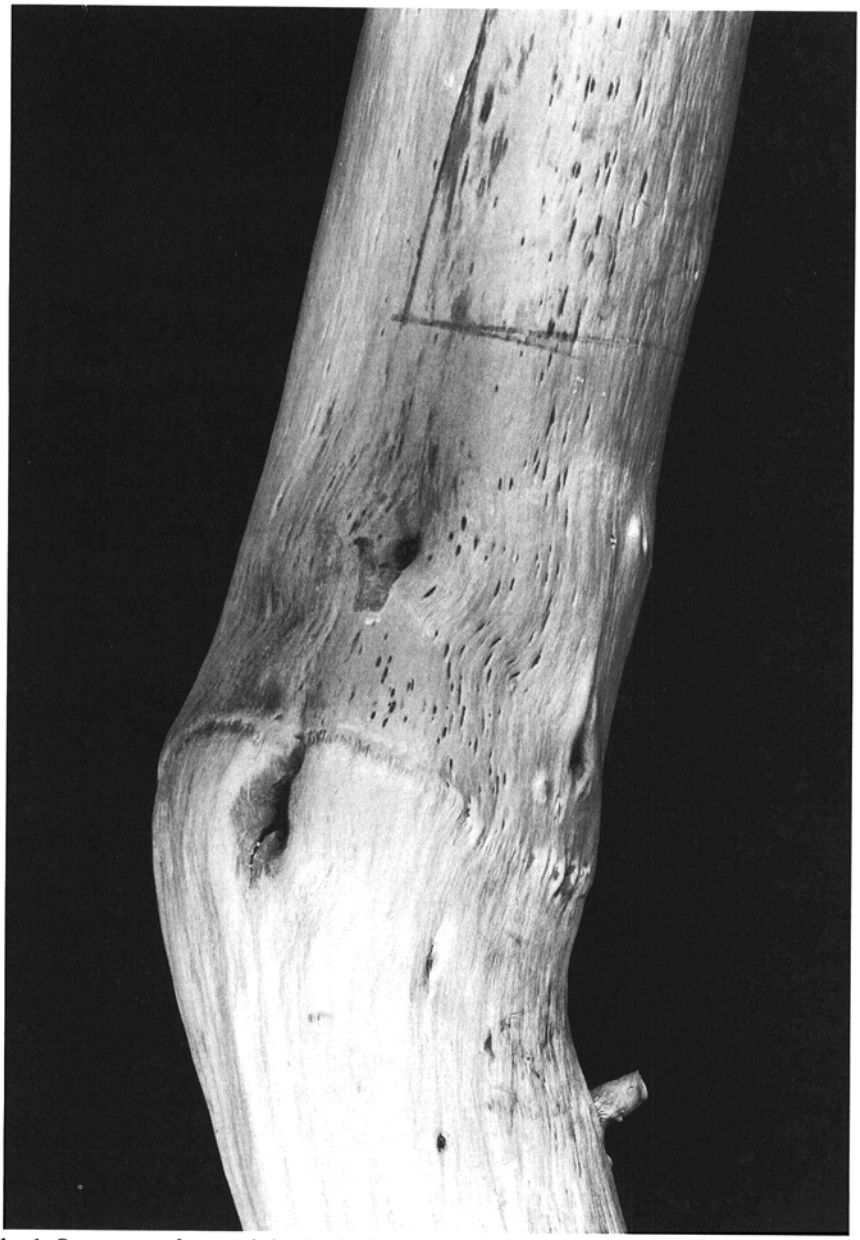

Fig. 1. Symptoms of stem pitting beginning at the union and on the Bing sweet cherry scion growing on mahaleb rootstock infected with cherry stem-pitting disease.

Table 1. Incidence of stem-pitting disease in Bing cherry orchards on three kinds of rootstocks

Orchard, number of trees and rootstock	No. diseased trees (%)				
	1986	1987	1989	1990	1991
C, 604 trees on mahaleb	...[a]	...	...	21 (4)	21 (4)
604 trees on Colt	...	...	...	0	0
E, 1,388 trees on mahaleb	610 (44)	330 (24)[b]	...	...	...
G, 1,400 trees on mahaleb	...	...	113 (8)	...	316 (23)
L, 1,096 trees on mahaleb	...	...	...	115 (10)	113 (10)
R-East, 168 trees on mazzard	...	...	12 (7)	...	10 (6)
168 trees on Colt	...	...	0	...	0
R-West, 162 trees on mazzard	...	...	...	...	13 (8)
162 trees on Colt	...	...	...	...	0
R-Center, 462 trees on mazzard	...	...	...	...	155 (34)
462 trees on Colt	...	...	...	...	0

[a]Not surveyed.
[b]Most cherry stem-pitting tree sites were replanted with sweet cherry on Colt rootstocks during the 1986–1987 dormant season.

Table 2. Incidence of cherry stem pitting in 1994 surveys of cherry replant trials

Year planted	Orchard	No. diseased trees/total (%)	
		Bing/mahaleb	Bing/Colt
1988	E	13/15 (87)	0/15
1989	M	1/10 (10)	0/10
	S	1/14 (7)	0/14

cc soil ranged from 0 to 4,290 for *X. americanum,* 0 to 300 for a *Paratylenchus* sp., 0 to 700 for *Pratylenchus vulnus* Allen and Jensen, and 0 to 1,650 for the spiral nematode *Paratylenchus* sp. *Micoletzky.* No *Trichodorus* sp. *Cobb,* or *Criconemella xenoplex* Raski, were recovered.

After 3 yr of observations, all Bing scions on mahaleb rootstocks that were exposed to *X. americanum* nematodes, or treated with wash effluents, and untreated (controls) developed vigorous healthy shoots without stem-pitting symptoms.

Mechanical assays on herbaceous indicators with extracts prepared from CSP and PSP sources failed to transmit any sap-transmissible virus. Similarly, all TmRSV assays by ELISA were negative. On *P. tomentosa,* CSP-grafted trees resembled that of healthy (ungrafted) ones. In contrast, the PSP source transmitted TmRSV to *P. tomentosa,* producing leaf symptoms consisting of chlorotic spots and vein chlorosis after a 6-wk incubation. After 1 yr, the infected trees exhibited small leaves and stunted shoots. TmRSV was readily sap-transmitted to cucumber, and identified in extracts of both cucumber and *P. tomentosa* plants by ELISA.

DISCUSSION

Our failure to associate TmRSV with CSP supports the previous findings of Hoy (4). Although the etiology of this disease remains a mystery, we have identified resistance in a commercially available cherry rootstock. Orchard surveys clearly indicated that cherry trees supported on Colt rootstocks were free of CSP symptoms and that certain orchards displayed low disease spread (Table 1, orchards C, L, and R-East), while in others it was found to be dynamic (orchards E and G). Similar observations were made in the rootstock trials, in which ongoing spread was evident in orchard E (same orchard as in Table 1) and a much slower rate of spread occurred in orchards M and S (Table 2). The Colt rootstock has continued to exhibit field resistance despite heavy disease pressure in some orchard sites.

Although the feeding trial was limited, exposure of mahaleb roots to *X. americanum* did not result in any virus transmissions. Even so, the soilborne nature of CSP is suggested by the fact that severe stem-pitting symptoms occurred on mazzard rootstocks and near the scion junction on mahaleb rooted trees, with the xylem pits becoming less evident higher up the tree trunk. Also, stem pitting of Bing cherry occurred only if it was on infected mahaleb and mazzard (i.e. susceptible) rootstocks, but not on Colt, which is apparently resistant and the rootstock of choice for diseased sites.

LITERATURE CITED

1. Al Musa, A. M., Mink, G. I., and Parsons, J. L. 1980. Attempts to transmit the causal agent of a cherry stem pitting disorder in Washington sweet cherry trees. Plant Dis. 64:1081-1083.
2. Flegg, F. F. M., and Hooper, D. F. 1970. Pages 5-12 in: Laboratory methods for work with plant and soil nematodes. J. F. Southey, ed. Tech. Bull. 2, Minist. Agric. Fish and Food, London.
3. Hansen, A. J. 1976. Xylem aberration. Pages 257-258 in: Virus Diseases and Noninfectious Disorders of Stone Fruits in North America. USDA, ARS, Agr. Handbk. No. 437.
4. Hoy, J. W. 1983. Studies on the nature of the prune brownline and sweet cherry stem pitting diseases and epidemiology and control of prune brownline. Ph.D. thesis, University of California, Davis.
5. Hoy, J. W., and Mircetich, S. M. 1984. Prune brownline disease: Susceptibility of prune rootstocks and tomato ringspot virus detection. Phytopathology 74:272-276.
6. Kirkpatrick, H. C., and Lindner, R. C. 1964. A mechanically transmissible virus latent in apple. Phytopathology 54:229-232.
7. Lott, T. B. 1967. Xylem abberation, a transmissible disease of stone fruits. Can. Plant Dis. Surv. 47:74-75.
8. Lott, T. B., Keane, F. W. L., and May, J. 1962. Gumming, distortion, and pitting in cherry and apricot. Can. Plant Dis. Surv. 42:229-232.
9. Mink, G. I., and Howell, W. E. 1980. Occurrence and distribution of stem pitting of sweet cherry trees in Washington. Plant Dis. 64:551-552.
10. Mircetich, S. M., Moller, W. J., and Nyland, G. 1977. Prunus stem pitting in sweet cherry and other stone fruit trees in California. Plant Dis. Rep. 61:931-935.
11. Smith, S. H., Stouffer, R. F., and Soulen, D. M. 1973. Induction of stem pitting in peaches by mechanical inoculation with tomato ringspot virus. Phytopathology 63:1404-1406.
12. Uyemoto, J. K. 1989. Union aberration of sweet cherry on *Prunus mahaleb* rootstock associated with X-disease. Plant Dis. 73:899-902.
13. Zhang, Y.-P., and Uyemoto, J. K. 1993. Detection and recovery of tomato ringspot virus from diseased peach by graft inoculations to *Prunus tomentosa.* Plant Dis. 77:101.