Pseudomonas Canker of Pear Trees in Oregon, Cultivar Resistance, and Effect of Trunk Guards on Canker Incidence and Bacteria Survival on Bark

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ABSTRACT

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Extensive cankers caused by *Pseudomonas syringae* pv. *syringae* were observed on young pear trees in early spring 1991 following low-temperature injury in December 1990. The majority of cankers were on the trunk and lower scaffold limbs, usually close to or extending into the trunk. Canker incidence was greater on trees with trunks enclosed in corrugated, white plastic guards than on trees without guards. Survival of *P. s. syringae* inoculated on the surface of the bark of Bartlett trees was significantly greater when tree trunks were covered with guards than when guards were not used. *P. s. syringae* was isolated from all canker margins in spring 1991, but incidence decreased to 30–50% of the cankers after two growing seasons. The cultivars Bosc and Comice were rated as susceptible to Pseudomonas canker. Red strains of d'Anjou and Bartlett were considered resistant in both field evaluations and a detached shoot test.

Pseudomonas syringae pv. *syringae* van Hall infects pear (*Pyrus communis* L.) and causes blossom blight, fruit and leaf spots, and cankers (1,14). The conditions for induction of blossom blast were described recently (21). Also, the distribution, population dynamics, and characteristics of *P. s. syringae* in deciduous tree fruit orchards in the Pacific Northwest have been studied (4). The canker phase of the disease on pome fruits, however, has not received much attention. Wilson (24) described the symptoms of limb cankers on pear trees in California as early as 1934. He reported that Bosc pear trees were more severely infected than Bartlett and that infected limbs and young trees were killed occasionally. McKeen (11) reported a blossom blast and canker disease of pear on Vancouver Island, British Columbia. The cankers often developed from diseased buds and peduncles, enlarged throughout the winter, and eventually killed young trees. Cameron (1) stated that infection of pear trees less than 5 yr old through pruning cuts and wounds caused considerable tree loss in Oregon. Recently, Larsen and Higgins (9) reported that a temperature drop from 16 to −21 C in early February caused longitudinal bark cracking on trunks of young Asian pear trees.

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During December 1990, Pseudomonas canker, in conjunction with low-temperature injury, was associated with serious disease and death of trees in some areas of the Pacific Northwest. In the lower Hood River Valley of Oregon, temperatures during November and the first half of December 1990 were seldom below freezing. On 20 December, the temperature dropped to −15.5 C and remained between −14 and −20 C for six nights. Trees between 2 and 5 yr old were most seriously infected, and those that had not hardened off properly were affected worst. Thus, growers who extended the normal growth cycle of their trees by late summer fertilization, irrigation, or training often experienced an epidemic of Pseudomonas canker in their orchards.

In this paper we report the results of a survey of five severely infected commercial pear orchards and present information concerning location of cankers on the tree, cultivar resistance, and origin of cankers in relation to pruning cuts, dead spurs, and tissue injured by low temperatures. Survival of *P. s. syringae* on the bark of pear trees with and without trunk guards was studied because white plastic wraps, which are used to protect trunks of young pear trees from winter injury, appeared to affect canker incidence.

MATERIALS AND METHODS

Field survey. Five commercial pear orchards near Parkdale, Oregon, were selected for determination of severity of Pseudomonas canker associated with low-temperature injury that occurred in December 1990. Three orchards, designated A, B, and C, were selected on the basis of the presence of multiple cultivars of the same age in the same or adjoining rows. Trees in orchards A, B, and C were planted in 1986, 1985, and 1985, respectively. Trees that had visible cankers of each of three cultivars were evaluated—23 in orchard A, 12 in orchard B, and 21 in orchard C. The length of all cankers on each tree was measured, and the location and origin (pruning cut, spur, or winter-injured tissue) of the cankers were recorded. Cultivar susceptibility data were analyzed with analysis of variance and LSD when the *F* test was significant.

In two orchards, designated D and E, trees with and without trunk guards (Fig. 1) were present in alternating rows throughout the orchards on two cultivars per orchard. Both orchards were established in 1989, and one-half of the trees of each cultivar in each orchard had trunk guards installed at the time of planting. Only the lower trunk of each tree was examined for Pseudomonas cankers, 72–80 trees of each cultivar in orchard D and 110–116 trees of each cultivar in orchard E. Data were analyzed by chi-square. Evaluations in all five orchards were done between 17 and 19 June 1991.

Effect of trunk guards on survival of *P. s. syringae* on the bark surface. A spontaneous, rifampicin-resistant mutant of *P. s. syringae* isolate 90-163 was grown on *Pseudomonas* agar F (PAF) at 23 C. Colonies were harvested from 48-hr-old

Fig. 1. Corrugated plastic guard with white outer and black inner surface around the trunk of a pear tree.

plates with sterile distilled water (SDW), and the suspension was adjusted to 0.3 A at 600 nm in a colorimeter. The suspension contained 2.9×10^9 cfu/ml as determined by standard dilution plate assay. The bacterial suspension was sprayed to runoff with a hand-pump sprayer on the lower 30 cm of the uninjured trunks of 5-yr-old Bartlett pear trees. After the bark was dry (approximately 1 hr), white, corrugated plastic trunk guards (Treewrap, Diversa-Plast, Minneapolis, MN) were placed around one-half of the trees. The trunk guards were 40 cm in height and were placed around the trunks so ends touched but did not overlap, leaving about 1 cm of space between the trunk and the guard. When trunks were dry and at weekly intervals thereafter, two 16-mm-diameter disks of bark were removed from each of three replicate trees with a sterile No. 9 cork borer and returned to the laboratory in sterile glass petri plates. The two disks were placed, outer bark surface down, in 5–10 ml of SDW and sonicated for 1 min. In a preliminary experiment, sonication for an additional 1 and 2 min did not result in greater numbers of bacteria recovered. After sonication, 100–500 μl of the undiluted suspension and of a 1:9 dilution suspension were spread on two replicate plates containing PAF amended with 100 μg of rifampicin and 40 μg of cycloheximide per milliliter. At the lower dilution, the detection limit was 2.5 cfu/100 mm^2 of bark. Plates were incubated 72 hr at 23 C and colonies were counted. Plate counts were converted to colony-forming units per square millimeter of bark, and log-transformed data were analyzed with an unpaired t test. The experiment was done twice, with inoculations on 14 November (trial 1) and 11 December 1991 (trial 2).

Survival of *P. s. syringae* in cankers of pear trees in the orchard. In orchard B, 10 trees each of cultivars d'Anjou, Bartlett, and Comice with a large canker on the main trunk were selected. Similarly, 10 infected Columbia Red d'Anjou trees were selected in an orchard near Hood River. On 20 June and 17 October 1991 and 6 October 1992, a 1 × 4 cm piece of bark tissue at the margin of each canker was removed with a sterile scalpel. In the laboratory, the outer bark was removed, and each sample was cut into five to seven pieces. Pieces were surface-sterilized in 0.42% sodium hypochlorite for 30 sec, rinsed in SDW, and placed on PAF. Plates were incubated 4–5 days at 23 C, then examined for colonies producing fluorescent pigment under UV light at 350 nm. Fluorescent colonies were purified on nutrient agar, then tested for oxidase activity (8). All fluorescent, oxidase-negative isolates recovered on 6 October were tested for pathogenicity with the tobacco hypersensitivity method (6) using *Nicotiana tabacum* L. 'Samsun'.

Detached shoot test for evaluation of resistance of pear cultivars to Pseudomonas canker. Actively growing terminal shoots about 20 cm long were removed from trees of 11 pear cultivars at the Mid-Columbia Experiment Station. Leaves and petioles were removed, and shoots were surface-sterilized in 0.05% sodium hypochlorite for 1 min, rinsed in SDW, dipped in 95% ethanol, and rinsed again in SDW. A 5-cm length was removed from the base of each shoot, then five shoots were placed in each of three sterile, 250-ml beakers containing 50 ml of SDW. Inoculum of *P. s. syringae* isolate 90-163 was prepared as described above. A 3-cm length was removed from the shoot apex, and a 10-μl drop of inoculum was placed on the cut surface. The inoculum was absorbed by the tissue within a few minutes, and the beaker was covered with a polyethylene bag secured with a rubber band. Control shoots were inoculated with 10 μl of SDW. Beakers were placed at 10 C, and canker length was measured after 14 days. The experiment was done twice. Data were analyzed by analysis of variance after an arcsine square root transformation to obtain a random distribution of residuals. The two trials were treated as blocks, and the block × cultivar mean square was used as the error mean square. Separation of treatment means was done with Tukey's HSD test.

RESULTS

Field survey. Extensive Pseudomonas cankers developed on pear trees in early spring of 1991 following low-temperature injury in December 1990. For example, Comice trees in orchard B had an average of 40 cankers per tree, with an average canker length of 10 cm. About one-half of the cankers were on major scaffold limbs, usually close to or extending into the trunk (Fig. 2). About one-third of the cankers were on the trunk below the first whirl of scaffold limbs.

In orchards A and B, 21–31% of the cankers were centered at a dead spur, 8–25% were at a pruning cut, and 54–61% were at locations with no visible mechanical injury or dead spurs and were probably associated with low-temperature injury. In orchard C, 77–95% of the cankers occurred in winter-injured tissue and very few were associated with dead spurs or pruning cuts. The percentage of cankers originating at dead spurs, pruning cuts, or low-temperature injury was similar for all three cultivars in each orchard. Direct cultivar comparisons of canker resistance were limited to those cultivars present in the same orchard. In these comparisons in orchards B and C, Cascade and Sensation Red Bartlett were most resistant based on number of cankers per tree or canker length (Table 1). Bosc and Gebhard Red d'Anjou in orchard A and Comice in orchard B were most susceptible to canker (Table 1). Bosc was more resistant than Comice in orchard D, and Max Red Bartlett was more resistant than Columbia Red d'Anjou in orchard E (Table 2).

The presence of tree guards affected canker incidence. In orchard D, more trees with than without guards had infected trunks; the difference was significant ($P = 0.05$) for Comice but not for Bosc. In orchard E, significantly ($P = 0.05$) more trees with than without guards had trunk cankers (Table 2). This effect was similar for both Max Red Bartlett and Columbia Red d'Anjou.

Fig. 2. Pseudomonas canker on trunk, crotch, and scaffold limb of Columbia Red d'Anjou pear tree.

Table 1. Pseudomonas canker on pear trees in the Hood River Valley in June 1991

Orchard Cultivar	Canker length per tree (cm)	Cankers per tree (no.)
A		
Columbia	39 a[z]	6 a
Bosc	99 b	11 b
Gebhard	89 b	13 b
B		
Sensation	63 a	10 a
Columbia	105 a	19 b
Comice	400 b	40 c
C		
Cascade	2 a	1 a
Sensation	4 a	1 a
Columbia	18 b	3 b

[z] Numbers followed by the same letter within columns for each orchard are not significantly different at $P = 0.05$ according to LSD.

Table 2. Effect of guards on infection of pear tree trunks by *Pseudomonas syringae* pv. *syringae*

Orchard Cultivar	Number of trunks infected[x]	
	Guard	No guard
D		
Bosc	44 a[y]	27 a
Comice*[z]	72 b	25 a
E		
Max Red Bartlett*	50 a	29 a
Columbia*	81 b	60 b

[x] Trunk cankers were counted in June 1991.
[y] Numbers followed by the same letter within columns for each orchard are not significantly different at $P = 0.05$ according to chi-square.
[z] * = Guard effect is significant at $P = 0.05$.

Effect of trunk guards on survival of *P. s. syringae* on the bark surface. Survival of *P. s. syringae* on the bark surface was significantly ($P = 0.01$) greater on trees with trunk guards than on those without guards (Fig. 3). Even with guards, however, populations decreased logarithmically, and few bacteria were recovered beyond 3–4 wk. The average daily maximum and minimum temperatures were 9.2 and 2.2 C, respectively, for trial 1 and 5.5 and 0.1 C, respectively, for trial 2. Initial *P. s. syringae* populations for trials 1 and 2 were 3.05×10^3 and 7.37×10^3 cfu/mm^2 of bark, respectively. After 4 wk, trial 2 was extended an additional 20 wk, and populations remained below 1 cfu/mm^2 of bark except on 17 April, when 11.9 cfu/mm^2 were recovered. No tree in either trial became infected.

Survival of *P. s. syringae* in cankers of pear trees in the orchard. On 20 June 1991, *P. s. syringae* was isolated from tissue at canker margins of all cankers. By 17 October, *P. s. syringae* was recovered from 60, 60, 78, and 67% of cankers of d'Anjou, Bartlett, Comice, and Columbia Red d'Anjou, respectively. On 6 October 1992, recovery decreased to 40, 30, 50, and 33%, respectively, of cankers of d'Anjou, Bartlett, Comice, and Columbia Red d'Anjou. Thus, while survival of *P. s. syringae* in tissue at canker margins declined over time, the bacterium was still recovered from one-third to one-half of the cankers even after two growing seasons. In the tobacco hypersensitivity test, isolates from 62% of the cankers from which *P. s. syringae* was recovered were positive.

Detached shoot test for evaluation of resistance of pear cultivars to Pseudomonas canker. Canker severity was greatest in detached shoots of Forelle and Cascade (Table 3). Although Sensation Red Bartlett and d'Anjou, including the two red strains, had the smallest cankers, disease severity in these cultivars did not differ significantly ($P = 0.01$) from that in all other cultivars except Forelle and Cascade (Table 3). In several tests, the outer bark was removed and the length of discolored wood was measured. In almost all cases, there was no difference between the length of external cankers and that of inner tissue discoloration. Control shoots usually were free of cankers, but those that formed were no longer than 1–2 mm.

DISCUSSION

When pear trees reach their maximum level of hardiness, they can withstand temperatures below −33 C (12,15). Factors that delay maturity and development of cold hardiness include excess nitrogen, late season irrigation, and early autumn pruning (20). These cultural practices are often used by growers to "push" young trees into rapid development and production. The differential development of cold hardiness throughout a tree was illustrated during 1990–1991 by a pattern of low-temperature injury on the lower trunk and crotches. This pattern also occurred on pome fruit trees in the Pacific Northwest in 1955 (20). The importance of freezing temperatures to development of canker caused by *P. s. syringae* has been well documented for apricot (7), poplar (2), peach (19), and sweet cherry (17).

The low-temperature injury and Pseudomonas canker that we observed differed from the typical pattern of injury to the southwest area of the trunk that occurs on cold, sunny winter days when a large temperature differential develops between the sunny and shaded sides of the trunk. Painting trunks white has been recommended to reduce this type of damage (5,13). Similarly, trunk-insulating wraps reduce temperature fluctuations by decreasing maximum day temperatures but not affecting minimum night temperatures (16). We found, however, that incidence of Pseudomonas canker was greater in orchards where trunk guards were used. These guards were wrapped loosely on the trunk with about 1 cm of space between the trunk and the guard. In early spring, we observed that the trunk and weeds inside the guards remained wet considerably longer than when guards were not used. The guards may alter the microclimate at the bark surface, resulting in increased survival of *P. s. syringae*. Trunk guards installed with a space between the ends or with large vent holes may be less likely to increase the canker problem described herein.

Even when trunk guards were used, populations of *P. s. syringae* on bark declined to very low levels within 1 mo after inoculation and failed to increase during the last 5 mo of the experiment. In a previous study on flower and leaf buds of d'Anjou pear, populations of *P. s. syringae* decreased from leaf fall to bloom (4). Similarly, populations of *P. s. syringae* declined to very low levels within 6 wk after inoculation in October of the surface of peach buds and leaf scars (3). In contrast, *P. s. syringae* sprayed on the branches of maple in July declined by November but increased throughout the winter until March (10). While surface populations of *P. s.*

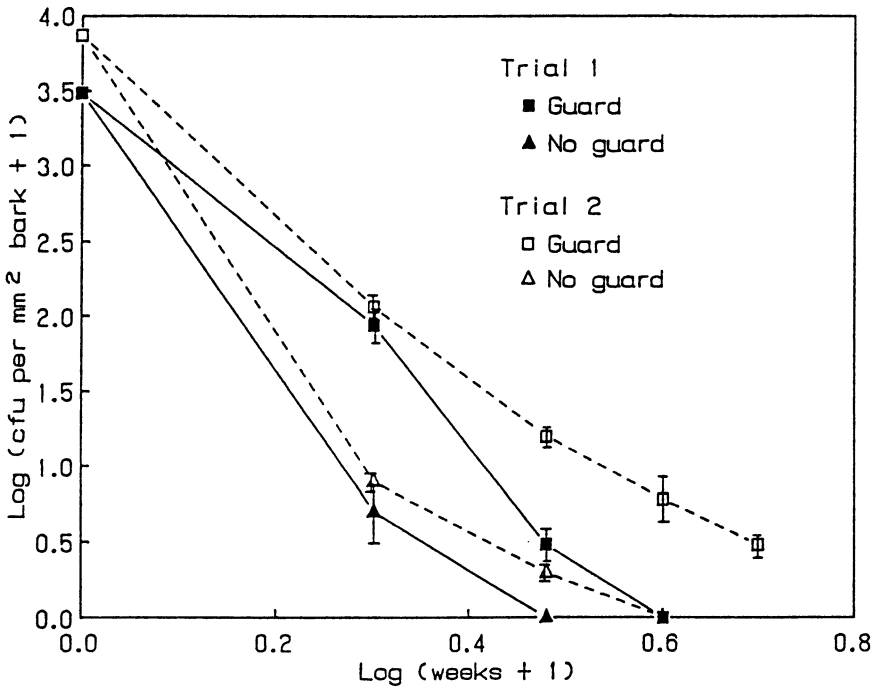

Fig. 3. Effect of trunk guards on the survival of *Pseudomonas syringae* pv. *syringae* on the surface of the bark of Bartlett pear trees. Vertical bars represent standard error of the mean.

Table 3. Resistance of detached shoots of 11 pear cultivars to canker caused by *Pseudomonas syringae* pv. *syringae*

Cultivar	Mean canker length[z] (mm)
Sensation Red Bartlett	2.4 a
Columbia Red d'Anjou	3.8 a
Gebhard Red d'Anjou	3.9 a
d'Anjou	4.3 a
Bartlett	6.1 ab
Hosui	6.2 ab
Max Red Bartlett	6.8 ab
Bosc	10.7 ab
Comice	13.6 ab
Cascade	29.9 bc
Forelle	47.7 c

[z]Shoots were evaluated 14 days after inoculation, and each value represents the mean of 30 shoots. Means are separated with Tukey's HSD at $P = 0.01$.

syringae often fluctuate greatly, internal populations are more stable, whether in peach twigs (3), sweet cherry buds (18), or pear stem and root tissue (23). Similarly, we found that *P. s. syringae* survived in bark tissue at the canker margins, even after the 1991 and 1992 growing seasons. The summer of 1992 was unusually hot, with the maximum daily temperature exceeding 32 C on 25 days between May and August.

Field observations and the detached shoot method for ranking resistance of pear cultivars to Pseudomonas canker generally were in agreement, with Comice and Bosc as susceptible and d'Anjou and red strains of Bartlett as resistant according to both methods. Wilson (24) also reported that Bartlett was more resistant than Bosc to Pseudomonas canker. The detached shoot test, however, only indicates the potential genetic resistance that can be altered by cultural and environmental factors in the orchard. For example, Cascade (a cross of Max Red Bartlett and Comice) was highly susceptible to canker in the detached shoot test but showed a low level of infection in the orchard. Tree vigor in the orchard appeared low, and trees probably developed early cold acclimation. Thus, low-temperature injury and canker were greatly reduced, even in a cultivar that is potentially susceptible to canker. Conversely, detached shoots of Gebhard Red d'Anjou were resistant to canker, but Gebhard trees in orchard A were severely infected, perhaps due to a greater response to irrigation and fertilization programs than other cultivars in the orchard. This resulted in increased late season vigor that predisposed trees to winter injury and subsequent bacterial infection.

A similar detached shoot method was used to rank resistance of pear to blossom blast caused by *P. s. syringae* (22). Forelle was considered more resistant and d'Anjou more susceptible to blossom blast than to canker. Rankings for red d'Anjou strains and for Bosc and Comice were similar for both canker and blossom blast.

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LITERATURE CITED

1. Cameron, H. R. 1962. Diseases of deciduous fruit trees incited by *Pseudomonas syringae* van Hall. Oreg. Agric. Exp. Stn. Tech. Bull. 66.
2. deKam, M. 1982. Damage to poplar caused by *Pseudomonas syringae* in combination with frost and fluctuating temperatures. Eur. J. For. Pathol. 12:203-209.
3. Endert, E., and Ritchie, D. F. 1984. Overwintering and survival of *Pseudomonas syringae* pv. *syringae* and symptom development in peach trees. Plant Dis. 68:468-470.
4. Gross, D. C., Cody, Y. S., Proebsting, E. L., Jr., Radamaker, G. K., and Spotts, R. A. 1983. Distribution, population dynamics, and characteristics of ice nucleation-active bacteria in deciduous fruit tree orchards. Appl. Environ. Microbiol. 46:1370-1379.
5. Kesner, C. D., and Hansen, C. M. 1976. Prevention of winter sunscald injury in Michigan orchards. J. Am. Soc. Hortic. Sci. 101:546-550.
6. Klement, Z., Farkas, G. L., and Lovrekovich, L. 1964. Hypersensitive reaction induced by phytopathogenic bacteria in the tobacco leaf. Phytopathology 54:474-477.
7. Klement, Z., Rozsnyay, D. S., Balo, E., Panczel, M., and Prileszky, G. 1984. The effect of cold on development of bacterial canker in apricot trees infected with *Pseudomonas syringae* pv. *syringae*. Physiol. Plant Pathol. 24:237-246.
8. Kovacs, N. 1956. Identification of *Pseudomonas pyocyanea* by the oxidase reaction. Nature 178:703.
9. Larsen, F. E., and Higgins, S. S. 1992. Longitudinal bark cracking on trunks of young Asian pear trees in response to a rapid drop in winter temperature. Fruit Var. J. 46:225-229.
10. Malvick, D. L., and Moore, L. W. 1988. Survival and dispersal of a marked strain of *Pseudomonas syringae* in a maple nursery. Plant Pathol. 37:573-580.
11. McKeen, W. E. 1955. Pear blast on Vancouver Island. Phytopathology 45:629-632.
12. Montano, J. M., Rebhuhn, M., Hummer, K., and Lagerstedt, H. B. 1987. Differential thermal analysis for large-scale evaluation of pear cold hardiness. HortScience 22:1335-1336.
13. Norris, E. R., Stout, B. A., and Mecklenburg, R. A. 1971. Thermal studies of fruit trees in relation to low temperature trunk splitting. Annu. Meet. Am. Soc. Agric. Eng. Pap. 71-344.
14. Panagopoulos, C. G., and Crosse, J. E. 1964. Blossom blight and related symptoms caused by *Pseudomonas syringae* van Hall on pear trees. Pages 119-122 in: Annual Report of East Malling Research Station, 1963.
15. Quamme, H. A. 1976. Relationship of the low temperature exotherm to apple and pear production in North America. Can. J. Plant Sci. 56:493-500.
16. Sharpe, R. R., Reilly, C. C., and Zehr, E. I. 1991. Evaluation of trunk-insulating wraps on cambium temperature fluctuations and peach tree short life development. Plant Dis. 75:803-806.
17. Sobiczewski, P., and Jones, A. L. 1992. Effect of exposure to freezing temperatures on necrosis in sweet cherry shoots inoculated with *Pseudomonas syringae pv. syringae* or *P. s. morsprunorum*. Plant Dis. 76:447-451.
18. Sundin, G. W., Jones, A. L., and Olson, B. D. 1988. Overwintering and population dynamics of *Pseudomonas syringae* pv. *syringae* and *P. s.* pv. *morsprunorum* on sweet and sour cherry trees. Can. J. Plant Pathol. 10:281-288.
19. Weaver, D. J. 1978. Interaction of *Pseudomonas syringae* and freezing in bacterial canker on excised peach twigs. Phytopathology 68:1460-1463.
20. Westwood, M. N. 1978. Temperate-Zone Pomology. W. H. Freeman, San Francisco, CA.
21. Whitesides, S. K., and Spotts, R. A. 1991. Induction of pear blossom blast caused by *Pseudomonas syringae* pv. *syringae*. Plant Pathol. 40:118-127.
22. Whitesides, S. K., and Spotts, R. A. 1991. Susceptibility of pear cultivars to blossom blast caused by *Pseudomonas syringae*. HortScience 26:880-882.
23. Whitesides, S. K., and Spotts, R. A. 1991. Frequency, distribution, and characteristics of endophytic *Pseudomonas syringae* in pear trees. Phytopathology 81:453-457.
24. Wilson, E. E. 1934. A bacterial canker of pear trees new to California. Phytopathology 24:534-537.