

Slowing the Spread of Fire Blight During Summer

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Fire blight is caused by the bacterium *Erwinia amylovora* and can affect apples, pears, and quince in most fruit growing regions of the United States. Most losses from fire

blight occur when epidemics are initiated during bloom. The blossom blight phase of fire blight has been studied extensively, and a variety of models have been proposed for predicting outbreaks of blossom blight and/or for timing streptomycin

“This work was supported in part by the New York State Apple Research and Development Program. Insects did not contribute to spread of fire blight in two of three field trials. Sprays of copper, sulfur, ProPhyt, and Serenade were ineffective, but regular removal of infected shoots minimized damage to inoculated trees.”

sprays (Anonymous, 2005; Smith, 1993; Steiner and Lightner, 1992). MaryBlyt and Cougarblight are the two most commonly used models. Streptomycin applied at the right times is very effective for controlling blossom blight except where streptomycin-resistant strains of *E. amylovora* are present, and streptomycin resistance has not been a problem in most of New York State.

Spread of fire blight after bloom during the shoot blight phase is not as well understood as the blossom blight phase. Shoot blight often results in significant loss of tree canopy and extensive loss of trees due to subsequent infection of susceptible rootstocks. McManus and Jones (1994) showed that initiation of shoot infections was correlated with windy rain events. However, in recent years, the spread of fire blight during summer has become an increasing problem in some regions due to factors that remain undefined.

Sucking insects have long been suspected of vectoring *E. amylovora* (i.e., carrying bacteria from tree to tree) or of facilitating infection via feeding injuries that provide entry points for *E. amylovora* already present on young leaf surfaces. Two species of aphid, the green apple aphid and the spirea aphid, and two species of leafhopper, white apple leafhopper and potato leafhopper (PLH), are common on terminal growth of apple in early summer. Several studies have shown that aphids are either inefficient facilitators for fire blight or that they play no role at all (Plurad et al., 1967, Clarke et al., 1992). Pfeiffer et al. (1999), using caged insects, showed white apple leafhoppers also failed to affect blight incidence whereas trees misted with *E. amylovora* and then exposed to PLH sometimes developed more fire blight than similar trees that were protected from insects. PLH was also implicated in some of the earliest studies of potential insect involvement with fire blight (Burrill, 1915; Stewart and Leonard, 1916). PLH does not overwinter in the Northeast, but instead



Figure 1: Six-tree plots of Lady Apple, each with its own yellow sticky trap for monitoring potato leafhopper populations, as they appeared at the start of Trial 1 on 4 June 08 (below) and on 1 July 09 (above) after trees became large enough to hide the sticky traps.

migrates into the region on storm fronts during early summer. Timing of the invasion is highly variable from year to year.

To further study the role of PLH and other factors affecting shoot blight, we established a small meadow orchard at the Hudson Valley Lab where, for the past two seasons, we have applied various treatments to determine if pesticides can be used to slow the spread of blight during summer.

Methods

A meadow orchard containing 240 Lady Apple trees on MM.111 rootstocks was established at the Hudson Valley Lab in 2007. Lady Apples were chosen as the test cultivar because of their vigor-

ous upright growth habit and their known susceptibility to shoot blight. MM.111 rootstocks were used to avoid rootstock blight and to generate a vigorously growing vegetative tree that would maintain active shoot growth into late summer.

The meadow orchard was designed with 40 six-tree plots arranged in a triple-row with 45 inches between rows, 3-ft between trees within rows, and a 6-ft in-row spacing between plots (Figure 1). Trees were closely spaced to favor PLH and to promote slow drying and higher relative humidity typical of a mature tree canopy. The plots were located along the western edge of existing orchards so that insecticides applied to other orchard blocks would have minimal impacts on immigrant PLH.

After trees were planted in 2007, shoots were thinned to five or six per tree. These shoots were headed back during dormant pruning in spring of 2008 to encourage multiple shoots to develop from each of the branches. The test trees were sprayed with conventional pesticides throughout the season to control fungal diseases, and a pyrethroid spray was applied at petal fall to suppress aphids and leaf rollers. Trees were fertilized using high rates of nitrogen in April and again in May and June to ensure that trees would continue growing into late summer.

In late May, yellow sticky traps were mounted on posts in the middle of each six-tree plot. Traps were changed at 7 to 10-day intervals and PLH on each trap were counted to determine if treatments had any impact on PLH numbers within the plots. Treatments evaluated in each trial are shown in Table 1. Treatments

were replicated five times using the six-tree plots previously described. All treatments were sprayed to drip with a handgun using a tractor-mounted PTO-driven sprayer. A non-inoculated control was included so that effectiveness of inoculations could be determined and so that spread from inoculated to non-inoculated plots could be studied. After the beginning of each trial, only those insecticides and bactericides noted in Table 1 were applied to trees.

Sulfur was included as a potential control because some observers suspected that regular applications of sulfur for mildew control had successfully suppressed spread of fire blight during summer. A literature search revealed that sulfur sprays had been recommended on some crops (e.g., potatoes, peanuts) in the 1930's as a method for suppressing PLH (DeLong, 1934; Menusan, 1938; Miller, 1942). Other more recent reports suggested that sulfur might change the leaf surface in ways that would make it more resistant to invasion by *E. amylovora*.

Trial 1: Treatments were applied as noted in Table 1. On 4 June 2008, all trees except those in the non-inoculated control

Table 1. Products and rates per 100 gallons for treatments that were evaluated in the shoot blight trials at the Hudson Valley Lab.

2008 (Trials 1 & 2) ^z	2009 (Trial 3) ^y
1. Non-inoculated control	1. Non-inoculated control
2. Provado 0.5 fl oz + ProPhyt 20 fl oz/100 gal	2. Microthiol Disperss 2 lb/100 gal (sulfur)
No-insecticide treatments	No-insecticide treatments
3. Inoculated control	3. Inoculated control
4. Microthiol Disperss (Sulfur) 1.5 lb/100 gal	4. Apogee 2 oz/100 gal
5. Cuprofix Disperss (Copper) 4 oz/100 gal	5. ProPhyt 21 fl oz + Serenade 1.3 lb/100 gal
Treatments with insecticide^x	Treatments with insecticide
6. Provado 0.5 fl oz/100 gal	6. Provado 0.5 fl oz + Asana XL 5 fl oz/100gal
7. Provado + Cuprofix Disperss	7. Provado/Asana +Apogee 2 oz/100gal
8. Provado +Microthiol Disperss	8. Provado/Asana/ProPhyt/Serenade

^z In Trial 1, treatments were applied 30 May, 7, 14, and 21 June and plots were inoculated on 4 June. In Trial 2, treatments were applied on 17 and 25 July and 2 and 12 August and plots were inoculated on 31 July. Where application rates are not shown, products were applied at the same rates shown for those products in preceding treatments.

^y Treatments for Trial 3 were applied 27 May, 6, 16, and 25 June, 8 and 23 July, and 8 Aug except that Apogee was applied only on 16 May, 6 and 25 June. Plots were inoculated on 19 June and again on 2 July.

^x In Trial 2, all three of the insecticide treatments include both Provado at 0.5 fl oz and Asana at 5 fl oz.

Table 2. Effects of treatments on the total numbers of blighted shoots removed from each 6-tree plot during the interval indicated for each of the three trials.

Treatments ^z	Trial 1: 13 June - 15 July 08	Trial 2: 18 July - 20 Aug 08	Trial 3: 12 June - 13 July 09
Insecticide effects			
No insecticide	15.9	7.5	1.9 b
With insecticide	12.7	6.6	0.7 a
<i>P-value for insecticide^y</i>	0.238	0.701	0.050
Blight-treatment effects			
Control	12.6	7.3	1.1
Fire blight treatment 1 ^x	12.4	6.3	1.6
Fire blight treatment 2 ^x	18.0	7.5	1.3
<i>P-value for blight treatment</i>	0.171	0.899	0.778
<i>P-value for insecticide X blight-treatment interaction</i>	0.334	0.424	0.479

^z For treatment details, see Table 1.

^y *P*-values were derived from a 2x3, two-way analysis of insecticide and blight-treatment effects. Means are significantly different only where *P* ≤ 0.05.

^x Fire blight treatment 1 was Cuprofix Disperss in Trials 1 & 2 and Apogee in Trial 3.

Fire blight treatment 2 was Microthiol Disperss in Trials 1 & 2 and Serenade + ProPhyt in Trial 3.

plots were misted with a suspension of *E. amylovora* using a Solo hand-pressurized backpack sprayer to apply approximately 2.1 gal of bacterial suspension that contained 10⁸ colony-forming units (cfu)/ml. This inoculation was made in the middle of a long misty wetting period and was followed by several days when high temperatures exceeded 90° F. The first symptoms of fire blight were evident on shoots by 9 June, and multiple shoots on every inoculated tree were showing symptoms by 10 June.

Beginning on 9 June and continuing through the rest of the season, blighted shoots were removed at one to three-day intervals and the number of shoots removed from each tree was recorded. The first evidence of infection usually involved the appearance of orange discoloration on the youngest leaves, sometimes accompanied by small droplets of bacterial ooze. Most shoots were removed before any wilting or dieback was evident. Cuts were usually made 4 to 6 inches below the lowest discolored tissue, although cuts sometimes were made within two inches of visibly damaged tissue if more severe pruning would have decapitated entire trees. Pruning shears were disinfested

with ethanol between each cut and blight shoots were removed from the test block so as to minimize any chance that our activities would contribute to spread of fire blight.

On 25 June, three weeks after the first inoculation, the entire block was sprayed with a pyrethroid to eliminate resident aphid and leafhopper populations and to “re-zero” the block for a second trial. However, disease incidence data was collected continuously through 18 July so as to detect any delayed effects of earlier treatments.

Trial 2: The same treatments used in Trial 1 were applied again on 17 and 25 July and 2 and 12 August except that the insecticide program was changed (Table 1). Provado treatments used in Trial 1 did not provide the rapid knock-down of immigrant PLH that we had expected. Therefore, in Trial 2 all plots that previously received Provado were sprayed with a mixture of Provado and Asana.

Inoculations for Trial 2 were delayed until 31 July because we had to wait for a convergence of wet weather, a resurgent PLH population, and two inches or more of re-growth from stubs where blighted shoots had been removed in Trial 1. Inoculations on 31 July were completed using the same methods as in Trial 1 except that we reduced the inoculum concentration to one-half of that used in Trial 1 so as to reduce the impact of the initial inoculations. Shoots in the test orchard continued growing until mid-September due to heavy fertilization and the growth-stimulating effects from removal of blighted shoots in Trial 1. However, PLH populations dropped by mid-August and PLH trapping was discontinued on 15 August.

Trial 3: Trees were pruned in the spring of 2009 with the objective of eliminating all growth that extended more than 3 feet above ground, thereby keeping trees small enough to allow observation of all terminal shoots as fire blight developed during summer. Yellow sticky traps were again deployed as for the previous trials. Treatments were applied using the same methods and design as in 2008, but plots were re-randomized and the treatment list was altered (Table 1).

All plots except the non-inoculated controls were misted with *E. amylovora* on 19 June and again on 2 July using the same methods and inoculum concentration (10^4 cfu/ml) that was used for Trial 2. When very little shoot blight appeared following the first inoculation on 19 June, we followed up with a second inoculation on 2 July using the same low concentration of inoculum.

Data analyses For trials 1 and 2, data on shoot blight incidence and PLH captures collected across multiple observation dates were combined in repeated measures analyses that included all of the eight treatments. Two-way analyses of just the six treatments involved in the plus/minus insecticide comparisons provided a more powerful assessment of how treatments affected the totals for blighted shoots and PLH captures recorded for each trial.

Results

In Trial 1, the proportion of actively growing shoots that developed blight by 16 June varied from 47 to 75% among the inocu-

Table 3. Effects of treatments on the total number of potato leafhoppers captured per plot for the intervals indicated.

Treatments ^z	Trial 1: 4 June - 3 July 08	Trial 2: 18 July - 15 Aug 08	Trial 3: 28 May - 9 Jul 09
Insecticide effects			
Control	79.5 a	26.1 b	7.7
Insecticide	126.1 b	13.9 a	5.8
<i>P</i> -value for insecticide ^y	<0.001	<0.001	0.060
Blight-treatment effects			
Control	99.0	21.8	6.6 ab
Fire blight treatment 1 ^x	110.3	22.1	5.2 a
Fire blight treatment 2 ^x	99.1	16.0	8.4 b
<i>P</i> -value for blight treatment	0.144	0.186	0.037
<i>P</i> -value for insecticide X blight-treatment interaction	0.343	0.404	0.684

^{z,y,x} See footnotes for Table 2.

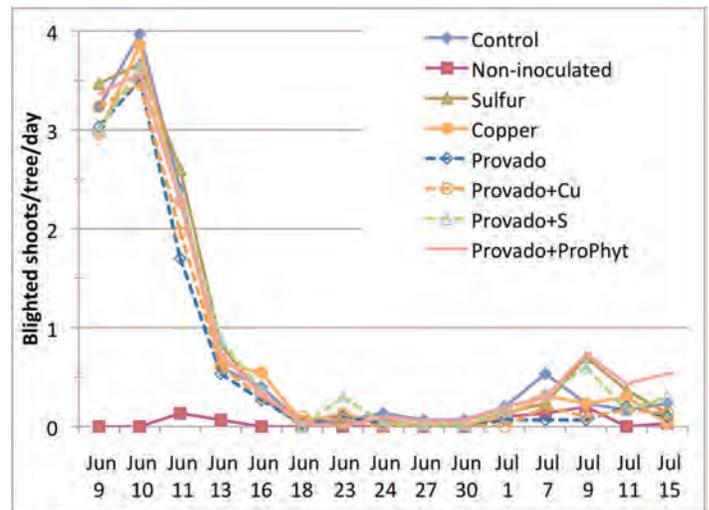


Figure 2. Numbers of blighted shoots per tree that were removed following the 4 June inoculation in Trial 1. The non-inoculated control remained relatively disease-free whereas all of the other treatments followed the same pattern of disease expression over time.

lated plots. The combination of ideal weather conditions, rapid shoot growth, high inoculum levels, and hot weather favored infection and rapid development of fire blight (Figure 2). None of the treatments provided any control. After 13 June, the appearance of new infections slowed, but infections continued to appear through 31 July when trees were re-inoculated for Trial 2. We excluded the first three observation dates (June 9, 10, and 11) from data analyses because our inoculations had apparently overwhelmed our test plants and our objective was to study the effects of treatments on the natural spread of fire blight following disease establishment within the plots.

When all eight treatments in Trial 1 were analyzed together, the only difference among treatments ($P \leq 0.05$) was that the non-inoculated control had less blight than all of the other treatments. The chemical treatments had no effect on blight in inoculated plots. The two-way analysis of the three treatments that were applied with/without insecticides also showed that none of the treatments affected the incidence of blight. Neither copper nor sulfur affected PLH trap captures, but more PLH were captured in the Provado-treated plots than in plots that did not receive Provado ($P < 0.001$, Table 3). The total numbers of PLH captured

on traps over the four-week period from June 4 to July 3 were 2,607 for the 15 plots that received Provado (treatments 6-7-8, Table 1) compared to only 1,627 for the comparable plots without Provado (treatments 3-4-5). Thus, Provado treatment resulted in a 60% increase in numbers of PLH captured even though typical “hopper burn” injury from PLH feeding was evident only on trees that did not receive insecticide. We suspect that contact with Provado-treated foliage irritated PLH, thereby causing increased movement among the trees that resulted in higher trap catches.

In Trial 2, both shoot blight incidence and PLH populations were lower than in Trial 1. In Trial 1 we removed a total of 2,526 blighted shoots from the test block between June 9 and July 15 (565 between June 13 and July 15) whereas in Trial 2 we removed a total of only 181 blighted shoots between July 24 and August 25. None of the treatments had any effect on shoot blight incidence in Trial 2 (Table 2). However, the combination of Provado plus Asana suppressed PLH trap captures by 52% compared to the control plots (Table 3). A total of 286 PLH were noted in traps in the 15 insecticide-treated plots as compared to 595 in comparable plots receiving no insecticide.

In Trial 3, heavy rains during June and July suppressed PLH populations. In 2008, we trapped a total of 5,115 PLH in nine weeks of trapping despite spraying out the entire block with insecticide between Trials 1 and 2. By comparison, traps in the same locations in 2009 captured only 836 PLH between 28 May and 3 Sep. PLH populations peaked in July in 2009 whereas they peaked in June in 2008.

The incidence of shoot blight that developed following inoculations was also much lower in 2009 than in 2008, but there was some natural spread of fire blight in our plots prior to the inoculation (Figure 3). In 2009, we found and removed only 46 strikes during the entire summer. Spread of blight is shown only for the period through 13 July because no additional infections were noted during subsequent weeks. The low infection rates following inoculations in 2009 may have been attributable to the reduced inoculum concentrations that we applied and to the cooler and wetter weather that prevailed through most of the 2009 season.

In Trial 3, individual treatments again showed no differences in their effect on shoot blight development. However, two-way analyses of insecticide-treated versus comparable non-insecticide plots showed that the insecticide-treated plots had 63 percent less shoot blight than plots without insecticides

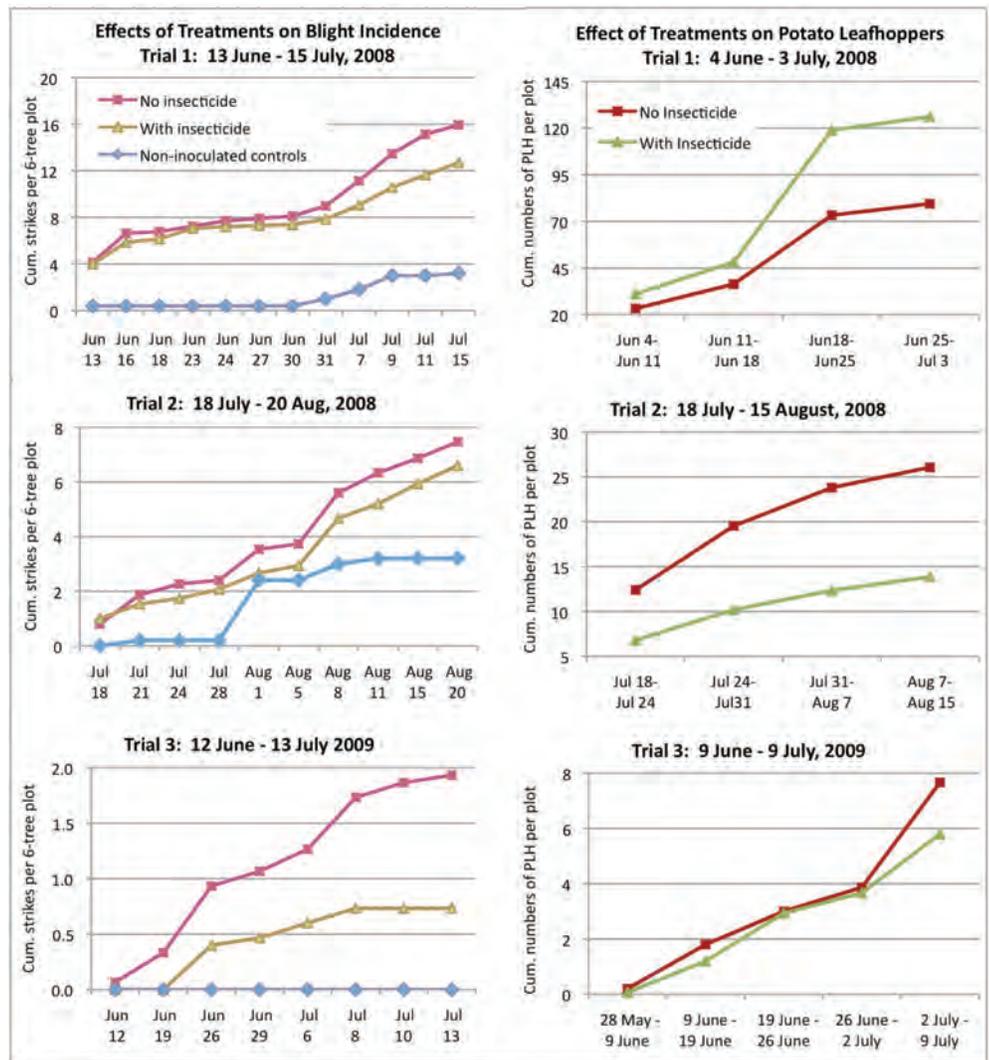


Figure 3. Effects of insecticide treatment on the cumulative incidence of fire blight (left column) and on the number of potato leafhoppers that were trapped over the periods indicated for each of the tree trials. Insecticide treatment had a significant effect ($P \leq 0.05$) on blight incidence only in Trial 3 and on potato leafhoppers only in Trials 1 and 2, although the effects of the insecticide were reversed in those two trials. Data for plots with insecticide and plots with no insecticide were derived from observations of 15 plots (five replicates in each of 3 sub-treatments as shown in Table 1). The lower line in the graphs of blight incidence show blight development in the non-inoculated controls.

($P < 0.05$, Table 2) even though insecticide treatment had no effect on numbers of PLH captured during the test interval ($P = 0.06$, Table 3).

Discussion

Insecticide treatment reduced shoot blight in all three trials (Figure 3), but differences were significant only in Trial 3. We were surprised that insecticide treatment affected blight incidence in 2009 when both PLH populations and blight incidence were very low whereas insecticide treatments had no significant effect on blight incidence in 2008 when both PLH and blight infections were abundant. Even though the Provado treatment used in Trial 1 stimulated higher rather than lower numbers of PLH on traps, we know that Provado was effective for controlling PLH in that trial because treated plants were free of other sucking insects and did not develop the hopper burn that was evident in the no-insecticide plots. Thus, if PLH played a significant role in blight development in 2008, the insecticide treatments in Trial 1 should have reduced blight incidence even though our

method for assessing PLH populations did not show reduced PLH numbers.

A logical conclusion from our three trials is that the role of insects in spreading fire blight in the field is relatively insignificant when compared to dissemination that occurs via wind and rain as documented by McManus and Jones (1994). Perhaps the minor role played by insect facilitators is undetectable when conditions favor rapid spread of blight as occurred in 2008 whereas the effects of insects might be easier to detect in years like 2009 when weather conditions were less favorable for dissemination. Also, insects might play a greater role where infected shoots are not removed as regularly as they were in these trials. The small size of our plots and the limited space between plots was less than ideal for maintaining differential populations of a mobile insect such as PLH, but managing larger plots would have been prohibitively expensive and might have resulted in greater variability in numbers of immigrant PLH within the test block.

We focused on PLH populations because that insect has been implicated in previous studies. We had hoped that we could eliminate aphids and white apple leafhoppers in our test plots by carefully timing pesticide applications prior to the start of this trial, but that proved to be impossible. In all of our trials, aphids and white apple leafhoppers were also present on trees that received no insecticide sprays. Thus, even when insecticide treatments in 2009 reduced the spread of blight, we cannot be certain that these differences were attributable to differences in PLH populations alone because treatments affected populations of other insect species at the same time.

In all three trials, we applied treatments at closer spray intervals than would be economically feasible in commercial orchards because we hoped to maximize chances of detecting treatment effects. Despite using very short spray intervals, none of the treatments aimed at suppressing *E. amylovora* had any effect on the spread of fire blight. Thus, we were unable to show any benefit from applying sulfur, copper, ProPhyt, or Serenade. The very low incidence of blight that developed in non-inoculated trees (Figure 3) showed that there was only limited movement of *E. amylovora* from one plot to another. The sudden rise in disease incidence in the non-inoculated plots in Trial 2 probably occurred because of the 6-8 mile-per-hr wind at the time that inoculum was applied on 31 July. After accounting for that incident, we noted very little spread of blight between inoculated and non-inoculated plots and we therefore should have been able to detect treatment effects if any of the treatments had been capable of reducing blight infection via toxicity to the pathogen or direct protection of the susceptible tissue.

The fact that Apogee failed to suppress blight incidence was surprising because Apogee has proven effective for controlling shoot blight in other trials (Fernando and Jones, 1999; Yoder, 2001). Apogee-treated trees in our plots showed the reduced shoot growth expected from Apogee treatment, so we know that treatments were affecting tree physiology. Perhaps Apogee effects would have been more apparent if we had used higher rates or run the test with less vigorous trees or with a less susceptible cultivar.

Even though our trials failed to provide a definitive conclusion about the role of PLH in fire blight epidemics, the trials reported here show that a small meadow orchard (about one-fourth of an acre) can be used for efficient evaluations of replicated shoot blight treatments. The small tree size allowed us to make

detailed observations on spread of blight to new shoots and to find infections before they caused extensive loss of wood within trees. By removing infected shoots promptly as they appeared, we were able to run repeated trials using the same trees. Over the two years, only one of the 240 trees in our test block was killed by fire blight. Furthermore, we demonstrated that test trees can be successfully and uniformly challenged with *E. amylovora* without creating artificial wounds on the test trees. This orchard design and test protocol should prove useful for further disease control and epidemiological studies with the shoot blight phase of fire blight.

Acknowledgements

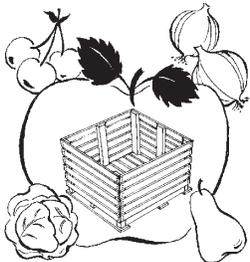
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