Evaluation of an Abatement Program for Western X-Disease in Sweet Cherry

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ABSTRACT


The components of a western X-disease (WXD) abatement program for sweet cherry in California were evaluated in nine cherry orchards from 1986 through 1990. Components of the program included removal of XD-infected trees to reduce XD inoculum in the orchards and post-harvest application of long-residual insecticides to reduce leaffopper populations. A stepwise linear regression of the percentage of new XD infections against the percentage of XD-infected trees remaining in the orchards from one year to the next and insecticide residual longevity showed that XD-infected tree removal was primarily responsible for reduction in new XD infections and that insecticide residual longevity contributed only a minor amount in suppression of new infections.

Additional keywords: Colladonius montanus, Fieberiella florii

Western X-disease (W XD) is a serious threat to sweet cherry (Prunus avium L.) production in California. It has eliminated most cherry production in Napa, Sonoma, and Solano counties (10,12) and threatens production in San Joaquin County, where the majority of sweet cherries are currently produced in California. The causal agent of XD is a mycoplasma-like organism (XMLO) (5) that is transmitted by several leaffopper species (1,6).

In California, two leaffoppers, Colladonius montanus (Van Duze) and Fieberiella florii (Sthl), are the most important vectors of XMLO (8,9,11,17). Colladonius montanus is the most abundant and dispersive vector species found in cherry orchards and is an efficient vector of XMLO. However, C. montanus survives poorly on cherry trees and prefers herbaceous hosts. Important herbaceous hosts of C. montanus are curly dock (Rumex crispus L.), burr clover (Medicago hispida L.), which can be a source for XMLO inoculum, and other clovers (Trifolium spp.). Colladonius montanus develops on these and other herbaceous hosts, and then disperses to adjacent cherry orchards. Since C. montanus is highly dispersive and the most abundant vector found in cherry orchards, C. montanus is believed to be the most likely vector to introduce XMLO into healthy cherry orchards (9). Fieberiella florii is much less abundant than C. montanus in cherry orchards. However, F. florii can reproduce on cherry and is an efficient vector of XMLO (2,7). Fieberiella florii is apparently responsible for rapid tree-to-tree spread of XMLO within infected orchards because diseased cherry trees are a primary source of inoculum in California (3,13).

Visual diagnosis of XD on Bing sweet cherry is based on fruit appearance and tree growth. With the rootstock having a major influence on disease symptomatology (1). Trees on Mazzard rootstock, P. avium, infected with XMLO produce fruit that are small, conical in shape, pale red in color and have shorter and thicker pedicels than normal fruit. Leaves are smaller, slightly chlorotic, often with wavy margins. Trees in advanced stages of decline lack vigor and the canopy exhibits a general "see-through appearance." XD-infected trees on Mazzard rootstock slowly decline in vigor and may survive for many years before dying (1,10). Trees on Prunus mahaleb L. rootstock infected with XMLO produce fruit and pedicel symptoms similar to those on Mazzard rootstock, but tree decline and death occur rapidly. This rapid tree death occurs because the XMLO causes a hypersensitive reaction at the P. mahaleb x P. avium graft union that girdles the scion. Trunk grafted "low-worked" XD-infected trees decline and die uniformly. However, in scaffold limb grafted "high-worked" trees, a single scaffold limb may become infected with XD and die in mid-season while the other high-worked scaffold limbs remain healthy. Visual symptoms of XD occur approximately 1 year after the tree is infected with XD (A. H. Purcell, unpublished data). Sole reliance on visual symptoms to provide positive identification of XD trees can lead to false positive results. However, diagnostic tests such as enzyme-linked immunoassay assay (ELISA) and DNA hybridization assays using cloned fragments of the XMLO chromosome (3,4) provide the means to unambiguously diagnose XMLO trees.

An abatement program for XD was developed by the University of California in 1985 to limit the spread of this disease in San Joaquin County. The program consists of removal of XD-infected trees to reduce XD inoculum in the orchard and post-harvest application of long-residual insecticides to reduce leaffopper populations (9). Identification of XD-infected trees is based on symptomatic fruit and tree growth prior to harvest. Immediately after harvest, a long-residual insecticide is applied to eliminate leaffopper vectors and all XD-infected trees are removed from the orchard. Thereafter, at approximately 6-week intervals, the orchard is retreated with a long-residual insecticide. This abatement program is now practiced in about 80% of bearing cherry orchards in San Joaquin County, although the second or third insecticide application is often omitted. Reported here is an evaluation of the effectiveness of the abatement program and its components (XD tree removal and insecticide applications) in suppression of XD.

MATERIALS AND METHODS

The study was conducted from 1986 through 1990 in mature Bing sweet cherry orchards located near Stockton, California. Prunus mahaleb was the principal rootstock, either scaffold limb or trunk grafted, while Mazzard (P. avium), Colt (P. avium x Prunus cerasus), or Stockton Morello (P. cerasus) were the principal replant rootstocks (Table 1). All cherry trees or tree positions in seven orchards (approximately 5 ha each) were visually inspected from 1986 through 1990. Two additional orchards were included in the study from 1987 through 1990. The orchards were inspected each year prior to harvest (mid-May to mid-June) and each tree or tree position was classified based on fruit appearance and tree growth as (i) healthy in appearance, (ii) replanted (a tree less than

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4 years old), (iii) WXD symptomatic, or (iv) other (dead or missing trees, or trees with the symptoms of crown or root rot caused by Phytophthora spp.). All WXD-symptomatic trees from six orchards 1987 and all orchards 1988 through 1990 were tested using polyclonal ELISA and a DNA hybridization assay, both of which used cloned fragments of the XMLO chromosome as probes (4). Trees that were WXD symptomatic but tested negative in the above tests were classified as healthy. In addition, WXD symptomatic trees that were not tested by ELISA and DNA hybridization assays (all orchards in 1986 and three orchards in 1987) and were healthy the next year were classified as healthy. This procedure prevented false classification of WXD-infected trees. Colladonus montanus and F. florii populations were monitored using yellow whitefly sticky traps (Seabright Enterprises, Emeryville, Calif.). Traps were placed about 1.8 m above the ground in six cherry trees in each orchard. The traps were placed a minimum of five trees and/or rows (30 to 40 m) apart in a circular pattern within the orchards. Traps were monitored weekly from 23 April through 7 October 1987 and from 8 April through 28 October 1988. Traps were replaced every month or when diet or debris interfered with their effectiveness.

The data were analyzed by multivariate linear regression using the MGLH module of SYSTAT (18). Percentage of trees newly infected with WXD was regressed as the dependent factor against the following two independent factors in a stepwise manner: (i) the percentage of WXD-infected trees remaining in the orchard from one year to the next year, i.e., the number of WXD-infected trees the first year and also infected the second year divided by the number of WXD-susceptible trees in the second year; and (ii) the post-harvest insecticide residual longevity (in days) of the insecticide program in the same year that the WXD-infected trees remained in the orchard transformed to the inverse of residual longevity + 1. Susceptible trees excluded trees less than 4 years old, dead or missing trees, or trees with symptoms of Phytophthora crown or root rot. Trees less than 4 years old are WXD asymptomatic. The minimum tolerance for entry of a independent factor in the regression analysis was P < 0.01.

The percentage of trees newly infected with WXD was calculated as the number of WXD-infected trees in the second year that were healthy the previous year divided by the number of WXD-susceptible trees in the second year with a 1-year lag. The 1-year lag of new WXD infections was used because appearance of visual symptoms of WXD occurs approximately 1 year after the tree is infected with XMLO. In inoculations of field-grown cherry trees by C. montanus, symptoms of WXD appeared the following summer, regardless of whether inoculations were made during the spring or summer (A. H. Purcell, unpublished data). Acquisition from cherry and transmission of the pathogen by C. montanus is most likely during June through September (13).

### RESULTS AND DISCUSSION

The WXD abatement program, which consisted of diseased tree removal and applications of long-residual insecticides, gained ingrower acceptance during the study period. Mean percent WXD-infected trees remaining in the orchards decreased from 1.7% in 1986 to 0.9% in 1988 (Table 3). If orchard 8, which did not follow the abatement program, is excluded from the mean, then mean percent WXD-infected trees remaining in the orchards decreased from 1.7% in 1986 to 0.3% in 1988 to 1989. Also, growers increased their use of long-residual insecticides from a mean residual longevity of 31.7 days in 1986 to 75.0 days in 1989 (Table 2). If orchard 8 is again excluded, then the mean residual longevity increased from 31.7 days in 1986 to 84.4 days in 1989. The mean number ± SEM of healthy trees per orchard increased from 837 ± 216 in 1986 to 941 ± 176 in 1990. This increase in the number of healthy trees was due to an-

### Table 1. Rootstocks, number of trees or tree positions and hectares inspected annually for western X-disease in nine sweet cherry orchards near Stockton, California

<table>
<thead>
<tr>
<th>Orchard</th>
<th>No. of ha</th>
<th>No. of trees inspected</th>
<th>Original rootstock&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Replant rootstock&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3.7</td>
<td>895</td>
<td>80% S. G. Mahaleb</td>
<td>S. G. Mahaleb</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>20% T. G. Mazzard</td>
<td>Mazzard</td>
</tr>
<tr>
<td>2</td>
<td>4.9</td>
<td>1,087</td>
<td>S. G. Mahaleb</td>
<td>Colt</td>
</tr>
<tr>
<td>3</td>
<td>4.0</td>
<td>1,195</td>
<td>T. G. Mahaleb</td>
<td>Colt</td>
</tr>
<tr>
<td>4</td>
<td>4.2</td>
<td>1,131</td>
<td>S. G. Mahaleb</td>
<td>Colt</td>
</tr>
<tr>
<td>5</td>
<td>6.4</td>
<td>1,525</td>
<td>S. G. Mahaleb</td>
<td>Colt</td>
</tr>
<tr>
<td>6</td>
<td>3.7</td>
<td>1,002</td>
<td>T. G. Mahaleb</td>
<td>Colt or Mazzard</td>
</tr>
<tr>
<td>7</td>
<td>6.9</td>
<td>1,293</td>
<td>S. G. Mahaleb</td>
<td>Mazzard</td>
</tr>
<tr>
<td>8</td>
<td>5.4</td>
<td>797</td>
<td>T. G. Mahaleb</td>
<td>Stockton Morello</td>
</tr>
<tr>
<td>9</td>
<td>5.0</td>
<td>1,345</td>
<td>Stockton Morello</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Orchards were originally planted on these rootstocks, where S. G. = scaffold limb grafted, T. G. = trunk grafted, Mahaleb = P. mahaleb, Mazzard = P. avium.

<sup>b</sup> Stockton Morello = Prunus cerasus L, Colt = P. avium x P. cerasus and Mazzard rootstocks were all trunk grafted.

### Table 2. Insecticide use pattern, residual longevity, and Colladonus montanus population in nine sweet cherry orchards near Stockton, California

<table>
<thead>
<tr>
<th>Insecticide use pattern and residual longevity (days)&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Mean no. of C. montanus per trap per season&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>---------</td>
<td>------</td>
</tr>
<tr>
<td>1</td>
<td>3 (3)</td>
</tr>
<tr>
<td>2</td>
<td>5 (42)</td>
</tr>
<tr>
<td>3</td>
<td>3 (3)</td>
</tr>
<tr>
<td>4</td>
<td>1 (1)</td>
</tr>
<tr>
<td>5</td>
<td>9 (168)</td>
</tr>
<tr>
<td>6</td>
<td>3 (3)</td>
</tr>
<tr>
<td>7</td>
<td>2 (2)</td>
</tr>
<tr>
<td>8</td>
<td>...</td>
</tr>
<tr>
<td>9</td>
<td>...</td>
</tr>
<tr>
<td>Mean</td>
<td>3.7 (31.7)</td>
</tr>
<tr>
<td>+ SEM</td>
<td>2.7 (53.7)</td>
</tr>
</tbody>
</table>

<sup>a</sup> Insecticides used post-harvest: 0 = untreated; 1 = dazinon application; 2 = dazinon applications; 3 = dazinon applications; 4 = carbaryl application; 5 = 1 esfenvalerate application; 6 = 1 esfenvalerate and 1 dazinon application; 2 = esfenvalerate applications; 8 = 3 esfenvalerate applications; and 9 = 4 esfenvalerate applications.

<sup>b</sup> Six traps per orchard monitored from April to October. No Fieberiella florii were captured during the study.
crease in the number of replanted trees
attaining the fourth year of growth, a de-
crease in the number of new WXD-infected
trees, and corresponding decrease in the
number of trees removed from the orchards.

The stepwise linear regression of per-
centage of new WXD infections against
insecticide residual longevity and the per-
centage of WXD-infected trees remaining
in the orchards from one year to the next
(N = 25) indicated that percentage of
WXD-infected trees remaining in orchards
was the best predictor of new infections
(Table 4). The regression analysis suggests
that the WXD-infected tree removal com-
ponent of the abatement program is pri-
marily responsible for the reduction of
new WXD infections and explains 75% of
the variation, whereas the insecticide re-
sidual longevity contributed only a minor
amount to the decrease of new infections.
Inclusion of both insecticide residual lon-
gevity and percent WXD-infected trees
remaining in the orchards explained 81% of
the variation.

Because leafhopper populations were
not monitored in 1986, leafhopper popu-
lations were not included in the stepwise
linear regression analysis. However, the
correlation of the mean number of C.
montanus per trap per year for 1987 and
1988 with insecticide residual longevity
for 1987 and 1988 (N = 18) was not signi-
ificant (F = 2.94; df = 1,117; P = 0.104; \(r^2 = 0.14\)). No F. florii were captured
during the study. Also, Spearman’s rank correla-
tion coefficients of the mean number of C.
montanus per trap per year for 1987 and
1988 with insecticide use pattern and in-
secticide residual longevity for 1987 and
1988 (N = 18) was not significant (−0.05
and 0.027, respectively, P > 0.1). The lack of
a significant correlation between C.
montanus populations and insecticide resi-
dual longevity was expected because C.
montanus is highly dispersive (9,17) and
would be captured in yellow sticky traps in
spite of lethal insecticide residues. In ad-
dition, because no F. florii that can cause
rapid tree-to-tree spread of WXD were
captured during the study, the low percent-
age (0.0 to 6.4%) of new WXD-infected
trees (Table 3) was expected and new in-
fecteds probably resulted from C. monta-
thus transmission.

Orchards 7 and 9 were adjacent to
cherry orchards that had large numbers of
WXD-infected trees. These adjacent or-
chards practiced no WXD abatement pro-
cedures, neither WXD-infected tree re-
moval nor applications of long-residual in-
ssecticides to reduce the leafhopper popu-
lations. Orchards 7 and 9 practiced thor-
ough WXD-infected tree removal (Table 3)
and insecticide programs (Table 2) but had
2.7 and 2.22% new WXD-infected trees
(respectively) in 1988 to 1989 evaluations
and 1.1 and 0.2% new WXD-infected trees
(respectively) in 1989 to 1990 evaluations
(Table 3). In 1987, orchards 7 and 9 had
the highest C. montanus population and in
1988 orchard 9 had the highest C. monta-
thus population (Table 2). We speculate
that the C. montanus transmitted WXD
from the adjacent orchard into orchards 7
and 9. The data were re-analyzed without
orchards 7 and 9 by stepwise linear re-
gression. The percentage of new WXD
infections was regressed against the per-
centage of WXD-infected trees remaining
in the orchards from one year to the next
and insecticide residual longevity (N =
20). The regression analysis again indi-
cated that the percentage of diseased trees
remaining in the orchards was the best pre-
dictor of new infections (Table 4). The
WXD-infected tree removal component of
the abatement program was primarily re-
sponsible for the reduction in new WXD
infections and explained 84% of the varia-
tion, while insecticide residual longevity
contributed only a minor amount of the
suppression of new infections. Inclusion of
both insecticide residual activity and per-
cent WXD-infected trees remaining in the
orchards explained 91% of the variation.
The increase in explained variation from
75%, when the percentage of new WXD
infections was regressed against the per-
centage of WXD-infected trees remaining
in the orchards from one year to the next
with orchards 7 and 9 included, to 84%,
with orchards 7 and 9 excluded from the
regression analysis, indicates that a large
immigration of WXD-infected C. monta-
thus was probably the cause of new infec-
tions in orchards 7 and 9. The high rate of
new infections in orchards 7 and 9, which
practiced thorough abatement procedures
but were adjacent to a large source of dis-
eased trees, suggests that WXD abatement
procedures should be practiced on an area-
wide basis to be most effective. Orchards
containing WXD-infected trees and not
practicing WXD abatement procedures can
serve as sources of both inoculum and
leafhoppers. Dispersal of WXD-infected
leafhoppers from these orchards can cause
new WXD infections in adjacent orchards
and elimination of dispersing WXD-in-
fected leafhoppers cannot be achieved by
repeated insecticide applications.

This study found that abatement of
WXD in cherry can be achieved by the
rigorous removal of WXD-infected trees
and repeated applications of long-residual
insecticides. WXD-infected tree removal is
primarily responsible for the reduction of
new WXD infections while insecticide resi-
dual longevity contributes only a minor
amount in suppression of new infections.
WXD abatement procedures should be
most effective when practiced on an area-
wide basis.

Table 3. Percentage of trees infected with western X-disease (WXD) remaining in the orchards from one year to the next and percentage of trees newly infected with WXD in nine sweet cherry orchards near Stockton, California

<table>
<thead>
<tr>
<th>Percent WXD-infected trees remaining</th>
<th>Percent new WXD-infected trees</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.5</td>
</tr>
<tr>
<td>2</td>
<td>1.5</td>
</tr>
<tr>
<td>3</td>
<td>0.7</td>
</tr>
<tr>
<td>4</td>
<td>0.4</td>
</tr>
<tr>
<td>5</td>
<td>0.0</td>
</tr>
<tr>
<td>6</td>
<td>9.1</td>
</tr>
<tr>
<td>7</td>
<td>0.0</td>
</tr>
<tr>
<td>8</td>
<td>3.9</td>
</tr>
<tr>
<td>9</td>
<td>...</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>1.7</td>
</tr>
<tr>
<td><strong>+ SEM</strong></td>
<td>3.0</td>
</tr>
</tbody>
</table>

* Number of WXD-infected trees remaining in the orchards from one year to the next year divided by the number of WXD-susceptible trees.

Table 4. Coefficients and corresponding statistics from the stepwise multivariate linear regression of new western X-disease (WXD) infections against WXD-infected trees remaining in the orchards from one year to the next and insecticide residual longevity in orchards near Stockton, California

<table>
<thead>
<tr>
<th>Coefficients</th>
<th>WXD-infected trees remaining</th>
<th>Insecticide residual longevity</th>
<th>Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nine sweet cherry orchards</td>
<td>0.74</td>
<td>...</td>
<td>68.1</td>
</tr>
<tr>
<td>0.56</td>
<td>2.1</td>
<td>48.6</td>
<td>2.23</td>
</tr>
<tr>
<td>Seven sweet cherry orchards</td>
<td>0.74</td>
<td>...</td>
<td>92.4</td>
</tr>
<tr>
<td>0.55</td>
<td>2.2</td>
<td>88.2</td>
<td>2.18</td>
</tr>
</tbody>
</table>

* Without orchards 7 and 9.
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LITERATURE CITED


