

Reductions in Yield of Processing Tomatoes and Incidence of Bacterial Canker

R. J. CHANG, S. M. RIES, and J. K. PATAKY, Department of Plant Pathology, University of Illinois, Urbana 61801

ABSTRACT

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Various incidences of systemic infection of bacterial canker, caused by *Clavibacter michiganensis* subsp. *michiganensis*, were established in field plots of tomato, *Lycopersicon esculentum*. In 2 yr, total fruit weight, percent maximum yield, and average fruit weight of processing tomatoes (cv. Heinz 1810) were related to incidence of systemic infection of bacterial canker 1 wk before harvest. For plants infected during clipping or seedling harvest, total fruit weight per plot decreased as much as 58 kg, maximum yield decreased as much as 46%, and average fruit weight decreased as much as 13 g when the highest incidence of systemic infection was 31–83%. Slope coefficients from regressions of maximum yield on incidence ranged from -0.5 to -0.7 , indicating that yield decreased about 5–7% for each 10% increase in incidence. The percentages of green and ripe fruit also were affected significantly by the incidence of systemic infection. The percentage of green fruit decreased as much as 41% and the percentage of ripe fruit increased as much as 41% when the highest incidence of systemic infection was 31–83%. The relationships between yield, green and ripe fruit, and incidence of bacterial canker were similar for plants infected during clipping of transplants and during harvest of transplants.

Bacterial canker, caused by *Clavibacter michiganensis* subsp. *michiganensis* (Smith) Davis et al, is one of the most destructive diseases of tomato (*Lycopersicon esculentum* Mill.). Although occurrence is sporadic, bacterial canker can be devastating, especially on transplanted or direct-seeded tomatoes that have been clipped or pruned (3,11). Outbreaks of canker have caused substantial losses in the United States and Canada, primarily because of the general decline or death of plants that were systemically infected (4,9). Bird's-eye spots, which result from secondary spread of the disease late in the cropping cycle, generally do not affect the quality of tomatoes grown for processing (14), but spotted fruits can cause substantial losses of fresh-market tomatoes, especially after extended periods of wet weather (13).

Yield losses attributable to bacterial canker in field and greenhouse tomato crops vary among years, locations, cultivars, and time of inoculation (5,6,13). Some growers in the United States, Canada, and Kenya have sustained losses

as high as 50–80% (4,13,16,17). Bryan (1) reported that losses in fields ranged from a trace to 100%, and lesions on fruit caused losses from 25 to 75% in some fields of fresh-market tomatoes. Kennedy and Alcorn (12) estimated that losses from bacterial canker were approximately \$200,000 in 1976. Generally, these reports (1,12,13) have been based on estimates rather than on experimental data. Relationships between reductions in yield of processing tomatoes and the incidence of bacterial canker would improve the management of this disease. Accurate estimates of yield losses could be useful in determining economic thresholds and in establishing tolerance levels for seedling certification programs (8,9). The objective of our study was to relate reductions in yield of processing tomatoes with the incidence of bacterial canker.

MATERIALS AND METHODS

Rifampin-resistant mutants and preparation of inocula. Ten strains of *C. m. michiganensis* obtained from diverse geographical locations (3) were selected for rifampin resistance as described by Weller and Saettler (20). Inocula were a mixture of equal proportion of the 10 strains of rifampin-resistant *C. m. michiganensis* prepared as described previously (3).

Field studies. Yield losses caused by bacterial canker were evaluated in two field studies in which various percentages of tomato seedlings became systemically

wilted after transplants were clipped or after seedlings were harvested and transplanted (3).

In the clipping study, tomato seeds (cv. Heinz 1810) were obtained from D. A. Emmatty (Heinz USA, Pittsburgh, PA) and sown in 15 and 18 beds on 10 May 1988 and 12 May 1989, respectively, at the University of Illinois Pomology Farm, Urbana, IL. Each bed was 21 m long and consisted of five rows spaced 28 cm apart with one seed per centimeter. Each bed contained approximately 10,000 seedlings and was separated from adjacent beds by 1.8 m.

Five or six treatments were used to establish various incidences of systemic infection. In 1988, the five treatments included initial disease incidences of 0.01, 0.05, 0.1, and 0.5% and an uninoculated control treatment. In 1989, an initial disease incidence of 0.2% also was included. Treatments were arranged in a randomized complete block design with three replicates. Seedlings were inoculated 3–4 wk after direct-seeding by removing the first true leaf at its point of attachment with scissors dipped in inocula as described by Thyr (19).

Seedlings within each bed were clipped successively eight times as described previously (3). After the final clipping, 100 and 120 seedlings were selected from various locations in each bed and transplanted to production fields in another area of the Pomology Farm on 12 July 1988 and 6 July 1989, respectively. These seedlings were dug and handled individually to minimize contamination among transplants. In the production fields, transplants were spaced 30 cm apart within rows and 90 cm between rows. Each plot (experimental unit) consisted of four rows with 25 and 30 transplants per row in 1988 and 1989, respectively. The incidence of systemic infection was based on visual observations at 7-day intervals after transplanting. Disease ratings on 2 October 1988 and 28 September 1989, 1 wk before harvest, were used to assess the effects of bacterial canker on yield. Incidence was converted to a percentage of diseased plants per plot.

Tomato fruits from 40 and 50 plants in the center two rows of each experimental unit were harvested on 9 October

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1988 and 5 October 1989 (about 90 days after transplanting), respectively. The plants were cut and the fruits were shaken into bushel baskets. The total fruit weight of each experimental unit was measured. Percentages of green, ripe, and rotten fruit were determined from one bushel of fruits per experimental unit. The average fruit weight was obtained from the total fruit weight per bushel divided by the total number of fruits per bushel.

In the seedling harvest study, tomato seeds (cv. Heinz 1810) were sown in 35 × 50 cm flats on 6 April 1988 and 7 April 1989 as described previously (3). Three flats of about 250 direct-seeded tomato seedlings were inoculated on 4 May 1988 and 10 May 1989 by cutting the first true leaf with scissors dipped in inocula as described above. Forty-eight flats of healthy seedlings were placed in a different greenhouse. When seedlings were about 25 cm tall, healthy seedlings were removed from flats by hand and mixed with asymptomatic, in-

oculated seedlings of the same age according to the four treatments (contamination rates) described below. Each mixture contained 200 seedlings per experimental unit. The mixtures of healthy and inoculated seedlings were vigorously shaken and slapped against a hard surface to remove soil from the roots.

Four treatments were evaluated. Three treatments were contamination rates of 1, 5, and 10% asymptomatic, inoculated seedlings with uninoculated seedlings. One treatment was an uninoculated control. For contamination rates of 1, 5, and 10%, two, 10, and 20 asymptomatic, inoculated seedlings were mixed with 198, 190, and 180 healthy seedlings, respectively, for each experimental unit. Treatments were arranged in a randomized complete block design with three replicates. A total of 200 seedlings per experimental unit were stored overnight in a cold room at 4 C and transplanted to a production field in the University of Illinois Pomology Farm on 26 May 1988

and 24 May 1989. In the production field, transplants were spaced 30 cm apart within rows and 90 cm between rows. Each experimental unit consisted of four rows with 50 transplants per row. The incidence of systemic infection was measured as the percentage of diseased plants per experimental unit by visual observations at 7-day intervals after transplanting. Disease ratings on 28 August 1988 and 24 August 1989, 1 wk before harvest, were used to assess the effects of bacterial canker on yield.

Tomato fruits from 80 plants in the center two rows of each experimental unit were harvested on 4 September 1988 and 31 August 1989 (about 100 days after transplanting) as described above. Total fruit weight and percentages of green, ripe, and rotten fruit were measured as described previously.

Statistical analyses. Data were analyzed by ANOVA with initial disease incidence (clipping study) and contamination rates (seedling harvest study) as independent variables to obtain estimates of the error mean squares used in subsequent analyses. Total fruit weight, average fruit weight, and percentages of rotten, green, and ripe fruit were regressed on incidence of systemic infection 1 wk before harvest. Regressions were done with treatment means and with data from each experimental unit. The intercept (b_0) of the regression of the total fruit weight on incidence of systemic infection was used as an estimate of maximum yield for the cultivar Heinz 1810 in each experiment. Total fruit weight from each experimental unit then was converted to the percentage of the maximum yield. The percentage of the maximum total fruit weight then was regressed on incidence of systemic infection 1 wk before harvest. For all regressions, F statistics ($P < 0.05$) were calculated with the error mean squares from the full (factorial) model. Coefficients of determination (r^2) were calculated to determine the variation explained by the model. Residuals from the regression models were evaluated for lack of fit and outliers to determine the appropriate models.

RESULTS

Incidence of bacterial canker. Incidence of systemic infection of bacterial canker 1 wk before harvest ranged from 0 to 83% in 1988 and from 4 to 51% in 1989 in the clipping study and from 0 to 52% in 1988 and from 1 to 31% in 1989 in the seedling harvest study.

Symptoms of systemically infected plants were first observed about 14–20 days after transplanting in the clipping study and 35–42 days after transplanting in the seedling harvest study. Symptoms appeared later in treatments with the lower levels of initial disease incidence or contamination rates. In 1989, wilted plants were observed in control plots 49

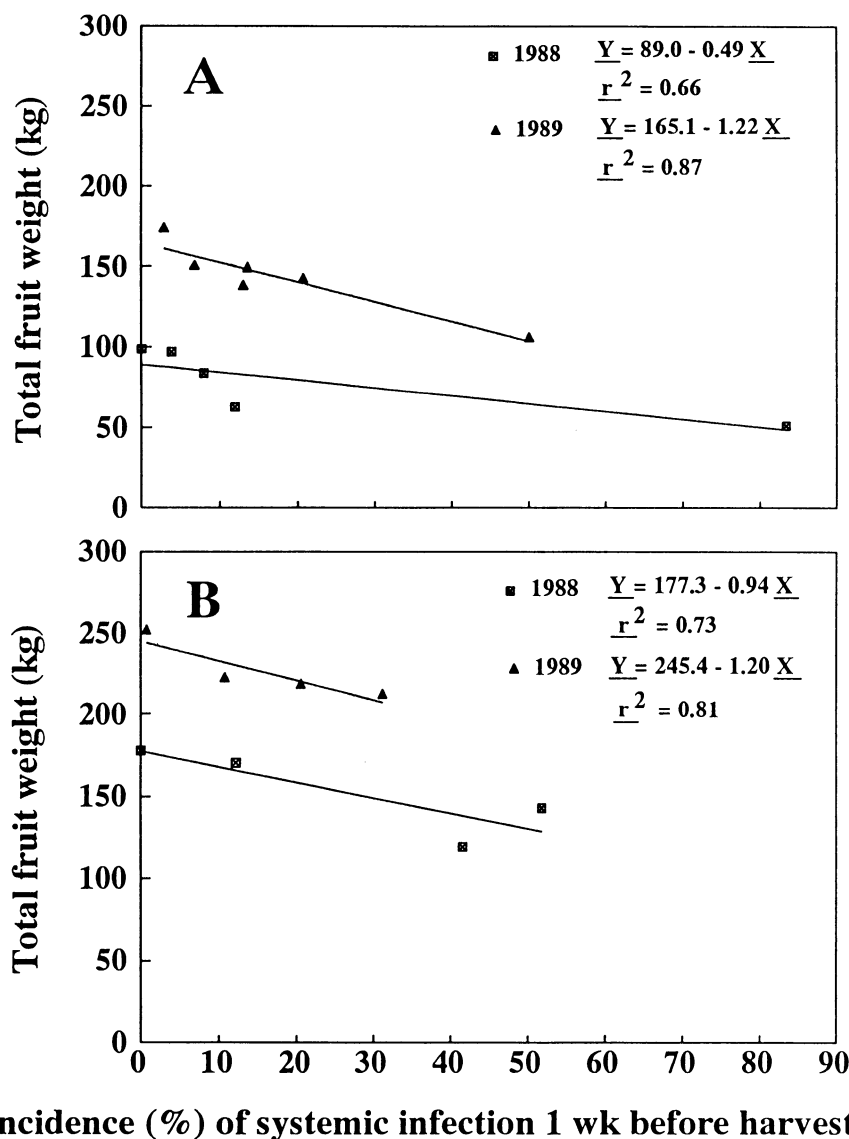


Fig. 1. Relationships between total fruit weight per plot of tomatoes and incidence of systemic infection of bacterial canker 1 wk before harvest in 1988 and 1989. Plants were infected during (A) clipping or (B) seedling harvest.

days after transplanting the clipped seedlings and 77 days after transplanting in the seedling harvest study. Within each study, disease development was similar between years, but disease incidence was higher in 1988 in both studies. Maximum disease incidence occurred 65–75 days after transplanting in the clipping study and 85–90 days after transplanting in the seedling harvest study. Typical one-sided wilting and stunting of systemically infected plants were observed at the end of each season. Some infected plants died before harvest. Symptoms of bird's-eye spots were observed on a few plants in seedling harvest plots in 1989.

Yield. Total fruit weight decreased as the incidence of systemic infection 1 wk before harvest increased (Fig. 1). Total fruit weight of plants infected during clipping decreased about 0.49–1.22 kg per plot for each 1% increase in incidence (Fig. 1A). Total fruit weight of plants infected during seedling harvest decreased about 0.94–1.20 kg per plot for each 1% increase in incidence (Fig. 1B). These relationships were described best by linear models.

Maximum yield (as a percentage of total fruit weight) decreased significantly as the incidence of systemic infection 1 wk before harvest increased (Fig. 2). Maximum yield of plants infected during clipping decreased about 0.6–0.7% for each 1% increase in incidence (Fig. 2A). Maximum yield of plants infected during seedling harvest decreased about 0.5% for each 1% increase in incidence (Fig. 2B). These relationships were described best by linear models.

Average fruit weight decreased as the incidence of systemic infection 1 wk before harvest increased (Fig. 3). Average fruit weight from plants infected during clipping decreased about 0.1–0.3 g for each 1% increase in incidence (Fig. 3A). Average fruit weight from plants infected during seedling harvest decreased about 0.2–0.3 g for each 1% increase in incidence (Fig. 3B). These relationships were described best by linear models.

Early fruit senescence occurred on plants infected with bacterial canker. The percentages of green, ripe, or rotten fruit were related significantly to the incidence of systemic infection 1 wk before harvest (Fig. 4). Although defoliation did not occur on systemically infected plants, sunscald was more prevalent on fruits from systemically infected plants in 1988. Generally, blossom end rot and anthracnose were the major fruit rots observed in both studies.

The percentage of green fruit decreased as the incidence of systemic infection increased (Fig. 4A and B). For plants infected during clipping, green fruit decreased about 0.3% in 1988 and 0.9% in 1989 for each 1% increase in incidence (Fig. 4A). For plants infected during seedling harvest, green fruit

decreased about 0.2–0.3% for each 1% increase in incidence (Fig. 4B).

The percentage of ripe fruit increased with the incidence of systemic infection in three of four trials (Fig. 4C and D). For plants infected during clipping, ripe fruit increased about 0.2% in 1988 and 0.9% in 1989 for each 1% increase in incidence (Fig. 4C). For plants infected during seedling harvest, ripe fruit was not related to incidence in 1988 but increased about 0.3% for each 1% increase in incidence in 1989 (Fig. 4D).

The percentage of rotten fruit was not related to incidence of systemic infection 1 wk before harvest except for plants infected during seedling harvest in 1988 (Fig. 4E and F). Rotten fruit increased about 0.2% for each 1% increase in incidence of systemically infected plants in 1988 (Fig. 4F).

DISCUSSION

Decreases in fruit weight and yield of processing tomatoes were related to the

incidence of systemic infection due to bacterial canker. Total fruit weight decreased about 0.5–1.2 kg per plot, maximum yield decreased about 0.5–0.7%, and average fruit weight decreased about 0.1–0.3 g for each 1% increase in incidence of systemic infection. Also, the percentage of green fruit decreased and the percentage of ripe or rotten fruit increased as the incidence of systemic infection increased. Thus, bacterial canker was associated with significant reductions in yield of processing tomato in our studies. Similarly, Emmatty and John (6) observed 11–99% yield reductions of tomatoes inoculated with *C. m. michiganensis*, and Carlton et al (2) reported higher disease incidence and lower yield after inoculation.

More ripe fruits and fewer green fruits were harvested from systemically infected tomato plants. In the clipping experiment, the percentage of green fruit decreased as much as 41% and the percentage of ripe fruit increased an equiv-

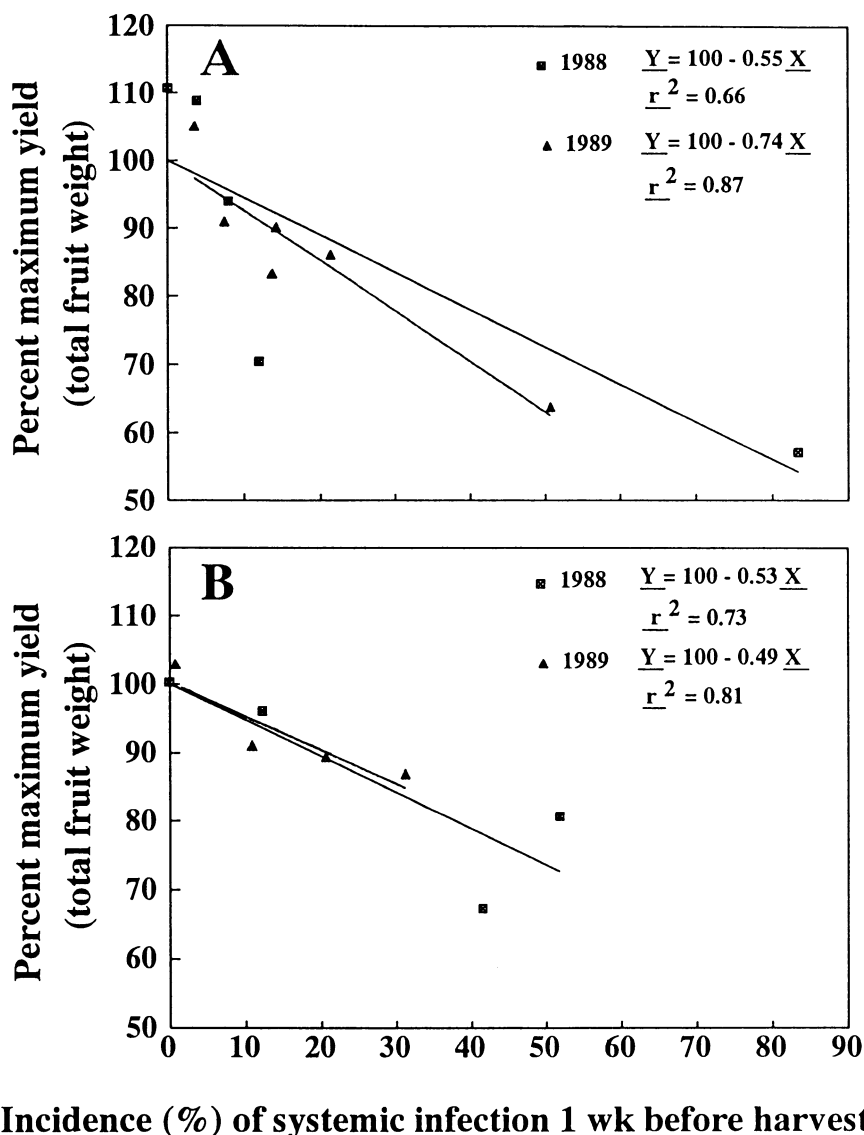
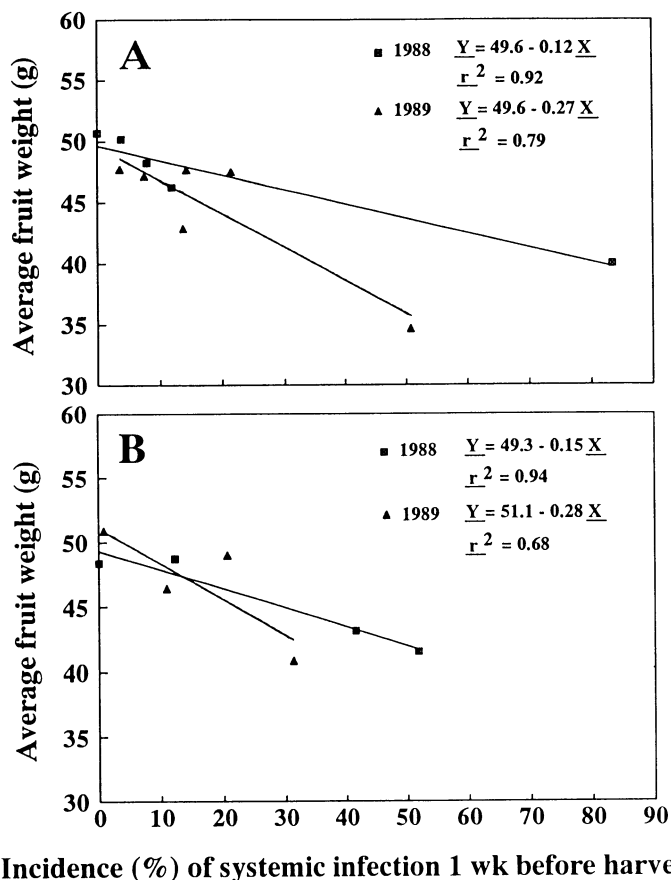
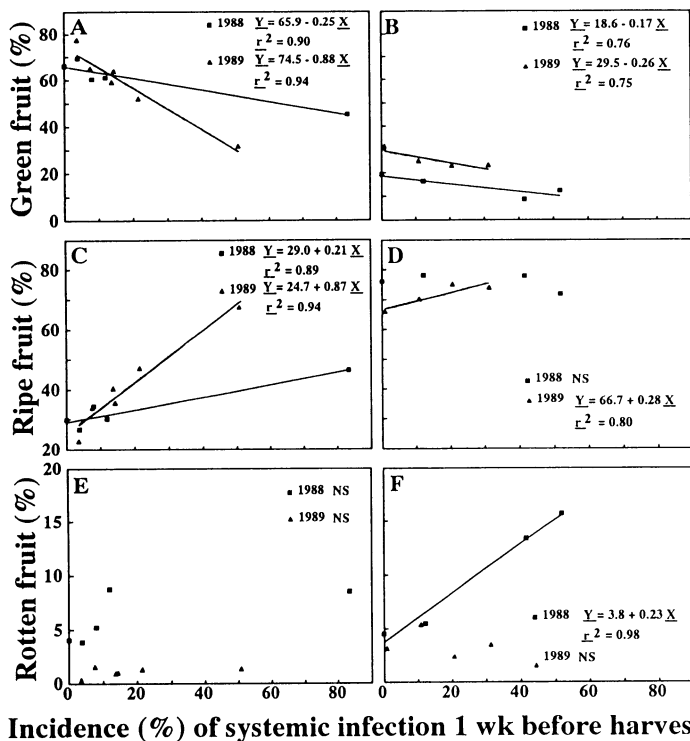


Fig. 2. Relationships between percentage maximum yield of tomatoes and incidence of systemic infection of bacterial canker 1 wk before harvest in 1988 and 1989. Plants were infected during (A) clipping or (B) seedling harvest.



Incidence (%) of systemic infection 1 wk before harvest

Fig. 3. Relationships between average fruit weight of tomatoes and incidence of systemic infection of bacterial canker 1 wk before harvest in 1988 and 1989. Plants were infected during (A) clipping or (B) seedling harvest.



Incidence (%) of systemic infection 1 wk before harvest

Fig. 4. Relationships between (A and B) percent green fruit, (C and D) percent ripe fruit, and (E and F) percent rotten fruit of tomatoes and incidence of systemic infection of bacterial canker 1 wk before harvest in 1988 and 1989. Plants were infected during clipping (A, C, and E) or seedling harvest (B, D, and F).

alent amount when incidence of systemic infection was more than 50%. Similarly, in the seedling harvest experiment, the percentage of green fruit decreased as much as 9%, and the percentage of ripe or rotten fruit increased that amount when incidence of systemic infection was 31–52%. Systemically infected tomato plants may produce larger quantities of ethylene than healthy plants, and this may enhance premature fruit ripening (7,10,15). Although most fruits from systemically infected plants did not have bird's-eye spots, stunted and distorted fruits commonly were observed from severely infected plants. Such fruits may have significantly decreased average fruit weight.

The relationship between rotten fruit and incidence of canker in the seedling harvest experiment in 1988 may have been due to a relatively high percentage of ripe fruit in that study. Tomato fruits were harvested 101 days after transplanting, by which time the percentage of ripe fruit was 76% for the healthy control treatment (0% inoculated plants). It is possible that fruits that ripened early because of disease had dropped or rotted before harvest. Early harvest of fields with a high incidence of canker may avoid losses due to rotting of fruit.

In the absence of individual models for specific cultivars, years, and locations, the regression models from our data provide the best estimates of damage due to systemic infection. These relationships, however, are based on a relatively susceptible cultivar, and they may differ substantially if cultivars with higher levels of resistance are developed. For example, relationships between yield reduction and incidence of infected plants differ considerably for Stewart's and Goss's wilt of sweet corn (18). The regressions were similar for plants infected during clipping and seedling harvest. The potentially devastating effects and economic importance of bacterial canker are illustrated by these relationships. A contamination rate of 1% inoculated transplants during seedling harvest resulted in 7–11% systemically infected plants (3). Based on relationships derived from our data, yield would be reduced by about 3–8% at that disease incidence. If the price of processing tomatoes is \$70 per ton and average production is 75 tons per hectare (D. A. Emmatty, *personal communication*), losses attributable to a 7–11% incidence would be from \$180 to \$430 per hectare. Thus, a tolerance level for seedling certification would need to be less than 1% to avoid significant economic loss if natural contamination of transplants is similar to those in our studies (3). For fresh-market tomatoes, in which secondary infection also reduces quality and yield, levels for seedling certification need to be lower.

Control practices for bacterial canker

of tomato have been based primarily on the use of certified seed and transplants (11,17). However, because of a long incubation period and lack of adequate methods to detect low populations of *C. m. michiganensis* from infested seed and symptomless transplants, the disease continues to be a threat. Hence, host resistance seems to be the most promising approach for disease management if resistance can be incorporated into high-yielding, adapted cultivars. Partial resistance (i.e., tolerance) to *C. m. michiganensis* has been expressed as reduced severity of symptoms, which probably is associated with a lower level of systemic infection by *C. m. michiganensis*, and corresponds to lower reductions in yield (6,19).

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