Dissemination of *Clavibacter michiganensis* subsp. *michiganensis* by Practices Used to Produce Tomato Transplants

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ABSTRACT

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Rifampin-resistant (Rif⁺) strains of *Clavibacter michiganensis* subsp. *michiganensis* were used to study the epidemiology of bacterial canker of tomato (*Lycopersicon esculentum*). Clipping tomato seedlings in transplant beds with a rotary mower transmitted Rif⁺ *C. m. michiganensis* from inoculated seedlings to healthy seedlings. Epiphytic populations of Rif⁺ *C. m. michiganensis* were first detected on clipped, asymptomatic seedlings 9–13 days after the first clipping. Populations of Rif⁺ *C. m. michiganensis* on clipped seedlings ranged from 0 to about 10^7 cfu/g fresh weight throughout the clipping period. Symptoms did not appear on these seedlings until 14–20 days after transplanting, or about 31–40 days after the first clipping. The incidence of systemically infected plants in production fields 1 wk before harvest increased about 10% for each 0.1% increase in incidence of infected seedlings in transplant beds before clipping. When healthy and diseased seedlings were harvested and mixed in bundles at various contamination rates, Rif⁺ *C. m. michiganensis* was

spread from diseased to healthy seedlings by seedling harvest practices. Populations of Rif⁺ *C. m. michiganensis* recovered from bundles of transplants, and incidence of systemically infected plants in production fields were affected significantly by the percentage of diseased seedlings per bundle (i.e., contamination rates). Symptoms of systemic infection on healthy seedlings mixed with infected seedlings were first observed 35–42 days after transplanting. Incidence of systemically infected plants in production fields 1 wk before harvest increased about 3–5% for each 1% increase in the contamination rate. Because Rif⁺ *C. m. michiganensis* is spread by clipping and seedling harvest practices used in the production of tomato transplants, bacterial canker can be a cultural practice disease. Seed transmission rates as low as 0.01–0.05% (one to five seeds per 10,000) could initiate an epidemic of bacterial canker in tomato production fields in the Midwest because of these cultural practices.

Bacterial canker of tomato (*Lycopersicon esculentum* Miller) caused by *Clavibacter michiganensis* subsp. *michiganensis* (Smith) Davis et al has been an infrequent but serious disease of processing and fresh market tomatoes. In 1984, severe outbreaks of bacterial canker occurred throughout the midwestern United States and in several Canadian provinces. Substantial losses to the processing tomato industry prompted our investigation of this disease.

A portion of the processing tomato industry in the Midwest depends on transplants from Georgia and Florida where tomatoes are direct seeded in transplant production beds. Seedlings are repeatedly clipped (a pruning practice accomplished with a rotary mower) to obtain plant uniformity and to increase seedling vigor. Clipped plants are inspected frequently and certified to be free of several diseases including bacterial spot and bacterial speck (11). When seedlings reach about 25 cm in height, they are harvested and shipped from Georgia and Florida to production fields in the northern Midwest where they are planted. In years such as 1984, bacterial canker devastated tomato crops in the Midwest despite seed treatment, crop rotation, and seedling certification procedures (8). These outbreaks of bacterial canker were traced to certified transplants from Georgia (8).

Bacterial pathogens can be transported epiphytically and as latent infections of seedlings (5,14,15,17). There is reasonable evidence that plants certified as disease-free are contaminated frequently with plant pathogenic bacteria, and thus, may provide inoculum for primary infection (20,29). The disease cycle for bacterial canker of tomato is not understood clearly. The primary source of inoculum for tomatoes grown commercially for processing may be infected or infested seed (2,7,8,12,21), but because of a long incubation period, infected seedlings may remain symptomless until after they are transplanted (9). Systemically infected

plants often appear 30–40 days after transplanting. *C. m. michiganensis* may ooze from open cankers and cause secondary, surface infection with symptoms, such as marginal leaf scorch of leaflets or bird's eye spots of fruit (21). Survival of *C. m. michiganensis* in production fields has been documented (2,21). Although *C. m. michiganensis* may survive in soil, plant debris, alternative hosts, or volunteer plants, the importance of such inoculum in the development of epidemics of bacterial canker as compared to infested seed and symptomless, infected transplants is not known completely.

Because transplants are used in tomato production in the Midwest, dissemination of *C. m. michiganensis* on symptomless, infected transplants could be extremely important. Because we suspected that *C. m. michiganensis* could be mechanically transmitted by cultural practices in the production of transplants (i.e., clipping, harvesting, shipping, and transplanting), we simulated these practices to follow *C. m. michiganensis* through a complete cycle of tomato production. The objective of this research was to evaluate the dissemination of *C. m. michiganensis* by the cultural practices used to produce tomato transplants. A preliminary report of this study has been presented (3).

MATERIALS AND METHODS

Rifampin-resistant (Rif⁺) mutants and preparation of inoculum.
Ten strains of *C. m. michiganensis* were selected for rifampin resistance as described by Weller and Saettler (28). These strains included OSURLC17, 47, and 61 from S. T. Nameth, Ohio State University; CMM-1 and DR60 from M. L. Gleason, Iowa State University; CM1, CM2, and CM5 obtained from D. A. Emmatty, Heinz USA; and ILCM1, ILCM2, which we isolated from diseased tomato plants in Illinois. All Rif⁺ *C. m. michiganensis* strains were confirmed as pathogenic by producing typical one-sided wilting on tomato plants (cv. Heinz 1810) and a hypersensitive

reaction on leaves of four-o'clock, *Mirabilis jalapa* L. (8). The Rif⁺ *C. m. michiganensis* strains were stored in King's B broth at −80 C and revived before each experiment to avoid repeated subculturing. Inoculum was prepared by transferring frozen cultures to King's B broth, which was incubated at room temperature on shakers for 48 h. Broth cultures then were streaked on nutrient-broth yeast extract agar (NBY) medium amended with rifampin (50 mg/L). Plate cultures were incubated at 25 C for 48–72 h and used as the source of inoculum. Bacteria were suspended in sterile phosphate buffer (0.01 M, pH 7.2) containing 0.85% NaCl (PBS) and adjusted to approximately 2×10^8 cfu/ml (A_{590} = 0.16). The inoculum was a mixture of the 10 strains of Rif⁺ *C. m. michiganensis* to account for variation in virulence that may have existed among strains.

Clipping practices. Tomatoes (cv. Heinz 1810) were direct seeded 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 study relationships between initial disease incidence in transplant beds before clipping and incidence of systemic infection in production fields. In 1988, the five treatments were initial disease incidence of 0.01, 0.05, 0.1, and 0.5% and an uninoculated control treatment. In 1989, an initial disease incidence of 0.2% was also included. Treatments were arranged in a randomized complete block design with three replications. Seedlings were inoculated 3–4 wk after planting by removing the first true leaf at its point of attachment with scissors dipped in inoculum as described by Thyr (25). For initial disease incidence of 0.01%, one seedling was inoculated at the beginning of each seedling bed. For initial disease incidence of 0.05%, five seedlings were inoculated at 4.2 m intervals within each bed. Likewise, for initial disease incidences of 0.1, 0.2, and 0.5%, 10, 20, and 50 seedlings were inoculated at about 2.1, 1.1, and 0.4 m intervals within beds, respectively. Inoculated seedlings were labeled with a colored tag for easy recognition.

Each bed was clipped eight times with a rotary mower when seedlings were 18, 19, 20, 21, 22, 23, 24, and 25 cm tall. The clipping practice followed a preset pattern of mowing, which moved from the uninoculated controls to higher levels of initial incidence and from the first to the third replicate within the same treatment. Thus, the mower always moved from low to high incidence. The mower was surface-sterilized with 0.8% dimethyl benzyl ammonium chloride (Roccal II) solution after each clipping date.

Epiphytic populations of Rif⁺ *C. m. michiganensis* were assayed after each clipping date from five seedlings selected from various locations in each bed. Populations were determined by dilution plate methods described below. 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 consisted of four rows with 25 and 30 transplants per row in 1988 and 1989, respectively. Incidence of systemic infection was measured as the percentage of diseased plants per plot based on visual observations at 7-day intervals after transplanting. Disease ratings were terminated on 2 October 1988 and 28 September 1989, 1 wk before harvest.

Seedling harvest practices. The harvest and shipping practices used by the tomato transplant industry were simulated in the greenhouse. Tomatoes (cv. Heinz 1810) were direct seeded in a sterilized mixture composed of equal proportions of soil, sand, peat, and vermiculite in 35- × 50-cm flats on 6 April 1988 and 7 April 1989. Each flat consisted of 80–90 seedlings. Flats were kept in a greenhouse (25 C day, 20 C night). Granular potash (FS Special, Growmark Inc. Bloomington, IL.) was applied every 2 wk. Three flats of direct-seeded tomato seedlings were inoculated

on 4 May 1988 and 10 May 1989 by the method described by Thyr (25). Forty-eight flats of healthy seedlings were placed in separate greenhouses from the inoculated seedlings. When seedlings reached about 25 cm, healthy seedlings were removed from flats by hand, and mixed with asymptomatic, inoculated seedlings of the same age according to the four treatments described below. Each mixture contained a total of 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 roots.

Four treatments were evaluated. Three treatments were mixtures of healthy and asymptomatic, inoculated seedlings with contamination rates of 1, 5, and 10% asymptomatic, infected seedlings. One treatment was a healthy control. Treatments were arranged in a randomized complete block design with three replications. A total of 200 seedlings per experimental unit were stored overnight in a cold room at 4 C, and shipped to the University of Illinois Pomology Farm. Before transplanting, four seedlings from each experimental unit were sampled for populations of Rif⁺ *C. m. michiganensis*, which were determined by dilution plate methods described below. The remaining seedlings were transplanted in a production field on 26 May 1988 and 24 May 1989. There was one plot for each experimental unit with four rows and 49 transplants per row. Transplants were spaced 30 cm apart within rows and 90 cm between rows. Incidence of systemic infection was measured as the percentage of diseased plants per plot by visual observations at 7-day intervals after transplanting. Disease ratings were terminated on 28 August 1988 and 24 August 1989, 1 wk before harvest.

Dilution plate methods to detect populations of Rif⁺ *C. m. michiganensis*. Populations of Rif⁺ *C. m. michiganensis* were measured from samples described previously to evaluate when bacteria began to spread in the seedling beds and to determine the size of populations of Rif⁺ *C. m. michiganensis* associated with contaminated transplants. One gram of plant tissue was selected from each experimental unit and submerged in 20 ml of sterile PBS in a 125-ml Erlenmeyer flask. Chlorothalonil (0.1%; 0.5 g a.i./L, w/v) was added to the buffer to inhibit growth of fungal contaminants (10). Plant tissues were washed for 1 h at room temperature on a reciprocating shaker. A modified CNS medium (13) (minus lithium chloride and polymycin B sulfate) amended with rifampin (50 mg/L) was used to recover epiphytic populations of Rif⁺ *C. m. michiganensis* by plating the resulting wash solution in 10-fold serial dilutions on the modified CNS medium. Plates were incubated at 25 C and colonies were counted after 5–7 days.

Statistical analyses. Incidence of systemically infected plants measured at each rating and epiphytic populations of Rif⁺ *C. m. michiganensis* recovered from asymptomatic transplants in the clipping and seedling harvest practices were analyzed by ANOVA. Incidence of systemically infected plants in production fields 1 wk before harvest was regressed on initial disease incidence from the clipping practice experiments, contamination rates from the seedling harvest practice experiments, and epiphytic populations of Rif⁺ *C. m. michiganensis* recovered from contaminated transplants in the seedling harvest practice experiments. *F* statistics ($P < 0.05$) were used to test the significance of the regression models and independent variables. Coefficients of determination (r^2) were calculated to determine the variation in incidence of systemic infection explained by the independent variables.

RESULTS

Clipping practices. Clipping tomato seedlings in transplant beds spread Rif⁺ *C. m. michiganensis* from inoculated seedlings to healthy seedlings. Symptoms on inoculated seedlings were first observed in transplant beds 26 and 17 days after inoculation in 1988 and 1989, respectively. Epiphytic populations of Rif⁺ *C. m. michiganensis* were first detected on clipped, uninoculated seedlings 21 and 18 days after inoculation, which were 13 and 9 days after the first clipping in 1988 and 1989, respectively. Populations of Rif⁺ *C. m. michiganensis* on clipped seedlings

ranged from 0 to about 10^7 cfu/g fresh weight throughout the clipping period (Table 1). In 1988, epiphytic populations were higher from treatments with a high incidence of initial disease than from treatments with a low incidence of initial disease. In 1989, epiphytic populations of Rif^+ *C. m. michiganensis* were detected at levels from about 10^2 to 10^7 when initial disease incidence was 0.2 and 0.5%. Populations fluctuated from 0 to about 10^5 for treatments with lower initial disease incidence (Table 1). In the control treatment (0% initial disease incidence), Rif^+ *C. m. michiganensis* was not recovered, which indicated that dissemination of Rif^+ *C. m. michiganensis* as aerosols or in debris from clipping was negligible between experimental units.

Incidence of systemically infected plants in production fields was related to initial incidence of infected seedlings in transplant

TABLE 1. Epiphytic populations of rifampin-resistant *Clavibacter michiganensis* subsp. *michiganensis* on tomato seedlings from beds that were clipped and had different levels of initial disease incidence

| Year | Day after inoculation | Log colony-forming units per gram fresh weight at initial disease incidence (%)[a] | | | | | |
		0.00	0.01	0.05	0.10	0.20	0.50
1988							
	21	0.0	0.0	0.0	1.5	ND[b]	1.9
	26	0.0	0.0	2.3	3.0	ND	4.1
	29	0.0	0.0	2.6	3.2	ND	4.6
1989							
	18	0.0	4.9	1.8	0.0	5.6	3.5
	23	0.0	5.2	2.8	3.7	6.0	6.7
	25	0.0	4.1	0.0	4.1	5.0	5.2

[a] Five clipped seedlings per bed were collected and pooled as a sample per experimental unit. Colony-forming units were determined by dilution plating (see text).
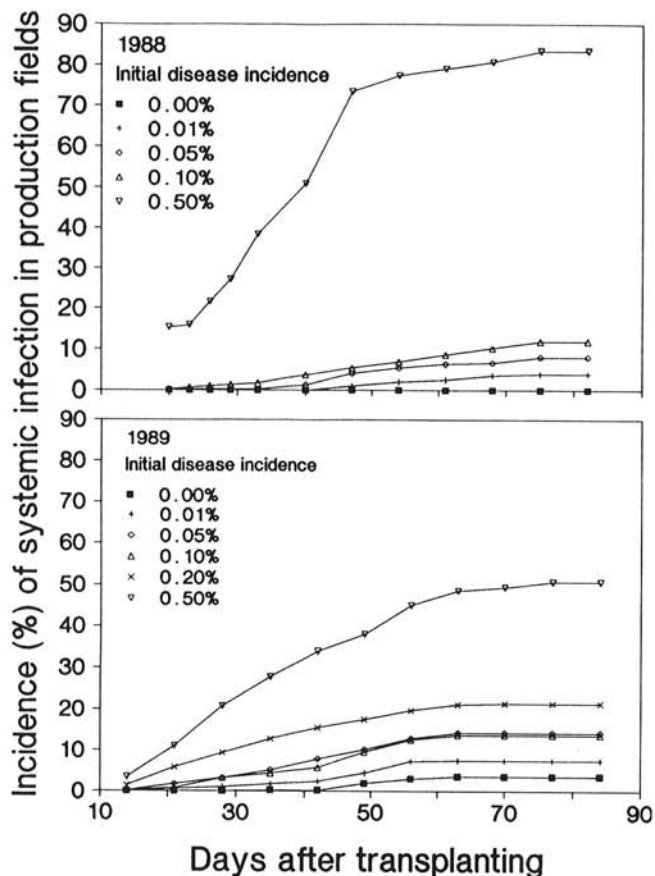[b] Not determined.

beds (Figs. 1 and 2). Symptoms of systemic infection were first observed about 20 days after transplanting in 1988, which was about 40 days after the first clipping. Systemically infected plants were first observed about 14 days after transplanting in 1989, which was about 31 days after the initial clipping (Fig. 1). Disease progress curves from both years showed a similar trend. Incidence of systemically infected plants progressed slowly, with a steady rate of increase during the period from about 20–60 days after transplanting. Maximum disease incidence occurred about 65–75 days after transplanting (Fig. 1).

Regressions of incidence of systemically infected plants 1 wk before harvest on initial incidence of infected seedlings before clipping were significant in both years (Fig. 2). These relationships were explained by linear regression models, $Y = 1.61 + 97.49(X) + 132.04(X)^2$ in 1988 ($r^2 = 0.99$, $P < 0.01$) and $Y = 5.87 + 88.47(X)$ in 1989 ($r^2 = 0.88$, $P < 0.01$), in which X was the incidence (%) of infected seedlings before clipping and Y was the incidence (%) of systemically infected plants 1 wk before harvest. Thus, incidence of systemically infected plants in production fields increased about 9–11% for each 0.1% increase in initial incidence of infected seedlings before clipping in transplant beds.

Seedling harvest practices. When healthy and diseased seedlings were harvested, mixed, shaken, and transplanted, these production practices spread Rif^+ *C. m. michiganensis* from diseased to healthy seedlings. Populations of Rif^+ *C. m. michiganensis* recovered from bundles of transplants were affected significantly by seedling harvest practices (Fig. 3). Populations of Rif^+ *C. m. michiganensis* on bundles of transplants were approximately 40, 2.8×10^4, and 2.0×10^5 cfu/g fresh weight in 1988 for the 1, 5, and 10% contamination rates, respectively (Fig. 3). In 1989, populations of Rif^+ *C. m. michiganensis* were about 9.1×10^3 for the three rates of contamination and zero for the control treatment (Fig. 3). Although a population of 2 cfu of Rif^+ *C. m. michiganensis* per gram fresh weight was recovered from two of 12 samples of the control treatment in 1988, bacterial canker was not observed
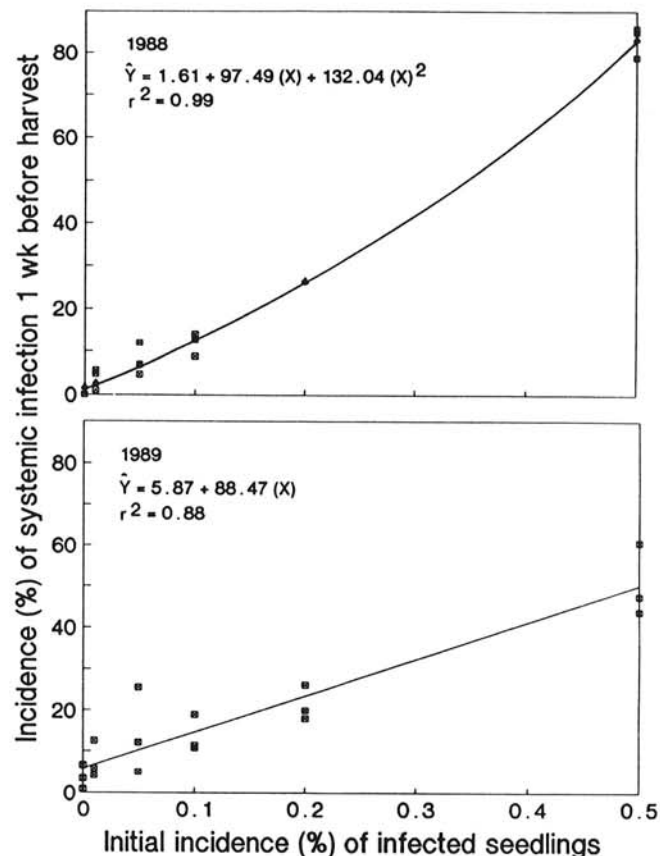

Fig. 1. Progress of bacterial canker in production fields of tomatoes after transplanting seedlings from transplant beds that had 0, 0.01, 0.05, 0.1, 0.2, and 0.5% initial incidence of infected seedlings before clipping.

Fig. 2. Relationships between incidence of systemic infection of bacterial canker in production fields of tomatoes 1 wk before harvest and initial incidence of infected seedlings in transplant beds before clipping.

when these plants were transplanted. When incidence of systemically infected plants 1 wk before harvest was regressed on populations of Rif$^+$ *C. m. michiganensis* recovered from contaminated transplants, larger populations of Rif$^+$ *C. m. michiganensis* resulted in significantly higher incidences of systemic infection (Fig. 4). The relationships were explained by a positive linear model, although there was considerable variation in both years.

Symptoms of systemic infection were first observed in the production fields 42 and 35 days after transplanting in 1988 and 1989, respectively (Fig. 5). Disease progress was similar in both years with incidence of systemic infection increasing during the period from about 35 to 85 days after transplanting (Fig. 5). Incidence of systemically infected plants was consistently higher throughout the season for the treatments with higher contamination rates. Contamination rates of 1, 5, and 10% diseased plants per bundle of transplants resulted in approximately 11, 32, and 57%, and 7, 19, and 33% systemically infected plants 1 wk before harvest in production fields in 1988 and 1989, respectively (Fig. 6). Regressions of incidence of systemic infection 1 wk before harvest on the contamination rate were significant in both years. The relationships were described best by positive linear models with slopes of 5.10 and 2.78 in 1988 and 1989, respectively (Fig. 6). Thus, incidence of systemically infected plants in production fields increased about 3–5% for each 1% increase in incidence of diseased plants in bundles of transplants.

DISCUSSION

Clipping tomato seedlings and mixing infected seedlings with healthy seedlings during harvest of transplants spread Rif$^+$ *C. m. michiganensis* from infected to healthy seedlings. Incidence

Fig. 4. Relationships between incidence of systemic infection of bacterial canker in production fields of tomatoes 1 wk before harvest and epiphytic population of rifampin-resistant *Clavibacter michiganensis* subsp. *michiganensis* recovered from contaminated transplants.
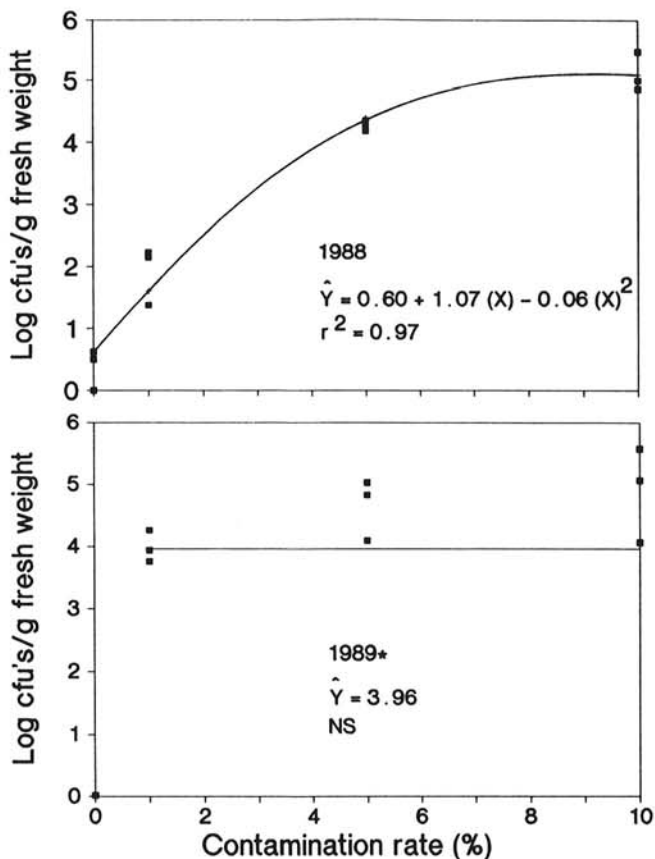
Fig. 3. Relationships between epiphytic populations of rifampin-resistant *Clavibacter michiganensis* subsp. *michiganensis* recovered from tomato seedlings before transplanting and percentages of asymptomatic, inoculated seedlings mixed with healthy seedlings (contamination rates) during seedling harvest practices. *The regression for 1989 did not include the 0% treatment.
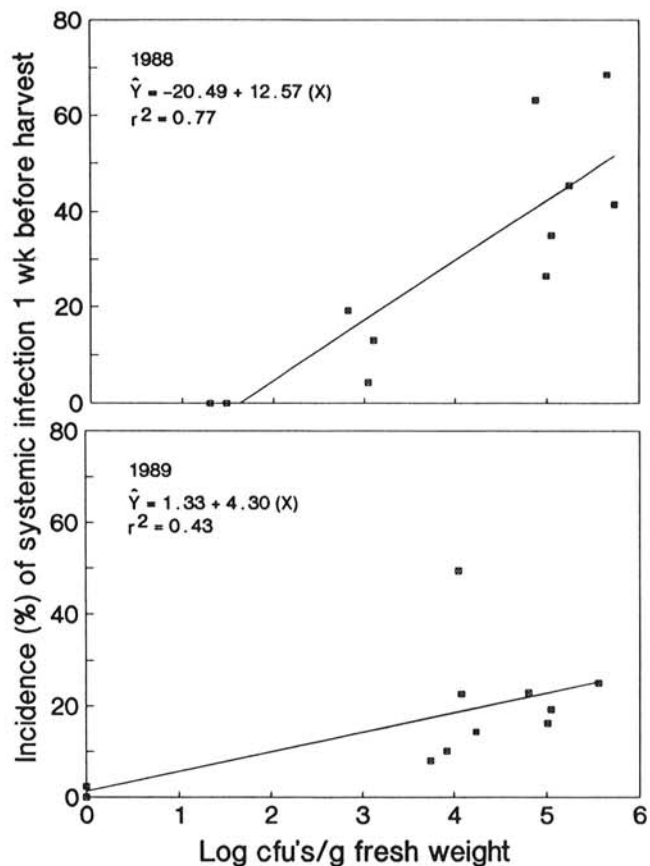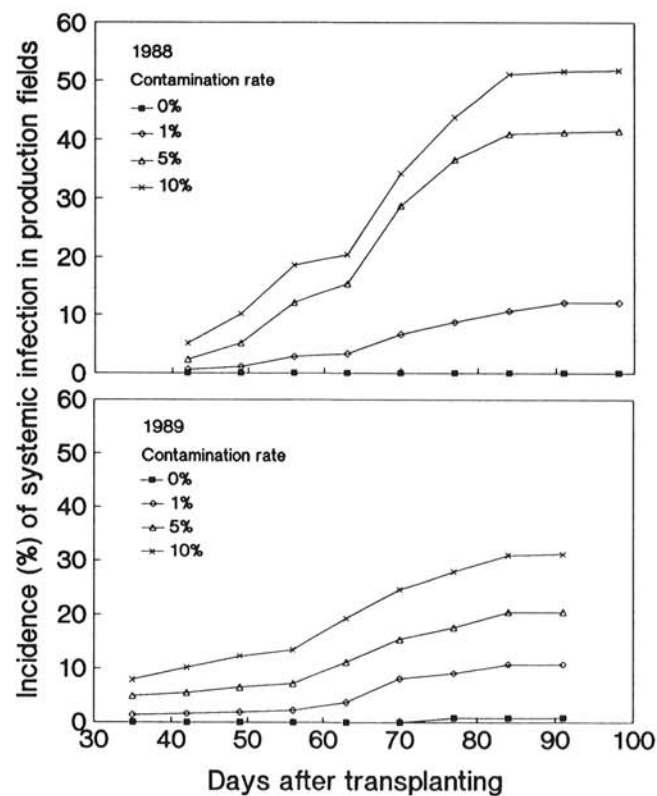
Fig. 5. Progress of bacterial canker in production fields of tomatoes after mixing asymptomatic, inoculated and healthy seedlings at contamination rates of 0, 1, 5, and 10% during seedling harvest practices.

of systemically infected plants in production fields 1 wk before harvest increased about 10% for each 0.1% increase in incidence of infected seedlings in transplant beds before clipping. Incidence of systemically infected plants in production fields 1 wk before harvest increased about 3–5% for each 1% increase in the proportion of diseased seedlings mixed with healthy seedlings during harvest of seedlings. Thus, these two production practices are extremely important in the spread and epidemic development of bacterial canker from transplants.

Clipping of tomato seedlings in direct-seeded beds and moving plants during thinning and transplanting have been reported to disseminate several bacterial and viral diseases of tomato (1,6,18,19). Greenhouse-grown transplants are not clipped, but clipping is considered essential for field-grown transplants and has been a routine practice in the production of these seedlings. Therefore, control of bacterial canker in the commercial production of transplants in the field has relied on certified seed (7,21). The levels of initial disease incidence in our clipping experiments were intended to simulate various rates of seed transmission. These treatments may not have adequately simulated seed transmission if plants that are systemically infected by seed transmission differ from those infected by our inoculation procedure for such factors as: type of symptoms, length of incubation period, production of secondary inoculum, etc. However, rates of seed transmission as low as 0.01–0.05% (one to five seeds per 10,000) may result in an epidemic of bacterial canker if the spread of *C. m. michiganensis* in transplant beds is similar from plants infected by seed and from plants infected by our inoculation procedure. In our studies, an initial incidence of 0.05% resulted in 7–10% infected plants following our clipping practices, and a contamination rate of 7–10% during the harvest of seedlings resulted in 24–57% systemically infected plants in production fields 1 wk before harvest. Thus, the sensitivity of all of the current methods used to detect *C. m. michiganensis* in seed lots (7,26) may be inadequate if the spread of *C. m. michiganensis* in trans-

plant beds from seedlings that are systemically infected from seed is similar to the results observed in our study.

Because of the long incubation period, bacterial canker is extremely difficult or impossible to detect by visual inspections of seedling beds. In our clipping study, epiphytic populations of Rif+ *C. m. michiganensis* were detected on seedlings 9–13 days after the first clipping, however symptoms did not appear on these seedlings until 14–20 days after transplanting or about 31–40 days after the first clipping. In the seedling harvest experiment, symptoms of systemic infection were first observed 35–42 days after transplanting "contaminated" seedlings. The difference in the time of appearance of symptoms in the clipping and seedling harvest experiments probably was due to age of plants, route of inoculation, and/or concentration of inoculum. Infected plants were younger in the clipping experiment than in the seedling harvest experiment, which may have affected length of incubation period. Also, appearance of symptoms on stem-inoculated plants from clipping may have been faster than those inoculated in contaminated bundles. Strider (22) also observed differences in appearance of symptoms based on methods of inoculation. Similarly, plants inoculated by the clipping practices may have been exposed to a higher concentration of inoculum than plants inoculated during the harvest of seedlings, and inoculum concentration may have affected the length of incubation period.

Presently, certification of seedlings is done by detection of symptomatic transplants (9). Detection of *C. m. michiganensis* from a large population of asymptomatic transplants is a challenging problem, because the test must be sensitive and specific enough to distinguish low populations of *C. m. michiganensis* from the background of a diverse microflora (9). By using Rif+ *C. m. michiganensis* strains for inoculation and by tagging all inoculated plants in the seedling beds, we were able to trace Rif+ *C. m. michiganensis* in the field without the detection problems described by Gitaitis et al (9). We attempted to recover Rif+ *C. m. michiganensis* and revertant wild type strains from clipped seedlings by using non-amended CNS medium to compare the proportion of Rif+ *C. m. michiganensis* and revertant wild type strains. However, these assays were unsuccessful due to background contamination by foliar microflora. This illustrates the dilemma faced by plant inspectors who must identify wild type *C. m. michiganensis* without a suitable selective media. Improved methods for detecting bacterial canker in asymptomatic seedlings are essential.

The method and timing of sampling of transplant fields also presents a difficult problem for certification of seedlings. Our results suggest that epiphytic populations of Rif+ *C. m. michiganensis* can be detected on asymptomatic seedlings 9–13 days after clipping, but determination of proper sampling methods still needs further study. For example, in 1989, populations of 10^4–10^5 cfu/g fresh weight of Rif+ *C. m. michiganensis* were recovered from the 0.01% treatment. Relatively high populations of Rif+ *C. m. michiganensis* may have been recovered from this relatively low incidence of initial disease because samples were consistently collected from plants adjacent to inoculated plants, thus illustrating the sampling problem in detection of *C. m. michiganensis* and in establishing an adequate tolerance level for seedling certification.

Our results also indicate that epiphytic populations of Rif+ *C. m. michiganensis* serve as sources of inocula for spreading bacterial canker during seedling harvest and transplant practices. Higher incidences of systemically infected plants were observed in treatments for which epiphytic populations of Rif+ *C. m. michiganensis* were higher. Thus, contamination of tomato transplants by *C. m. michiganensis* during shipping is an important consideration.

In 1989, systemically infected plants were observed in the control treatments of clipping and seedling harvest experiments. It is not clear whether these systemic infections were the result of external secondary infection of the foliage or incomplete surface sterilization of the mower used to clip seedlings. Another possibility could be the secondary dispersal of Rif+ *C. m. michiganensis* from adjacent transplant beds through aerosols or wind-splashing
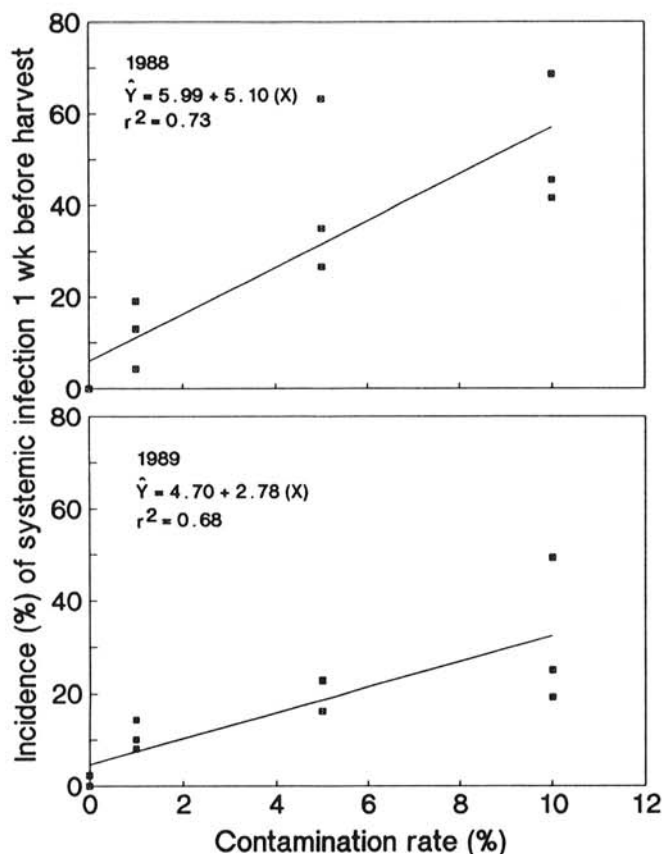

Fig. 6. Relationships between incidence of systemic infection of bacterial canker in production fields of tomatoes 1 wk before harvest and contamination rates during seedling harvest practices.

rain after clipping, although Rif⁺ *C. m. michiganensis* was not recovered as epiphytes in the control plots. Dry weather in 1988 may have decreased secondary dispersal in transplant beds, and thus, higher epiphytic populations of Rif⁺ *C. m. michiganensis* were recovered from treatments with a higher incidence of initial disease.

Many of the phytopathogenic corynebacteria are nonmotile wound pathogens (27). They can be mechanically transmitted by cultural practices and produce systemic, rather than localized, infections (27). For example, *Clavibacter michiganensis* subsp. *sepedonicus*, the causal agent of potato ring rot disease is spread by cutting infected seed tubers or by using spikes to place individual seed pieces into furrows (4). Likewise, *C. m. insidiosus*, the causal agent of bacterial wilt of alfalfa, infects plants through wounds created during harvest (24). The complex symptoms of bacterial canker of tomato can be differentiated into systemic and surface infections (21). Although wounding was reported to be unnecessary for infection (16,23), it provides a logical avenue for entrance of *C. m. michiganensis* to produce systemic infections (1,21). Obviously, wounds are created by the clipping and seedling harvest procedures. When epiphytic populations of *C. m. michiganensis* are high, these cultural practices are extremely favorable in establishing systemic infection. Likewise, a single systemically infected seedling in a transplant bed of thousands may provide sufficient inoculum to transmit *C. m. michiganensis* to many other seedlings when these seedlings are wounded by clipping. Although the natural occurrence of bacterial canker of tomato from local sources of inoculum is not fully understood, this disease can be increased to epidemic proportions by the cultural practices used in the production of transplants provided that low levels of initial inoculum occur and conditions are conducive. As in 1984, bacterial canker of tomato can be shipped from southern states despite plant certification procedures, because the incubation period is long, and adequate methods to detect low populations of *C. m. michiganensis* in seedling beds are lacking.

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