Effect of Secondary Spread of *Clavibacter michiganensis* subsp. *michiganensis* on Yield of Northern Processing Tomatoes

MARK D. RICKER, Former Research Associate, Campbell Institute for Research and Technology, Campbell Soup Company, Napoleon, OH 43545, and RICHARD M. RIEDEL, Professor, Department of Plant Pathology, The Ohio State University, Columbus 43210

ABSTRACT

Ricker, M. D., and Riedel, R. M. 1993. Effect of secondary spread of *Clavibacter michiganensis* subsp. *michiganensis* on yield of northern processing tomatoes. Plant Dis. 77:364-366.

Test plots of the determinate tomato (*Lycopersicon esculentum* Mill.) cultivar Easy Winner were established in a field near Fremont, OH, in 1986–1988 to study the effects of dispersal of the bacterial canker pathogen on yield. Leaf symptoms indicative of secondary spread of *Clavibacter michiganensis* subsp. *michiganensis* were widespread in 1986 and 1987; in 1988, a single area (about 6 m in diameter) of symptomatic plants with marginal scorching of leaflets and fruit spotting was present. The bacterium systemically infected scattered plants throughout the field, not just in the scorched area, but caused little or no wilting. Yields of inoculated plants were greatly reduced, but compensation by adjacent plants allowed plots with inoculated plants to yield as much fruit as the control plots. A simple graphing procedure designed to relate average plant yield to distance from the closest inoculated plant was tested. Plants adjacent to inoculated ones yielded on average over the 3 yr 12% more than the mean of all noninoculated plants.

Bacterial canker is an important disease of tomato (*Lycopersicon esculentum* Mill.) that occurs in most tomato-producing regions of the world (11). The disease, caused by *Clavibacter michiganensis* subsp. *michiganensis* (Smith) Davis et al, occurs sporadically in the midwestern United States. Bacterial canker-induced mortality of young, southern-grown transplants was as high as 30% in at least one commercial processing field in northwestern Ohio (M. D. Ricker, *unpublished*). No commercial tomato cultivars with resistance to bacterial canker are currently grown in the Midwest.

We have observed that in Ohio, systemically infected transplants usually die soon after they are planted or, if they survive, produce stunted plants that bear no marketable fruit. Large numbers of systemically infected transplants in a field can reduce plant populations (stand) and lower yields. This effect of bacterial canker on yield may not be apparent before harvest because adjacent healthy plants frequently grow over and hide dead or stunted plants. Symptoms that appear later in the season on mature plants are more conspicuous and cause more concern to growers, but late infections on mature processing tomatoes appear to be less detrimental to yield. Infestation of plots with *C. m. michiganensis* lowered fruit quality but not yield in one study (12). In two other studies, yield reductions depended on age of plants at the time they became systemically infected (2,3).

Studies with greenhouse tomatoes indicate that *C. m. michiganensis* is spread readily (9). The rate and extent of dissemination of the bacterium in commercial tomato fields are less well documented, in part because of the difficulty of distinguishing secondary symptoms from late-appearing primary infections. Likewise, the economic impact of secondary infections in tomato fields is unclear. Information of this type could be helpful in deciding whether fields that were planted with large numbers of infected transplants should be destroyed and replanted.

Numerous models describing the spatial occurrence of diseased plants have been developed (1,8). Their use, however, depends on the existence of a rapid and proven method for detecting either the pathogen or diseased plants. Such a method was not available for bacterial canker when this study was conducted. The models also provide little information on the effects of infection on plant yield. The objectives of this study were to determine the extent of spread of the pathogen in a field of processing tomatoes maintained with typical grower practices and to assess the impact of such spread on yield.

Present address of first author: Heinz USA, Agricultural Research Department, Bowling Green, OH 43402.

Use of trade names does not imply endorsement by The Ohio State University or by Campbell Soup Company or any of its subsidiaries of the products named or criticism of similar ones not mentioned.

Reprint requests to R. M. Riedel.

Accepted for publication 5 November 1992.

MATERIAL AND METHODS

Transplant production. Tomato seeds of the cultivar Easy Winner (Campbell Soup Co., Napoleon, OH) were sown in sterile soil (1:1 mixture of loam and Promix) in greenhouse beds. Seedlings were watered and fertilized as needed. When seedlings were 6 wk old, the second-oldest leaf on each plant to be inoculated was snipped off with scissors dipped in a turbid aqueous suspension of *C. m. michiganensis*. The inoculum was produced on yeast dextrose-calcium carbonate agar inoculated with a single strain (CmT10 from E. Echandi, North Carolina State University, Raleigh) obtained from a tomato fruit in North Carolina in 1984 and previously shown to be pathogenic. The culture used in this study had been stored under mineral oil. Inoculated plants in bundles of 20 were planted into flats of soil and isolated from the remaining seedlings.

Plot establishment. Plots were established in 1986–1988 in an isolated part of the Vegetable Crops Branch of The Ohio State University in Fremont. The field had not previously been cropped to tomatoes. Noninoculated transplants grown for 8 wk in the greenhouse were set 75 to the row (30 cm apart) in each of 24 rows. Each row of 75 plants was a plot. Rows were 1.5 m apart and were oriented north-south, and each transplant received a numbered stake. Plots were bordered with buffer rows of noninoculated plants.

Four to 10 days after transplanting, zero to five plants in each test row were replaced with inoculated seedlings. The positions of the inoculated plants within each row and the rows receiving each number of plants were randomly selected. Four complete blocks were included in each year's experiment.

To harvest the fruit, each plant's main stem was severed, and the plant was shaken upside-down in a large plastic bag. The stems remained on the surface until fall bedding.

Plots were fertilized with 900 kg of 0-26-26 per hectare plowed down and 225 kg of each of 34-0-0 and 0-0-60 per hectare before transplanting. Napropamide (2.2 kg a.i./ha) and metribuzin (0.22 kg a.i./ha) were applied for weed control. Chlorothalonil (2.25 kg a.i./ha; Bravo 720) and mancozeb (2.7 kg a.i./ha; Dithane F45) were applied as needed for control of fungal diseases. Fungicides were applied with an R&D CO_2 tractor-mounted, one-row boom sprayer with five Delavan HC-8 tips. Sprays were applied at 483 kPa in 562 L of water per hectare. Machinery was steam-cleaned before entering the plots. Rows with no inoculated transplants were sprayed first, followed by those with one to five inoculated plants in increasing order. The same pattern of travel was always followed.

Twelve healthy-looking top leaves collected from the entire block in August 1986 and August 1987 were sampled for the presence of *C. m. michiganensis.* The leaflets were ground in phosphate buffer (pH 7.5, 0.02 M), and portions of the extract were applied to plates of SCM medium (6) and used to inoculate tomato seedlings. No attempt was made to distinguish infection from infestation or to quantify populations. Stems with possible discoloration were also sampled every year. Stems of 20 healthy plants from border rows were surface-disinfested and sampled in 1987.

Yield data. Fresh weight of all fruit from each plant at harvest was used to compute plot yields and was subjected to analysis of variance. Dunnett's procedure (10) was used to compare treatment means to the control (plots with no inoculated plants). Expected yields of rows with inoculated transplants were computed from the average yield of rows without inoculated transplants, as if the inoculated transplants died without producing fruit, the adjacent plants did not fill in with compensatory growth, and secondary spread of the bacterium did not affect plant yields.

Yield data were also graphed against distance from inoculated plants to assess the effect of inoculated plants on yield of neighboring plants. We assumed first that the farther a plant is from a source of inoculum, the less likely it is to become infected, second that yield loss is inversely proportional to plant age at the time of infection, and third that plants farther from a source of inoculum are older when they become infected and therefore yield more than plants closer to an inoculum source. We computed the distance from each plant to the nearest inoculated one in the direction of choice. Directions and distances were trigonometrically computed from each plant's position and row number. Yields of all plants within the same distance-direction class were averaged. Average plant yield was then plotted versus distance north, south, east, west, down-row, within-row, or in any direction. "Down-row" was the distance the tractor traveled after passing the last inoculated plant. Each curve was ended when points representing 95% of the data had been plotted or when the mean yield was from only one plant.

RESULTS

Field studies. Growing conditions during the three growing seasons (1986–1988) were diverse. Eleven percent of the transplants died in 1986 as a result of flooding. Plot yields were estimated from the remaining plants. In 1987, the spring was wet and the summer dry. In 1988, plots were overhead-irrigated with 1.25 cm of water on 10, 13, and 17 June and 1 July because of exceptionally dry growing conditions.

Secondary spread of *C. m. michiganensis* was widespread in each year. The bacterium was found on every leaflet sampled in August, even symptomless ones. In 1987, 11 of the 20 plant stems collected from border rows were systemically infected, even though none showed any symptoms. Two wilted plants were observed in 1987 and were found to be infected with *C. m. michiganensis* and *Pseudomonas solanacearum* (Smith) Smith. In addition, two unwilted plants with slight vascular discoloration found in plot rows also contained *C. m. michiganensis.*

Other evidence of secondary spread of the pathogen appeared only near the end of each growing season. Marginal scorching of foliage became evident in essentially every plant in late July of 1986. It was less severe the following year and in 1988 was confined to a circular area about 6 m in diameter. No bird's-eye spotting of fruit was observed until 1988, when it was found in the one area of plants with scorched foliage. Plants adjacent to inoculated ones appeared as healthy as ones farther away.

Actual treatment yields were less than "expected" only four times during 1986–1988 (Table 1) and by only 0.1–0.7 metric tons per hectare. None of the plots with inoculated transplants yielded statistically less fruit than the control. Yields of Easy Winner in commercial fields in this area were approximately 58.2 and 51.4 t/ha in 1986 and 1987, respectively.

Distance-yield graphs. Yields of inoculated transplants were depressed by more than 90% in 1986 and 1987 but by only 63% in 1988 (Table 2). Because of the frequent irrigation in 1988, inoculated plants survived longer and had higher yields. Fruit weight from plants adjacent to inoculated ones was 20 and 14% above average in 1987 and 1988, respectively, but only 2% above average in 1986. Excessive rainfall and flooding soon after transplanting in 1986 stunted vegetative growth well into the growing season.

Figure 1 presents yields plotted against distance down-row from inoculated transplants. Graphs of the other directions are not shown. In most of them, the second point is the highest, illustrating the above-average yields of plants adjacent to inoculated ones, and the remaining points fall close to the mean. Peaks were not evident for east or west graphs. The peak in 1987 is greater than that in 1986, as predicted by the data in Table 2. The 1988 peak is moderate, but variation around the mean appears slightly greater than in 1986 or 1987.

DISCUSSION

These results demonstrate the large losses that bacterial canker can cause in processing tomatoes if plants are infected before transplanting. Yields of inoculated plants were reduced 63–93%, depending on environmental conditions. No evidence was collected in 3 yr of field studies to suggest that secondary spread of *C. m. michiganensis* in a northern field caused any loss in tonnage of processing tomatoes. The bacterium spread rapidly under field conditions in Ohio, causing marginal leaf scorching in all 3 yr and bird's-eye fruit spotting in 1988. Secondary spread resulted in some systemically infected plants but caused little or no wilting.

None of the plots with artificially inoculated plants yielded statistically less fruit than the control plots. This suggests

Table 1. Actual and expected fruit yields[a] (t/ha) of 75-plant plots of Easy Winner tomatoes with zero to five transplants per plot inoculated with *Clavibacter michiganensis* subsp. *michiganensis* (1986–1988)

	1986		1987		1988	
IT[b]	Actual	Expected	Actual	Expected	Actual	Expected
0	56.0	56.0	52.5	52.5	72.0	72.0
1	59.4	55.3	55.1	51.8	70.9	71.0
2	63.2	54.5	52.3	51.1	69.9	70.1
3	54.8	53.8	51.1	50.4	69.6	69.1
4	54.7	53.0	51.1	49.7	71.9	68.2
5	52.1	52.3	50.7	49.0	...	...

[a]Expected yields were calculated by multiplying the yield of the control by the percentage of noninoculated transplants. None of the treatment means differ from the control, according to Dunnett's procedure ($P = 0.05$, two-tailed test).
[b]Number of inoculated transplants per plot.

Table 2. Fruit yields (kilograms per plant) of Easy Winner tomato plants inoculated with *Clavibacter michiganensis* subsp. *michiganensis*, grown next to inoculated plants, or grown in the rest of the field (1986–1988)

Position	1986	1987	1988
Inoculated transplants	0.2	0.2	1.2
Noninoculated transplants adjacent to an inoculated one	2.8	3.0	3.8
All noninoculated transplants	2.7	2.5	3.3

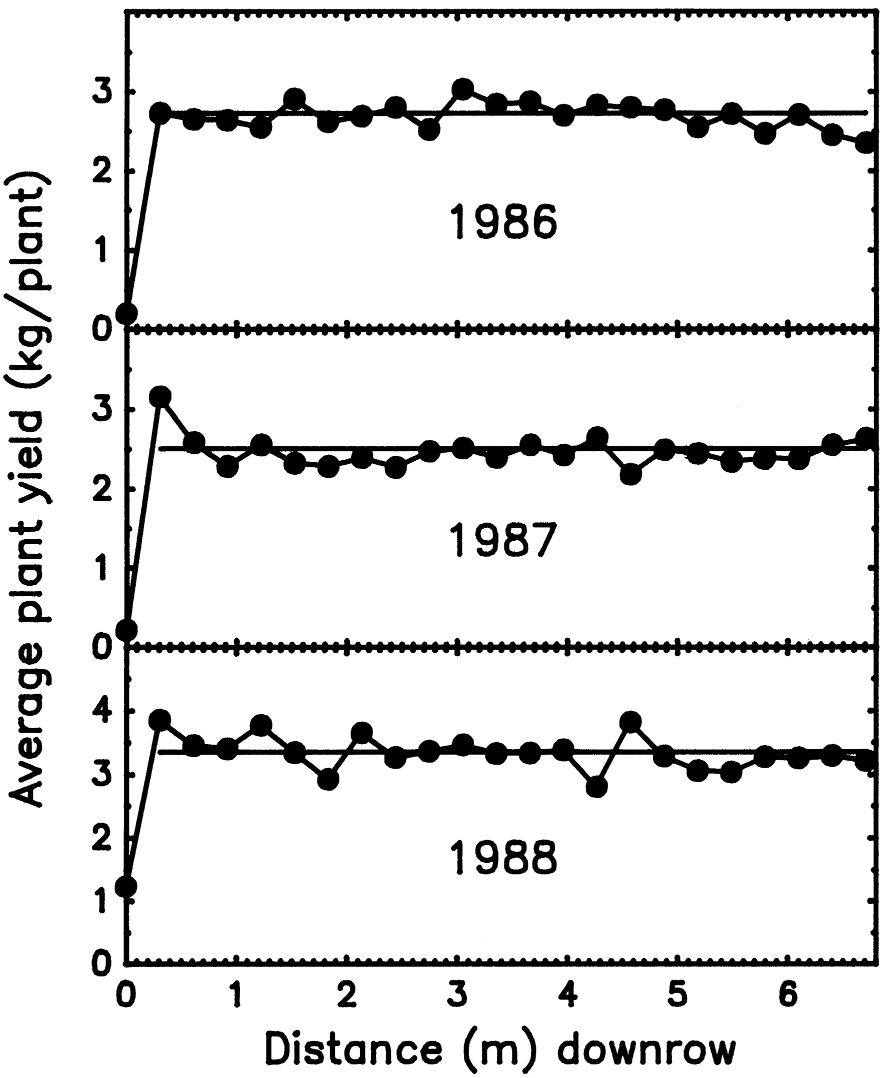

Fig. 1. Average fruit yield (kilograms per plant) of Easy Winner tomatoes plotted against distance down-row from the nearest plant inoculated with *Clavibacter michiganensis* subsp. *michiganensis* in field trials in 1986–1988. The horizontal line in each graph is the average yield of all noninoculated plants for that trial.

that plants adjacent to the inoculated ones were compensating with increased growth. Interpretation of these data in distance-yield graphs supported this conclusion. We had expected plants adjacent to inoculated transplants to yield poorly because of rapid infection with the bacterium; however, their yields were equal to or greater than the average yields for plants at all other representative distances. Because inoculated transplants either died during the season or were severely stunted, decreased competition for light, moisture, and nutrients probably accounted for the increased yields of the adjacent surviving plants.

These results also suggest that serious losses can occur in commercial fields when significant numbers of young plants are systemically infected early in the season. *C. m. michiganensis* can overwinter in debris in Ohio soil (4), and plants set into infested soil could become diseased, as was reported recently in Iowa (7). We did not detect fruit losses in 1987 or 1988 despite a moderate problem with volunteer tomato plants. Serious yield losses in processing tomatoes probably occur when transplants are infected in southern transplant production fields (13), and clipping practices in these transplant production fields probably increase the number of systemically infected transplants (5).

Where the appearance of tomato fruits is important in determining marketability of the crop, as for example with fresh-market tomatoes, or where fruit spotting can interfere with processing, as with whole-pack tomatoes, secondary spread of *C. m. michiganensis* may result in significant losses. Secondary spread could also cause losses in fields intended for seed production, because some seeds from diseased plants could carry the bacterium (14).

ACKNOWLEDGMENTS

We thank C. Willer, D. McCartney, T. Shambaugh, D. Biederstedt, and K. Hetrick for their assistance and E. Echandi for the culture of *C. m. michiganensis* (originally designated CmT10) used in this study.

LITERATURE CITED

1. Campbell, C. L., and Noe, J. P. 1985. The spatial analysis of soilborne pathogens and root diseases. Annu. Rev. Phytopathol. 23:129-148.
2. Dullahide, S. R., Moffet, M. L., Heaton, J. B., and Giles, J. 1983. Effect of time of inoculation of *Corynebacterium michiganense* subsp. michiganense on yield of trellised tomatoes. APP, Australas. Plant Pathol. 12:15-16.
3. Emmatty, D. A., and John, C. A. 1973. Comparison of yield loss to bacterial canker of tomato in a resistant and a susceptible variety. Plant Dis. Rep. 57:787-788.
4. Farley, J. D. 1971. Recovery of *Corynebacterium michiganense* from overwintered tomato stems by the excised-petiole inoculation method. Plant Dis. Rep. 55:654-656.
5. Farley, J. D., and Miller, T. D. 1973. Spread and control of bacterial canker in tomato transplants during clipping. Ohio Agric. Res. Dev. Cent. Res. Summ. 65:23-25.
6. Fatmi, M., and Schaad, N. W. 1988. Semiselective agar medium for isolation of *Clavibacter michiganense* subsp. michiganense from tomato seed. Phytopathology 78:121-126.
7. Gleason, M. L., Braun, E. J., Carlton, W. M., and Peterson, R. H. 1991. Survival and dissemination of *Clavibacter michiganensis* subsp. *michiganensis* in tomatoes. Phytopathology 81:1519-1523.
8. Gray, S. M., Moyer, J. W., and Bloomfield, P. 1986. Two-dimensional distance class model for quantitative description of virus-infected plant distribution lattices. Phytopathology 76:243-248.
9. Layne, R. E. C. 1967. Foliar trichomes and their importance as infection sites for *Corynebacterium michiganense* on tomato. Phytopathology 57:981-985.
10. Steel, R. G., and Torrie, J. H. 1980. Principles and Procedures of Statistics, 2nd ed., pp. 188-189. McGraw-Hill, New York.
11. Strider, D. L. 1969. Bacterial canker of tomato caused by *Corynebacterium michiganense:* A literature review and bibliography. N.C. Agric. Exp. Stn. Tech. Bull. 193.
12. Strider, D. L. 1969. Foliage blight phase of bacterial canker on tomato and survival of *Corynebacterium michiganense* in toxicants and in air-dried condition. Plant Dis. Rep. 53:864-868.
13. Strider, D. L. 1970. Tomato seedling inoculations with *Corynebacterium michiganense*. Plant Dis Rep. 54:36-39.
14. Walker, J. C. 1952. Diseases of tomato. Pages 448-449 in: Diseases of Vegetable Crops. McGraw-Hill, New York.