Spread of *Pseudomonas syringae* pv. *tomato* and Role of Epiphytic Populations and Environmental Conditions in Disease Development

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ABSTRACT

Smitley, D. R., and McCarter, S. M. 1982. Spread of *Pseudomonas syringae* pv. *tomato* and role of epiphytic populations and environmental conditions in disease development. Plant Disease 66: 713-717.

In field plots near Athens, GA, *Pseudomonas syringae* pv. *tomato*, which causes bacterial speck of tomato (*Lycopersicon esculentum*), spread rapidly during the cool and moist growing season of 1979 but failed to spread during 1980 when mild to hot and dry weather occurred. In 1979, numbers of epiphytic bacteria and lesions on foliage of Chico III tomato plants increased rapidly from transplanting in late April until early June but then declined with the onset of low-moisture conditions and mean daily temperatures often above 25 C. Epiphytic populations and lesion development continued to decline at high temperatures in July, although moisture levels were again high. Fruits that developed during cool, moist weather in early season were more severely infected than those that developed later. *P. syringae* pv. *tomato* spread rapidly by natural means, and clipping of transplant beds increased disease severity. Although total fruit yields were not significantly reduced by bacterial speck, quality was markedly reduced because 70% of fruit were infected in early harvests. Streptomycin and cupric hydroxide reduced spread and fruit infection. In the growth chamber and field, *P. syringae* pv. *tomato* survived as an epiphyte on symptomless tomato plants for extended periods to produce disease under conducive environmental conditions.

Although first reported in widely separated areas in the early 1930s (2,13), bacterial speck of tomato (*Lycopersicon esculentum* (Mill.)) caused by *Pseudomonas syringae* pv. *tomato* (Okabe) Young et al (hereafter referred to as PST) was not considered to be economically important until the mid-1970s. Several recent occurrence and research reports (7,14,21,22) suggest that the disease is becoming more widespread and damaging.

Bacterial speck was not known to be economically important in the tomato transplant industry of the southern United States until 1978, when a major outbreak occurred both in transplant fields and in northern production areas where some infected transplants were shipped inadvertently (8). Georgia growers incurred major losses to bacterial speck in 1978 when 160 ha of tomato transplants was rejected for certification by the Georgia Department of Agriculture. Although no estimates on monetary losses in fruit-producing areas of the eastern United States are available, the disease has caused reduced quality in fresh market (14) and processing (8) tomatoes, and reports from other areas (3,15,19,22) suggest that direct yield reductions result from early infections by the speck organism.

Several recent studies (1,4,16–18,22) have contributed to a better understanding of the ecology and epidemiology of PST, but some aspects of the pathogen are still poorly understood. For example, little has been reported on its spread under field conditions and its effect on yield. This paper reports observations on the development of bacterial speck in the field during two growing seasons with markedly different weather conditions. The roles of selected bactericides and leaf surface epiphytic populations of PST in disease development are also considered.

Portion of an M.S. thesis by the senior author.

Supported by USDA, Hatch, state, and grower funds allocated to the Georgia Agricultural Experiment Stations.

Accepted for publication 10 November 1981.

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0191-2917/82/08071305/$03.00/0

MATERIALS AND METHODS

Three field studies and a series of greenhouse and growth chamber experiments were conducted during 1979 and 1980. The isolate of PST used in all studies was obtained in 1978 from a diseased tomato seedling growing near Tifton, GA. The isolate (designated Field 3) was grown for 24–48 hr at 25 C on plates of medium B of King et al (KMB) (9) for inoculum production. Tomato plants were inoculated by spraying to runoff with a Burgess model VS-860 paint sprayer (Burgess Vibrocrafters, Inc., Grayslake, IL 60030). Unless indicated otherwise, all cell suspensions used for inoculum contained 10^8 colony-forming units (CFU) per milliliter.

Field studies. *Natural spread of PST.* Field plots were established at the University of Georgia Plant Sciences Farm near Athens during 1979 and 1980 to study natural spread of PST from artificially established inoculum sources and the effect of bacterial speck on yield and fruit quality. In 1980, bactericide spray treatments also were included to determine their effect on spread. The studies were conducted from April through July each year in plot areas that were considered to be free of PST. Standard commercial practices for land preparation, fertilization, and weed and insect control were used. Sprinkler irrigation was used as needed.

In 1979, 20 five-row plots (3.9 × 10.8 m) spaced 2.9 m apart were established. Half of each plot was planted with 60 (12 per row) Chico III (susceptible processing cultivar) tomato plants and half with 45 (9 per row) Marion (susceptible fresh-market cultivar) plants. Plants were spaced 45 cm (Chico III) or 60 cm (Marion) apart in the row, and rows were 0.98 m apart. Five treatments with four replicates in a randomized complete block design were established by transplanting different percentages (0, 1, 10, 30, and 100%) of plants infected with bacterial speck into the plots. The 1% treatment consisted of one diseased plant transplanted into the center of each Chico or Marion subplot. In other treatments, diseased plants were spaced within the plots to facilitate maximum spread. Chlorothalonil was used for fungal disease control; earlier work (5) showed that it was not toxic to PST.

In 1980, 16 five-row plots (3.9 × 10.5 m) spaced 7.7 m apart were established. Chico III plants (24 per row, 120 per plot) were spaced as in 1979. Four treatments were arranged in a randomized complete block design with four replicates. In three treatments, two speck-infected plants were transplanted near the center of each plot, and three separate chemical spray programs were used: chorothalonil (Bravo 6F, 2.5 L/ha); mancozeb (Manzate 200, 2.3 kg/ha) plus cupric hydroxide (Kocide 101, 4.5 kg/ha); and mancozeb plus streptomycin (Agri-Mycin 17, 200 ppm or equivalent to 1.1 kg/ha). A fourth treatment consisted of chlorothalonil applied to plots where no diseased plants were transplanted. Cupric hydroxide and streptomycin are currently recommended by the Georgia Cooperative Extension Service for bacterial speck control on tomato transplants. All

chemicals were applied to runoff at 7- to 10-day intervals with a back-mounted, motorized Solo Mistblower (Solo Motors, Inc., Newport News, VA 23605).

Plants for the tests were grown from disease-free seed in the greenhouse for 7 wk (18–20 cm tall) in 266-ml paper cups filled with a methyl bromide-fumigated soil:sand:vermiculite (3:1:1, v/v) mix. Diseased plants for inoculum sources were produced by spray-inoculating healthy plants with PST 10 days before transplanting, enclosing the plants in polyethylene bags for 36 hr in a growth chamber at 18 C, and returning the plants to the greenhouse (18–25 C) to allow disease development. Inoculated plants had 300–500 lesions at the time of transplanting. Healthy and diseased plants were transplanted in the field on 20 April in 1979 and 29 April in 1980. Care was taken to prevent spread of bacteria from diseased to healthy plants during transplanting and cultivation.

In 1979, detailed studies on spread of PST were conducted in plots that originally received a single infected plant (1% treatment). Spread within each plot was determined by monitoring epiphytic populations of PST on selected plants at 10-day intervals from planting on 20 April until 28 July and recording incidence of foliar lesions on all plants at 10-day intervals until 10 June, when lesions became too numerous to count. Fourteen plants evenly distributed throughout each plot were staked, and these same plants were sampled repeatedly during the growing season. At each sampling, 10 symptomless leaflets were collected at random from each plant, and samples were processed separately in the laboratory.

Each sample was placed in 100 ml of sterile distilled water in a 250-ml flask, shaken vigorously for 10 min on a wrist-action shaker, and 10 ml was removed to prepare a 1:10 dilution for plating in five plates of KMB. Plates were incubated for 48 hr at 25 C, and colonies were counted. Plates of KMB streaked with pure cultures of PST were used for comparison with the dilution plates to assist in distinguishing and eliminating saprophytic fluorescent pseudomonads from the counts. Isolated colonies (15 to 20 per sample) that resembled PST were picked at random from dilution plates to confirm their identity. Diagnostic tests included oxidase (11), arginine dihydrolase (20), tobacco hypersensitivity (10), and pathogenicity on tomato.

In 1980, all plants in each plot except those originally established as inoculum sources were examined at 7- to 14-day intervals, and incidence of speck was recorded. Epiphytic populations were determined as in 1979 except that two (early samples) to five (later samples) plants on each side of the point inoculum sources were sampled. We intended to sample progressively farther from the inoculum source as the season advanced, but the occurrence of adverse weather completely stopped spread of the bacterium and eliminated the need for wider sampling.

Six harvests of pink or ripe fruit were made by hand each year. At each harvest the fruits were weighed and the percentage with bacterial speck determined. In 1980, only fruit in the first two harvests had bacterial speck; later counts were thus not made.

Temperature and relative humidity (RH) were recorded at two separate positions within the plot area. Weather data from instruments maintained at the University Plant Sciences Farm (1 km away) and by the National Weather Service at Athens (26 km away) were also used in the weather analyses.

Spread of PST by clipping. Because tomato transplants produced in southern Georgia are commonly clipped with rotary mowers to increase uniformity and to control size when markets are not available, we determined whether PST could be spread by clipping as is reported for other bacterial pathogens (6,12). A field plot area on the University Plant Sciences Farm with no history of tomato production was seeded (60 seeds per meter of row) with Chico III tomato in rows 20 cm apart in early May of 1979. Sprinkler irrigation was used as needed to promote seedling emergence and normal vegetative growth.

When plants were approximately 10 cm tall, plots (2.9 × 7.0 m) were established. Two plants with bacterial speck, produced in the greenhouse as previously described, were transplanted into the center of each plot when the field plants were 15 cm tall. Plots scheduled to be clipped were clipped 1 wk later with a modified rotary lawn mower (12) adjusted to a cutting height of 15 cm. The clipped plots were mowed twice (8 and 24 June). Fourteen days after the second clipping, 20 plants were collected randomly from each plot, and bacterial speck lesions were counted. The presence of PST was confirmed by randomly selecting lesions, making isolations on KMB, and conducting identification tests listed above.

Greenhouse studies. *Epiphytic survival of PST on emerging seedlings and established tomato plants.* Because results of field studies suggested that PST may survive epiphytically on foliage of tomato under adverse conditions, greenhouse and growth chamber studies were conducted to study this survival mechanism. Experiments were conducted to determine whether seedlings emerging from infested soil were contaminated and the survival period of any resulting epiphytic populations. Chico III tomato seed were planted, 100 per pot and 1.4 cm deep, in 15-cm pots filled with a soil mix fumigated with methyl bromide; the pots were then placed in a growth chamber at 28 C, a temperature too high for disease development (18). At seeding, the covering soil was drenched with 100 ml of a suspension containing PST at 5×10^7 CFU/ml. The pots of tomato seedlings were watered from the bottom to avoid cross-infestation.

At emergence and at 4, 14, and 21 days thereafter, 10 cotyledons and 10 leaves from plants in each pot were collected and assayed for PST. The samples were placed in 75 ml of sterile distilled water in 250-ml flasks, shaken vigorously for 20 min on a wrist-action shaker, and samples of the rinse water were plated on KMB. Resulting colonies were counted under ultraviolet light after incubation of plates for 48–72 hr at 25 C as described in the field studies. Immediately after each sampling, the pots of seedlings were placed in a chamber at 20 C, covered with polyethylene bags for 36 hr, and lesions were counted after 10 days. Six replicates of each treatment and controls (no bacteria added to the soil) were used.

Bacterial suspensions containing 5×10^7 CFU/ml were also misted gently (to minimize infiltration) with a paint sprayer onto both leaf surfaces of 15-cm-tall Chico III tomato plants, and epiphytic survival was studied. In one study, misted plants were placed in growth chambers at 20 and 30 C, and assays made daily from 0 to 7 days. In another study, plants were placed in growth chambers at 19 and 28 C and in the greenhouse, where the temperature ranged from 23 to 32 C. Assays were made at 0, 5, 15, and 25 days after misting of the cell suspension. Both experiments were repeated twice, and 10 replicates of each treatment were used.

Foliage assays were made by randomly collecting 20 leaf disks (7 mm diameter) with a hand hole-punch from each plant, placing the disks in 100 ml of sterile distilled water, and assaying in the laboratory in plates of KMB as previously described. At the time of each assay, the same plants were also enclosed in polyethylene bags to provide high RH and placed at 20 C for 10 days to allow disease development.

RESULTS

Field studies. *Natural spread of PST.* In 1979, populations of PST on symptomless leaves were similar on Chico III and Marion plants; combined data for the cultivars are thus presented (Fig. 1). Populations increased rapidly from transplanting in late April until early June but declined to low levels by July. High populations occurred when the temperature was low, generally averaging below 23 C (Fig. 1A), and moisture levels were high (Fig. 1B). May was unusually cool and moist for northern Georgia. Although the 10.9 cm of recorded rainfall was not excessive, it was distributed so that a significant amount was recorded on each of 14 days,

keeping foliage wet for extended periods and enhancing bacterial spread. In contrast, only 1.9 cm of rainfall occurred on 6 days in June. Higher mean daily temperatures and lower moisture levels in June resulted in a sharp decline in epiphytic populations of PST. Populations continued to decline in July when the weather was humid but hot.

Lesion counts on plants in plots originally established with one diseased plant were related closely to leaf populations of PST, with a 10- to 12-day lag period required for infection and symptom expression (Fig. 1). Initially, plants near the originally established diseased plant had higher lesion counts than plants more distant, but rapid spread soon eliminated this difference. Plants in each plot had average counts of more than 100 lesions per plant by 28 May. Lesion counts were discontinued on 12 June when the large number of lesions on plants made counting impractical. Once started, the disease spread rapidly in all plots regardless of the percentage of plants originally infected. No lesions occurred on plants in check plots (established without diseased plants) until mid-May, although numerous lesions had already appeared on plants established with a single diseased plant. However, rapid spread occurred even in check plots once the bacterium was introduced.

The rapid spread of the bacterium and high disease severity in all treatments were reflected in heavy fruit infection (Fig. 2). Incidence of fruit infection sometimes reached 70% in the first three harvests but then declined in later harvests. In the first harvest, fruit infection levels were significantly higher in plots established with 30 and 100% infected plants than in plots with 0, 1, and 10% infected plants. There were also significant differences among treatments in the second but not later harvests. In the first two harvests, yields of total fruit (healthy and diseased) were also significantly lower in plots originally established with 100% infected plants than in other plots, but there were no significant yield differences among the other four harvests. Total yields (diseased plus healthy fruits) were not significantly different among treatments when all harvests were considered.

Weather conditions during 1980 were unusually dry and warm for northern Georgia. No rainfall occurred during the 14 days following transplanting on 29 April. The only appreciable rain early in the season came between 14 and 24 May, when 22.1 cm was recorded. This brief rainy period, which initiated some increase in epiphytic populations of PST and subsequent disease development, was followed by 23 days without recorded rainfall. Only a total of 13.2 cm of rain occurred during June and July. These extended periods of dry weather that kept the foliage dry plus high mean temperatures (>24 C) during most of June and July resulted in little spread from the original disease loci. Epiphytic populations on foliage were low and erratic on symptomless leaves, even on plants adjacent to the established disease loci. Populations never reached 10% of those recorded in 1979, and the bacterium was usually not detected by laboratory plating after mid-June.

Low foliage populations were accompanied by low infection levels on both foliage and fruits (Table 1). Plant infection incidence was below 7%, and only a few scattered lesions (usually five to 30) occurred on the infected plants. In some cases, the mean number of lesions recorded on plants early in the season was reduced later because of the rapidly developing disease-free foliage. Applications of either mancozeb + cupric hydroxide or mancozeb + streptomycin significantly reduced epiphytic bacterial populations and infection rates on foliage and fruits when compared with chlorothalonil alone in plots where disease loci were established (Table 1). There were no differences in fruit yields among treatments.

Spread of PST by clipping. Lesion counts on the 20 young tomato plants collected at random from clipped plots were significantly higher than on plants from unclipped plots. Mean lesion counts were 2 and 19 for unclipped and clipped plants, respectively. PST was consistently isolated from the lesions.

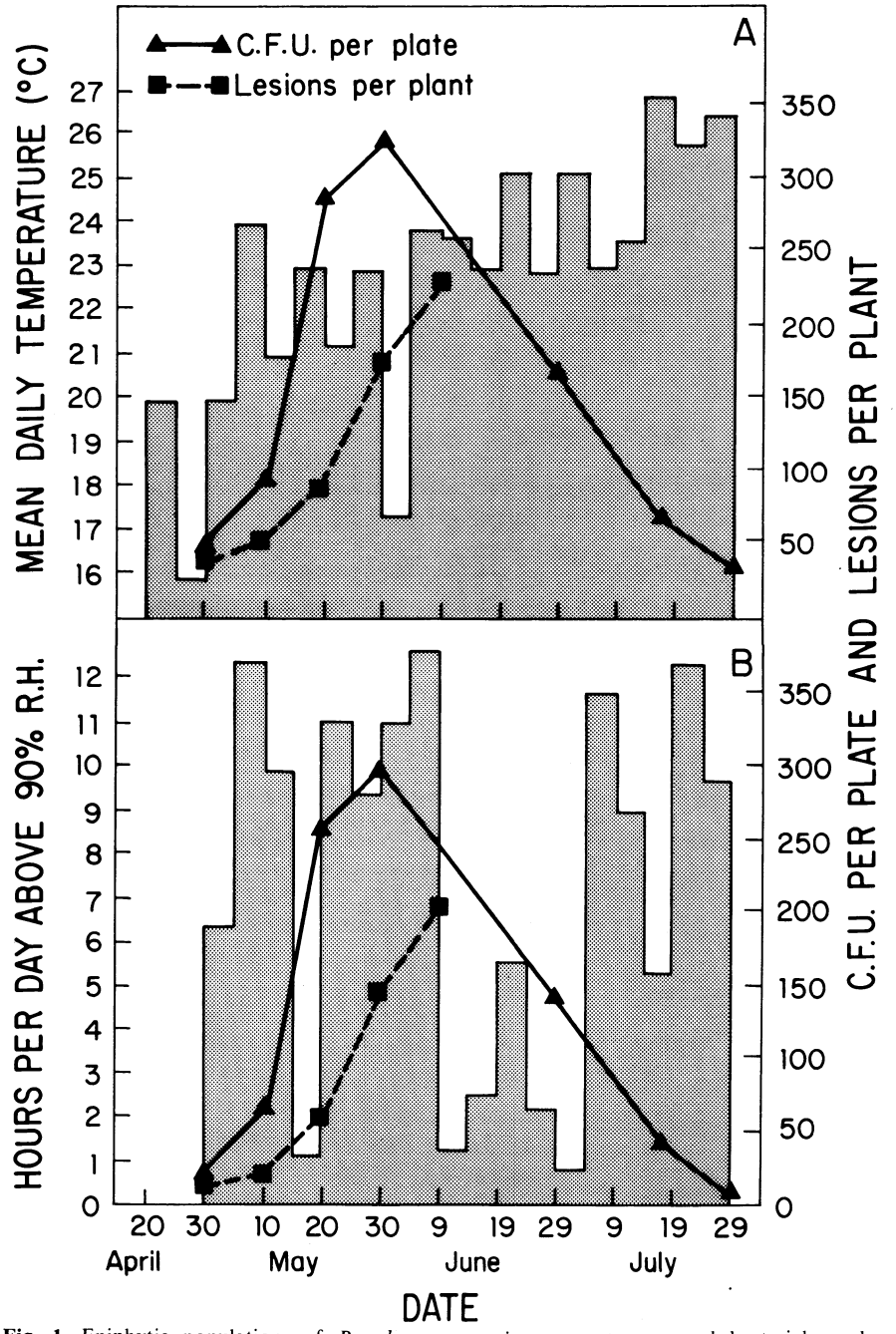

Fig. 1. Epiphytic populations of *Pseudomonas syringae* pv. *tomato* and bacterial speck development as related to temperature **(A)** and moisture **(B)** in 1979 in field plots near Athens, GA.

Table 1. Incidence of bacterial speck lesions on foliage and fruits of Chico III tomato plants receiving various treatments in field plots near Athens, GA, during 1980

Treatment[a]	Plants infected (%)				Fruit infected[b] (%)	
	27 May	3 June	10 June	23 June	26 June	7 July
Chlorothalonil	6.7 y[c]	5.5 x	3.8 x	1.2 y	3.8 x	2.3 x
Mancozeb + cupric hydroxide	0.6 z	0.8 y	0.0 z	0.0 z	1.2 y	1.1 y
Mancozeb + streptomycin	0.4 z	0.0 z	0.0 z	0.0 z	0.6 y	0.9 y
Chlorothalonil (check)	0.4 z	0.2 y	0.2 y	0.0 z	0.0 z	0.1 z

[a] All treatments except the check had two speck-infected plants transplanted in the center of each plot to provide initial inoculum. See text for rates and method of chemical application.
[b] No significant lesions occurred on fruit except in the first (26 June) and second (7 July) harvests.
[c] Values followed by the same letter are not significantly different according to Duncan's multiple range test ($P = 0.05$).

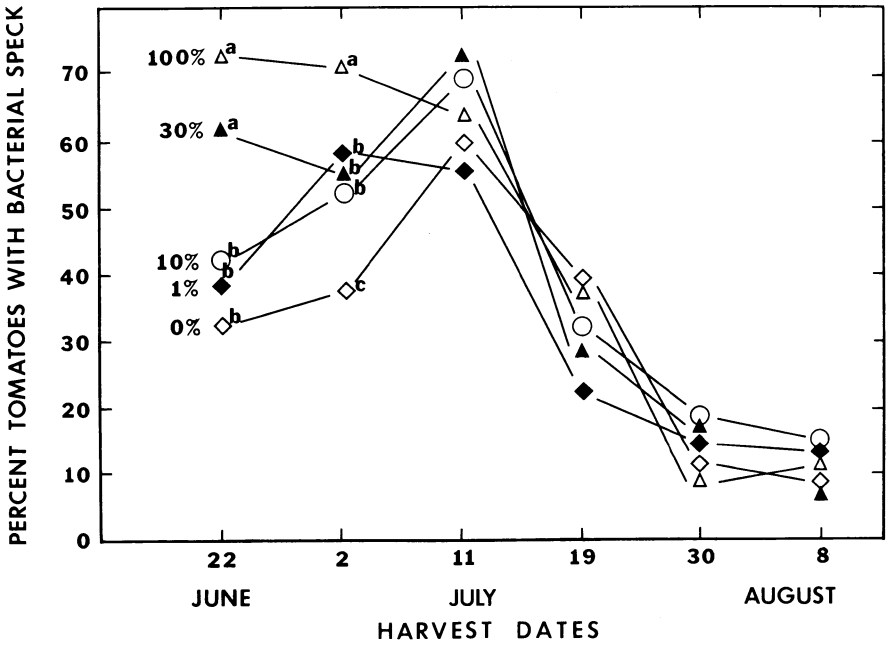

Fig. 2. Percentage of Chico III and Marion (combined data) fruits with bacterial speck harvested from field plots planted initially with different percentages (0–100%) of infected plants in 1979 near Athens, GA. Lowercase letters denote significant differences ($P = 0.05$).

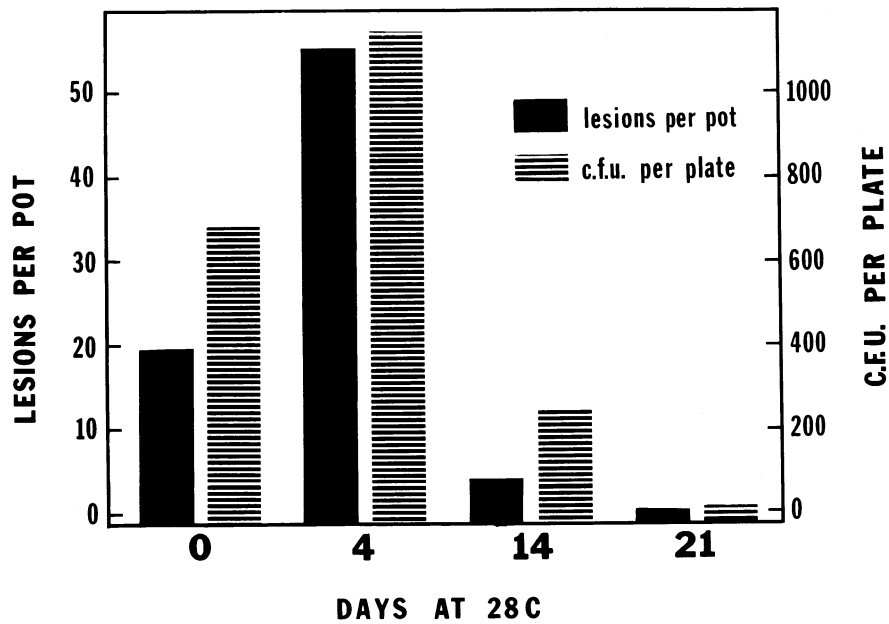

Fig. 3. Isolation of *Pseudomonas syringae* pv. *tomato* from tomato seedlings at emergence and at 4–21 days at 28 C after emergence from soil artificially infested and corresponding disease severity when the same plants were placed for an additional 10 days under moisture (enclosed in polyethylene bags for 36 hr) and temperature (20 C) conditions that were optimum for disease development.

Greenhouse studies. *Epiphytic survival of PST on emerging seedlings and established plants.* PST was recovered readily from Chico III tomato seedlings that emerged from infested soil at 28 C (Fig. 3). Populations were significantly higher 4 days after emergence than at emergence but declined to low levels at 14 and 21 days. Lesion counts on the emerged seedlings placed under high-moisture conditions (enclosed in polyethylene bags at 20 C) for 36 hr and held at 20 C for 10 days were related closely to laboratory plate counts made on tissue samples (Fig. 3). Symptoms were absent before plants were given the high-moisture and low-temperature treatment except for a few lesions along the margins of the cotyledons. Lesions on plants given a high-moisture and low-temperature period for 10 days after 14 and 21 days at 28 C usually occurred on the bottom leaves near cotyledons or on leaves that had been in close proximity to the apical meristem at the time that the high-moisture, low-temperature treatment was given.

When established (15-cm-tall) Chico III tomato plants were misted with a suspension of PST and placed in growth chambers at 20 and 30 C for 7 days, epiphytic populations and corresponding lesion counts (after plants were given 36-hr high-moisture period and held at 20 C) increased daily from 1 to 5 days and then declined to low levels by 7 days. Lesion counts made after appropriate incubation were significantly higher on plants held for 1–5 days at 20 C than on those held for corresponding periods at 30 C.

Trends, as related to temperature, were similar in another study of plants kept for 25 days in growth chambers at 19 and 28 C and in the greenhouse at fluctuating temperatures (23–32 C). Five days after plants were misted with bacteria, counts of epiphytic populations were about 15 times higher on plants at 19 C than at 28 or 22–32 C. Lesion counts (after appropriate incubation of test plants) were also two or three times higher on plants from the treatment at 19 C than from the other two treatments. Bacterial populations and subsequent lesion counts increased significantly from 0 to 5 days after misting at 19 C, but both counts were similar or declined between the two test periods at the higher temperature. Epiphytic populations and subsequent lesion counts declined significantly between 5- and 15-day samplings at all temperatures, but at 19 C both counts were about double those at 28 or 23–32 C. Although PST could not be detected by plating methods from any temperature treatment 25 days after misting, some lesions (mean of 10–55 lesions per plant) developed on all plants when placed under optimum condition for disease development, indicating that the organism had survived in low populations.

DISCUSSION

The markedly contrasting weather conditions during 1979 and 1980 provided an unusual opportunity to relate temperature and moisture conditions to spread of PST and disease development in the field. In 1979, epiphytic populations of PST on tomato leaves and numbers of foliar lesions increased rapidly during the early season but declined in mid-June with the onset of mean daily temperatures often above 25 C and of low-moisture conditions. Failure of foliar populations or lesions to increase in July when moisture again became high showed that both low temperature and high moisture are required for disease development. Fruits that developed during the early season were also more severely infected than those that developed at higher temperatures. PST did not spread appreciably from artificially established disease loci during 1980 when extended periods of dry and cool or later dry and hot weather occurred.

In presenting our 1979 results, we graphically related disease incidence to high (>90%) RH. However, we realize that periods of free moisture were probably determining factors in the infection process. We presented RH as an indirect measure because data on free moisture on leaf surfaces were not available. Our data generally agree with findings in California (18) that infection was inhibited at 22.8 C and in Israel (22) that high disease incidence was correlated with temperatures below 25 C and RH above 80%. The cool, wet conditions that occurred at Athens during the early season of 1979 were similar to those occurring in tomato transplant fields of southern Georgia in 1978, when major losses to bacterial speck occurred. A similar outbreak of bacterial speck in certain fruit-producing areas of Florida during early 1978 was attributed to an unusually cool and wet season (14). The occurrence of unusually favorable weather conditions may explain some of the major outbreaks of bacterial speck in recent years. However, failure of the disease to develop under equally favorable conditions in other years suggests that other factors may be operative.

The results of our 1979 field studies show that PST has a high potential for spread under conducive environmental conditions. The rapid spread of the bacterium from a single infected plant in the field shows the danger of transplanting even a low percentage of infected plants in northern production areas where early season growing conditions are often conducive for spread; it also emphasizes the need for effective control measures in Georgia fields where transplants originate. Clipping of tomato transplants apparently results in spread of PST, as was reported for two other bacterial pathogens (6,12).

We are confident that the spread that occurred in our field plots originated entirely from our introduced inoculum and not from outside sources. Plants for the test were grown from disease-free seed, and uninoculated plants remained free of disease during the 6 wk in the greenhouse and for 3–4 wk after transplanting in check plots in the field. Plants from the same seeding used in another nearby test remained free of speck throughout the growing season. The plot area was selected because it was considered free of PST, and there was no evidence that any infection resulted from inoculum originating from the soil or weed hosts.

We attempted to maintain a disease-free check so that any yield reductions resulting from bacterial speck could be determined. The eventual spread of the bacterium into the check plots in 1979 did not allow for disease-free plots. Under these conditions, bacterial speck did not cause direct yield reductions when all harvests are considered collectively. However, significantly higher yields from two early harvests in plots that originally had fewer infected plants than in plots that had more infected plants support earlier findings (15,19) that bacterial speck delays maturity and decreases yield because of the foliar damage. Reduction of fruit quality is apparently a greater threat, as 70% of fruit were infected in some of our early harvests.

Our field and growth chamber studies provide convincing evidence that PST survives epiphytically for extended periods on tomato foliage. Isolation of the organism from symptomless tomato leaves collected from the field at Athens and from plants under low-moisture and high-temperature conditions in growth chambers after 2 wk or more supports the conclusion of Schneider and Grogan (16) that PST survives as an epiphyte on foliage to become infective when environmental conditions are suitable. The possibility of epiphytic survival of PST on transplants shipped from fields in southern Georgia to northern areas suggests the need for control measures to eliminate epiphytic populations even when conditions may not be conducive for disease development in the South. Although little bacterial speck developed in our plots in 1980, the data collected and earlier work (5) indicate that treatments of streptomycin or cupric hydroxide combined with fungicides currently recommended for transplant production significantly control epiphytic populations and should be used on a preventive basis until more effective control measures are devised.

ACKNOWLEDGMENT

We thank Jan Fowler for technical assistance.

LITERATURE CITED

1. Bashan, Y., Okon, Y., and Henis, Y. 1978. Infection studies of *Pseudomonas tomato*, causal agent of bacterial speck of tomato. Phytoparasitica 6:135-144.
2. Bryan, M. K. 1933. Bacterial speck of tomatoes. Phytopathology 23:897-904.
3. Burki, T. 1972. *Pseudomonas tomato* (Okabe) Alstatt, Erreger einer für die Schweiz neuen Tomatenbakteriose. Schweiz. Landwirtsch. Forsch. 11:97-107.
4. Chambers, S. C., and Merriman, P. R. 1975. Perennation and control of *Pseudomonas tomato* in Victoria. Aust. J. Agric. Res. 26:657-663.
5. Conlin, K. C., and McCarter, S. M. 1980. Effectiveness of bactericides and bactericide-fungicide combinations in inhibiting *Pseudomonas tomato* and controlling bacterial speck. (Abstr.) Phytopathology 70:566.
6. Farley, J. D., and Miller, T. D. 1973. Spread and control of *Corynebacterium michiganense* in tomato transplants during clipping. Plant Dis. Rep. 57:767-769.
7. Goode, M. J., and Sasser, M. 1980. Prevention—The key to controlling bacterial spot and bacterial speck of tomato. Plant Dis. 64:831-834.
8. Kim, S. H. 1979. Dissemination of seed-borne *Pseudomonas tomato* by transplants. (Abstr.) Phytopathology 69:535.
9. King, E. O., Ward, M. K., and Raney, D. E. 1954. Two simple media for the demonstration of pyocyanin and fluorescin. J. Lab. Clin. Med. 44:301-307.
10. Klement, Z., Farkas, G. L., and Lovrekovich, L. 1964. Hypersensitive reaction induced by phytopathogenic bacteria in the tobacco leaf. Phytopathology 54:474-477.
11. Kovacs, N. 1956. Identification of *Pseudomonas pyocyanea* by the oxidase reaction. Nature (London) 178:703.
12. McCarter, S. M., and Jaworski, C. A. 1969. Field studies on spread of *Pseudomonas solanacearum* and tobacco mosaic virus in tomato plants by clipping. Plant Dis. Rep. 53:942-946.
13. Okabe, N. 1933. Bacterial diseases of plants occurring in Formosa. II. Bacterial leaf spot of tomato. J. Soc. Trop. Agric. Taiwan 5:26-36.
14. Pohronezny, K., Volin, R. B., and Stall, R. E. 1979. An outbreak of bacterial speck on fresh-market tomatoes in Florida. Plant Dis. Rep. 63:13-17.
15. Reid, W. D. 1948. Bacterial speck of tomato. N.Z. J. Sci. Technol. 30:5-8.
16. Schneider, R. W., and Grogan, R. G. 1977. Bacterial speck of tomato: Sources of inoculum and establishment of a resident population. Phytopathology 67:388-394.
17. Schneider, R. W., and Grogan, R. G. 1977. Tomato leaf trichomes, a habitat for resident populations of *Pseudomonas tomato*. Phytopathology 67:898-902.
18. Schneider, R. W., and Grogan, R. G. 1978. Influence of temperature on bacterial speck of tomato. (Abstr.) Phytopathol. News 12:204.
19. Schneider, R. W., Hall, D. H., and Grogan, R. G. 1975. Effect of bacterial speck on tomato yield and maturity. (Abstr.) Proc. Am. Phytopathol. Soc. 2:118.
20. Thornley, M. J. 1960. The differentiation of *Pseudomonas* from other Gram-negative bacteria on the basis of arginine metabolism. J. Appl. Bacteriol. 23:37-52.
21. Wilkie, J. P., and Dye, D. W. 1974. *Pseudomonas tomato* in New Zealand. N.Z. J. Agric. Res. 17:131-135.
22. Yunis, H., Bashan, Y., Okon, Y., and Henis, Y. 1980. Weather dependence, yield losses, and control of bacterial speck of tomato caused by *Pseudomonas tomato*. Plant Dis. 64:937-939.