Incidence and Development of *Pseudomonas syringae* pv. *syringae* on Tomato Transplants in Georgia

R. D. GITAITIS, Coastal Plain Experiment Station, University of Georgia, Tifton 31793; J. B. JONES, Agricultural Research and Education Center, University of Florida, Bradenton 33508; and C. A. JAWORSKI, USDA, ARS, and S. C. PHATAK, Coastal Plain Experiment Station, University of Georgia, Tifton 31793

ABSTRACT

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During a 5-yr period (1980–1984), *Pseudomonas syringae* pv. *syringae* (PSS) was the most frequently recovered (58% of all diagnoses) bacterial plant pathogen from tomato transplants inspected in the Georgia plant-certification program. An additional 9.2% of the time, PSS was recovered from mixed infections with *Xanthomonas campestris* pv. *vesicatoria* (XCV), but PSS was never found in conjunction with *P. syringae* pv. *tomato* (PST), the causal agent of bacterial speck. Twenty tomato cultivars grown as transplants varied greatly in susceptibility to PSS. Campbell 28 and Ohio 7663 were highly resistant, whereas Veepro, Peto 95, and Hunt 304 were moderately susceptible. The bacterium was recovered from leaf washings of symptomless wild cherry and rye leaves that were adjacent to or within tomato transplant fields.

Additional key words: *Lycopersicon esculentum*, *Prunus serotina*, *Secale cereale*

Since its initial report (7), a high incidence of *Pseudomonas syringae* pv. *syringae* van Hall (PSS) has continued to be found in southern field-grown tomato (*Lycopersicon esculentum* Mill.) transplants. In Georgia, the disease causes only minor damage and regresses as plants develop and mature; however, it is of major importance to the Georgia plant-certification program. Plants with bacterial speck caused by *P. syringae* pv. *tomato* (Okabe) Young, Dye, & Wilkie (PST) or bacterial spot caused by *Xanthomonas campestris* pv. *vesicatoria* (Doidge) Dye (XCV) are a potential source of inoculum for epiphytotics in northern tomato-growing areas. Symptoms on tomato foliage caused by PSS are often difficult to distinguish from those of bacterial speck or bacterial spot. The problem is compounded by the diversity of tomato cultivars being grown (3) and variation among bacterial strains (14), which affect symptom expression. This creates difficulty for plant inspectors, who in the past, evaluated plants solely on the basis of symptoms. It is the current practice to identify the causal pathogen under laboratory conditions using certain bacteriological methods (6). Plants diagnosed as having PSS retain certification, whereas plants with XCV or PST are placed under quarantine. The average production of plants is about 800,000 plants per hectare, with an approximate value of $9,200.00/ha. Consequently, the retention of certification of plants with PSS, a disease of only cosmetic significance, is of economic importance to growers in southern Georgia.

Although there is much information on PSS in association with other crops and weed hosts (1,11,15,16), little is known about the ecology of PSS in tomato transplant fields. This work was done to determine the incidence of PSS in transplants, to evaluate the relative susceptibility of tomato cultivars, and to ascertain overseasoning mechanisms of the bacterium. Because plants with PSS retain certification and because of a concern that the presence of PSS might interfere with the ability to detect PST, the possibility of mixed infections was investigated.

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MATERIALS AND METHODS

Disease survey and diagnosis. Isolations were made from diseased tomato plants collected by Georgia Department of Agriculture plant inspectors from approximately 51, 15, 70, 58, and 170 ha in 1980, 1981, 1982, 1983, and 1984, respectively. In all cases, lesions from three replicates were triturated separately in drops of sterile water from which a loopful (0.005 ml) was streaked for isolation onto nutrient agar (NA) and medium B of King et al (KMB) (8). After incubation at 30 C, fluorescent colonies from KMB were tested for oxidase, arginine dihydrolase, and ice-nucleation reactions as well as for acid production in erythritol (2,6). Yellow-pigmented bacteria characteristic of XCV that were isolated on NA were characterized by gram reaction; flagellar configuration; starch and gelatin hydrolysis; oxidase, catalase, and nitrate-reduction reactions; acid production in glucose aerobically and anaerobically; and cellulytic reaction on a carboxymethyl cellulose medium. This latter medium consisted of 0.5 g of $NH_4H_2PO_4$, 0.1 g of KCl, 0.5 g of $MgSO_4 \cdot 7H_2O$, 0.5 g of yeast extract, 1.5 g of agar, 12.5 g of sodium salt of carboxymethyl cellulose (CMC), and 500 ml of distilled water. The CMC and agar were added last while blending at high speed until the medium had a gel-like consistency without lumps. It was autoclaved in an oversized flask (500 ml/2-L flask) because the medium is highly viscous. Because of its soft texture, streaking was done gently to avoid gouging. An antibubble additive such as Pourite (Analytical Products, Inc., Belmont, CA) was used to ensure a smooth surface.

Resistance of tomato cultivars to PSS. Twenty tomato cultivars were seeded to simulate commercial transplant production as described previously (3). Recommended fertilizer and pest control practices were maintained (5). The experimental design was a randomized complete block with four replicates. Twenty plants were sampled from the center of each plant bed and evaluated for disease severity. Disease was established by natural infection. Disease severity was estimated by using the Horsfall-Barratt rating system (4). Data were analyzed by analysis of variance and the Waller-Duncan *k*-ratio test.

Selective isolation of PST from mixtures of PSS and PST. Tomato plants (cultivar FM 6203) grown in the greenhouse were inoculated with either PSS (strain RT) or PST (strain PT 80-16). Inoculum was prepared from nutrient-broth shake cultures that had been started from a single colony-forming unit (cfu) and were incubated at 30 C for 24 hr. Inoculum was harvested by centrifugation and adjusted in sterile tap water to a concentration of about 10^8 cfu/ml. Tomato plants were inoculated

by rubbing Carborundum-dusted leaves with a cotton swab saturated with the bacterial suspension. Plants were placed in a mist chamber overnight, then removed to a greenhouse bench. After 7–10 days, individual lesions were dissected from the leaves and grouped in the following combinations: 10 PSS lesions/0 PST lesions, 9 PSS/1 PST, 5 PSS/5 PST, 1 PSS/9 PST, and 0 PSS/10 PST. Three replicates of each group of lesion combinations were triturated in 10 ml of sterile water. The resulting suspensions were streaked onto KMB and a medium with erythritol as the sole carbon source (13). In addition, tomato plants (FM 6203) were used as a bioassay for selective recovery of PST. Suspensions from the various lesion combinations were diluted to 25 ml with sterile water and filtered through cheesecloth to remove the largest particulate matter. Inoculum was applied with an aerosol chromatography sprayer, then plants were placed on a greenhouse bench. These conditions (absence of wounding and lack of incubation in a mist chamber) had proven acceptable for bacterial speck development but not for infection with PSS (7). Representative lesions that resulted from each treatment were collected, macerated in a drop of water, and streaked onto the media described earlier. Fluorescent bacteria were picked from plates of KMB and characterized for arginine dihydrolase, ice-nucleation, and oxidase reactions as well as for acid production in erythritol. Three isolations were made from each replicate.

Mixed infections of PST and PSS were determined in commercially field-grown plants by the laboratory procedures and bioassay outlined before. Plants of the tomato cultivars Peto 95, Heinz 2653, Heinz 727, FM 6203, and New Yorker were collected in 1983 from fields at five locations that had been diagnosed as having only PSS. Three isolations were made from each and the bacteria were characterized in the laboratory. In addition, bacterial suspensions obtained directly from macerated lesions were diluted to 25 ml and applied as a spray mist to plants of FM 6203. Evaluations were made after 7, 14, and 21 days. Controls of PSS (RT) and PST (PT 80-16) were used for comparison. Any resulting lesions were to be used for isolations and the bacteria to be characterized by key tests that separate PSS from PST.

Survival of PSS. Five locations in southern Georgia with a history of plants infected with PSS were studied in 1983. Samples were taken from soil, weeds, cover crops, and windbreaks periodically between February and November 1983. This time span encompassed periods that corresponded with overwintering and oversummering inoculum as well as the entire transplant season. Plant and soil samples were shaken with sterile buffered saline (0.01 M K_2HPO_4-KH_2PO_4, pH 7.0, and 0.85% NaCl) for 30 min. Dilutions were plated on KMB and a DL-lactate medium (3.33 ml of 60% sodium lactate syrup, 1 g of NaCl, 0.2 g of $MgSO_4 \cdot 7H_2O$, 1 g of $(NH_4)_2HPO_4$, 0.5 g of KH_2PO_4, 15 g of agar, 20 ml of 0.04% phenol red, and 980 ml of distilled H_2O). Suspect fluorescent colonies were characterized by physiological tests (2), tobacco hypersensitivity (9), and pathogenicity on tomato and California Blackeye No. 3 cowpea (10).

RESULTS

Disease survey and diagnosis. Bacteria isolated from lesions on tomato foliage that were fluorescent on KMB, negative

Table 1. A survey of bacterial diseases of tomato transplants in Georgia during a 5-yr period (1980–1984)

	Number of diagnoses by weeks for each pathogen or mixture										
	March	April				May				June	Percentage of total diagnoses for all pathogens
Bacterial strain[a]	4	1	2	3	4	1	2	3	4	1	
1980											
PSS	0	0	33	8	21	21	5	5	0	0	60
PST	0	0	0	0	0	7	0	0	7	7	14
XCV	0	0	0	15	0	15	0	0	0	0	20
PSS + XCV[b]	0	0	0	0	0	9	0	0	0	0	6
PSS + PST	0	0	0	0	0	0	0	0	0	0	0
PST + XCV	0	0	0	0	0	0	0	0	0	0	0
1981											
PSS	0	0	5	13	8	0	0	0	0	0	56
PST	0	0	0	0	0	0	0	0	0	0	0
XCV	0	0	3	7	3	0	0	0	0	0	28
PSS + XCV	0	0	0	5	2	0	0	0	0	0	16
PSS + PST	0	0	0	0	0	0	0	0	0	0	0
PST + XCV	0	0	0	0	0	0	0	0	0	0	0
1982											
PSS	2	0	19	19	32	42	0	0	2	0	56
PST	0	0	0	0	0	2	2	2	0	2	4
XCV	0	0	7	4	28	18	4	0	4	0	30
PSS + XCV	0	0	4	8	8	0	0	0	0	0	10
PSS + PST	0	0	0	0	0	0	0	0	0	0	0
PST + XCV[b]	0	0	0	0	0	0	0	0	0	2	1
1983											
PSS	2	0	3	3	13	3	9	9	5	0	28
PST	0	0	0	0	0	2	2	4	20	0	16
XCV	0	0	0	0	4	4	11	6	31	0	32
PSS + XCV	0	0	0	0	4	4	0	2	6	0	10
PSS + PST	0	0	0	0	0	0	0	0	0	0	0
PST + XCV	0	0	0	0	0	0	2	2	20	0	14
1984											
PSS	0	6	15	3	72	96	15	9	3	3	88
PST	0	0	0	0	0	0	0	3	3	3	4
XCV	0	0	0	0	0	0	0	6	3	0	4
PSS + XCV	0	0	0	0	0	0	0	0	6	3	4
PSS + PST	0	0	0	0	0	0	0	0	0	0	0
PST + XCV	0	0	0	0	0	0	0	3	0	0	1

[a] Abbreviations for bacteria: PSS = *Pseudomonas syringae* pv. *syringae*, PST = *P. syringae* pv. *tomato*, and XCV = *Xanthomonas campestris* pv. *vesicatoria*.
[b] Isolated as mixed infections.

Table 2. Disease severity ratings of 20 processing tomato cultivars infected with *Pseudomonas syringae* pv. *syringae* in southern Georgia

Cultivar	Disease severity[y] (%)
Veepro	35.0 a[z]
Peto 95	29.5 ab
Hunt 304	25.0 abc
New Yorker	21.0 bcd
Heinz 1630	18.5 bcde
Heinz 727	17.0 cdef
Campbell 38	15.0 cdef
Heinz 2653	14.0 def
VF-134-1-2	14.0 def
FM 6203	13.0 def
Libby 8990 A	10.5 efg
Libby 7241	9.0 fg
Campbell 37	7.0 gh
Libby 68	4.5 hi
Chico III	4.0 hij
Heinz 722	3.0 ij
Heinz 1706	3.0 ij
Heinz 318	2.5 ijk
Ohio 7663	0.8 jk
Campbell 28	0.4 k

[y] Percentage of disease severity based on conversions from Horsfall-Barratt ratings (4).
[z] Mean separation according to Waller-Duncan *k*-ratio ($P = 0.05$).

for oxidase and arginine dihydrolase but positive for ice nucleation, growth on DL-lactate, and acid production in erythritol were identified as PSS. Similar bacteria that were negative for ice nucleation, did not grow on DL-lactate, and were unable to produce acid in erythritol were identified as PST. Bacteria that had a non-water-soluble yellow pigment and were oxidase-negative, catalase-positive, strictly aerobic, proteolytic, and cellulytic were identified as XCV.

Disease levels varied, resulting in differing sample numbers from year to year. During the transplant season, PSS consistently was the first pathogen detected, in late March to mid-April. A mean of 85% of all cases occurred by the first week in May (Table 1). Bacterial spot caused by XCV, on the other hand, was not detected until mid- to late April and was found until the end of the season, which was the last week of May or early June. PST was never detected until the first week of May and like XCV was found throughout the remainder of the season, except in 1981, when PST was not found at all. During the 5-yr period (1980–1984), PSS accounted for 58% of all diagnoses. PSS was also found in mixed infections with XCV an additional 9.2% of the time but was never found in conjunction with PST. PST made up a mean of only 7.6% of all diagnoses, with an additional 3.2% as mixed infections with XCV. XCV accounted for a mean of 22.8% of all diagnoses from 1980 to 1984 (Table 1). In mixed infections of PSS with XCV or PST with XCV, the fluorescent pseudomonad was the predominant organism isolated on NA or KMB. In nearly all instances, XCV (although distinctive) was found in the mass streak only.

Resistance of tomato cultivars to PSS. The tomato cultivars were infected naturally in field plots in 1982. In all treatments from four replicates, the causal pathogen was identified as PSS by isolation of the organism and its characterization by key tests (6,7). Disease levels ranged from moderately susceptible (35%) for Veepro to slight (0.4%) for Campbell 28 (Table 2). In susceptible cultivars, lesions were 1–3 mm in diameter and often coalesced to form extensive necrotic areas. In these cases, individual lesions and blighted areas were associated with distinct chlorotic halos. In resistant cultivars, individual lesions were not distinct, halos were generally absent, and lesions rarely exceeded 1 mm in diameter.

Selective isolation of PST from mixtures of PSS and PST. Fluorescent bacteria were recovered from all five PSS/PST lesion combinations on KMB and from four of the combinations on erythritol. No fluorescent bacteria were recovered on the erythritol medium from the tomato lesion combination of 10 PST/0 PSS. Three fluorescent colonies were picked from each treatment and replicate on KMB and characterized for arginine dihydrolase and acid production in erythritol. All 45 colonies were negative for arginine dihydrolase, but the pattern of the utilization and production of acid in erythritol correlated with the original ratio of PSS to PST lesions.

In the bioassay, disease levels corresponded with the proportion of the original number of PST lesions. Severe bacterial speck developed from the 0 PSS/10 PST and 1 PSS/9 PST inoculum combinations, moderate levels developed from the 5 PSS/5 PST inoculum source, low levels developed from the 9 PSS/1 PST inoculum, and no disease from the 10 PSS/0 PST inoculum. A random selection of lesions from all treatments yielded only PST upon isolation. The bioassay could be used selectively to differentiate PST from PSS.

Commercially field-grown plants from five sites that had been diagnosed as having PSS yielded only PSS upon isolation. In addition, all were negative for bacterial speck development when analyzed by the bioassay method.

Table 3. Comparison of known *Pseudomonas syringae* pathovars with *Pseudomonas* strains from rye and cherry leaves in tomato transplant fields

Test	Number of strains with positive reactions: Unidentified test strains	pv. *tomato*	pv. *syringae*	pv. *lachrymans*	pv. *tabaci*	pv. *coronafaciens*
Strains tested	9	8	4	2	1	2
Fluorescent on KMB	9	8	4	2	1	2
Oxidase	0	0	0	0	0	0
Arginine dihydrolase	0	0	0	0	0	0
Levan	9	8	4	2	1	2
Tobacco hypersensitivity	9	8	4	2	0	2
Nitrate reduction	0	0	0	0	0	0
Catalase	9	8	4	2	1	2
Starch hydrolysis	0	0	0	0	0	0
Gelatin hydrolysis	9	0	3	2	0	0
Urease	9	(8)[a]	4	(2)	1	2
Tyrosinase	0	0	0	0	0	2
Ice nucleation	9	0	4	NT[a]	NT	2
Cowpea pathogenicity[b]	9	0	4	0	0	2
Tomato pathogenicity[b]	9	8	4	0	0	0
Utilization of						
D (−)tartrate	5	8	2	0	0	0
DL-lactate	9	0	4	0	0	2
Succinate	9	8	4	2	1	2
Citrate	9	8	4	2	1	2
Production of acid in						
Erythritol	9	0	4	2	1	2
Glycerol	9	8	4	2	1	2
Inositol	9	8	4	2	1	2
Sorbitol	9	8	4	2	1	2
Mannitol	9	8	4	2	1	2
Sucrose	9	8	4	2	1	2
Glucose	9	8	4	2	1	2
Fructose	9	8	4	2	1	2
Galactose	9	8	4	2	1	2
Rhamnose	9	8	4	2	1	2
Xylose	9	8	4	2	1	2
Arabinose	9	8	4	2	1	2
Cellobiose	8	8	3	2	1	2
Lactose	7	6	3	2	1	1

[a] Values in parentheses indicate a weak reaction after 7 days, becoming stronger after 14 days of incubation. NT = not tested.
[b] Pathogenicity tests based on Carborundum rub-inoculation method with plants placed in a mist chamber.

Therefore, within the sample, the infection and presence of PSS did not mask a mixed infection with PST.

Survival of PSS. Fluorescent bacteria suspected of being PSS were recovered as epiphytes from leaf washings of symptomless rye (*Secale cereale* L.) and wild cherry (*Prunus serotina*). Bacteria pathogenic on tomato were recovered from these plants from February through November in 1983, a time span that encompassed the transplant season and oversummering months. No pathogenic bacteria were detected in any of the soil recovery attempts. The DL-lactate medium was helpful in the recovery of PSS because many saprophytic pseudomonads and other background microflora were eliminated compared with KMB. The identity of the epiphytes was confirmed as PSS by physiological tests (Table 3) and pathogenicity on tomato and California Blackeye No. 3 cowpea.

DISCUSSION

The occurrence of PSS in southern-grown tomato transplants has created both problems and questions. The most immediate concern of the tomato industry was for regulatory personnel to be able to make a rapid and accurate diagnosis. Within the limitations of manpower and sample size, these problems have largely been overcome (6). This study provides additional information about the ecology and epidemiology of PSS. During the 5-yr period (1980–1984), PSS was the predominant pathogen in the early part of the transplant season, with a mean of 85% of all occurrences being detected by the end of April. However, occurrence may be somewhat dependent upon environmental conditions. In 1983, cool temperatures and excessive rainfall significantly delayed planting and retarded growth. A possible effect of this delay was that in 1983, PSS accounted for a smaller proportion of the total bacterial disease present. In addition, the incidence of PSS in May was more prevalent than normal. Environmental conditions also affected disease levels in 1981, in which case high temperatures and low levels of rainfall probably accounted for the fact that no disease was observed in May. Because PSS is most likely to be confused with PST (both are fluorescent pseudomonads) these patterns can be helpful in diagnosis and understanding of the two diseases. PST, unlike PSS, was not observed until May, a period when levels of PSS are usually declining. Overall, PSS accounted for 58% of all bacterial disease from 1980 through 1984. Consequently, the ability to diagnose this disease and retain plant certification reduced losses by one-half during that period.

Another concern was that PSS infections might mask the presence of PST in mixed infections. Several lines of evidence indicate that this is not the case. Plants with PSS transported to northern growing areas or as excess plants in Georgia have not displayed problems with bacterial speck developing at a later date. Also, PSS and PST were both detected in laboratory isolations when mixed together even when the ratio of the mixture was 9:1 in favor of one or the other pathogen. In addition, by manipulation of inoculation conditions, PST can be detected in a PSS/PST mixture by a bioassay. When natural infections of PSS were evaluated by the bioassay, PST was not detected. Consequently, mixed infections do not appear to be significantly involved in the epidemiology of the two diseases.

The cultivar response to PSS in terms of quantitative resistance was similar to that reported for bacterial speck (3). In each case, Ohio 7663 and Campbell 28 were highly resistant. It is unlikely that symptom expression or disease severity among cultivars could be used effectively to differentiate PSS from PST infections.

Both wild cherry and rye appear to be potential sources of inocula for PSS infections in tomato. Rye is not only used as a cover crop and turned into the soil but is also used as a protective windbreak between plant beds. Wild cherry trees are common along fences, roadways, and field perimeters in southern Georgia. It is also common to find wild cherry leaves as scattered debris within the transplant bed throughout the season. The close proximity of both of these plants to problem areas and their association with PSS are highly incriminatory as to their role in the disease. Although PSS and PST are similar in their physiological characterization, PSS has a diverse host range as a pathogen and epiphyte (1,15,16). Previous work indicated that PST could not overseason on weeds or in the soil because of the high temperatures found in southern Georgia (12). The data presented here are evidence that PSS, unlike PST, can overseason in the coastal plain area in association with rye and cherry.

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