Resistance in Tomato and Wild Relatives to Crown and Root Rot Caused by *Phytophthora capsici*

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ABSTRACT

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Phytophthora capsici causes root, crown, and fruit rot of tomato, a major vegetable crop grown worldwide. The objective of this study was to screen tomato cultivars and wild relatives of tomato for resistance to *P. capsici*. Four *P. capsici* isolates were individually used to inoculate 6-week-old seedlings (1 g of *P. capsici*-infested millet seed per 10 g of soilless medium) of 42 tomato cultivars and wild relatives of tomato in a greenhouse. Plants were evaluated daily for wilting and death. All *P. capsici* isolates tested caused disease in seedlings but some isolates were more pathogenic than others. A wild relative of cultivated tomato, *Solanum habrochaites* accession LA407, was resistant to all *P. capsici*

isolates tested. Moderate resistance to all isolates was identified in the host genotypes Ha7998, Fla7600, Jolly Elf, and Talladega. *P. capsici* was frequently recovered from root and crown tissue of symptomatic inoculated seedlings but not from leaf tissue or asymptomatic or control plants. The phenotype of the recovered isolate matched the phenotype of the inoculum. Pathogen presence was confirmed in resistant and moderately resistant tomato genotypes by species-specific polymerase chain reaction of DNA from infected crown and root tissue. Amplified fragment length polymorphisms of tomato genotypes showed a lack of correlation between genetic clusters and susceptibility to *P. capsici*, indicating that resistance is distributed in several tomato lineages. The results of this study create a baseline for future development of tomato cultivars resistant to *P. capsici*.

Tomato (*Solanum lycopersicon* L.) is a major vegetable crop grown worldwide. Each year, 160,000 to 280,000 ha of processing and fresh market tomato crops are planted in the United States, with an approximate value of \$2 billion (2). *Phytophthora capsici* Leonian is a destructive soilborne pathogen with a broad host range that includes solanaceous, cucurbitaceous, and fabaceous crops (9). On tomato, the pathogen causes root, crown, and fruit rot (28,54), all of which have been reported in several regions, including Michigan (18), California (4,8,54), Colorado (28), and Florida (52).

In Michigan, tomato fruit are grown for fresh market and processing. Fresh-market production utilizes plastic-covered raised beds, trickle irrigation, and trellising to hold the plants upright; these management practices also limit disease caused by *P. capsici* (M. K. Hausbeck, *unpublished data*). Fresh-market tomato fruit are harvested by hand and have a higher profit margin than processing tomato fruit. Because processing tomato fruit are mechanically harvested and have a lower profit margin, plants are grown on flat ground where fruit typically come into direct contact with the soil. The growing system used by processing-tomato growers is more conducive to *P. capsici* infection than the fresh-market system. In Michigan, crop rotation and fungicide applications are commonly used to manage *P. capsici*. The success of crop rotation is limited by the long-term survival of oospores in the soil (32) and the number and diversity of susceptible hosts (15,45). Applications of the commonly used fungicide mefenoxam may not protect susceptible crops from resistant *P. capsici* populations, which have been documented

throughout the United States (30,31,33,43). Host resistance should be a key component of a disease management strategy for *P. capsici* on tomato. Regrettably, only a few moderately resistant tomato cultivars are available with commercially acceptable horticultural traits (4,20). More sources of resistance from commercial cultivars or wild species need to be identified. Breeders use wild *Solanum* spp. as sources of genes controlling traits of economic importance such as fruit characteristics, nutritional content, and general disease resistance (49). Identifying sources of resistant tomato germplasm would aid in the development of cultivars suitable for production in *P. capsici*-infested fields.

The objective of this study was to determine whether different cultivars of *S. lycopersicon* L., *S. pimpinellifolium*, *S. pennellii*, or *S. habrochaites* show resistance to *P. capsici*. Specifically, we sought to determine (i) whether or not commercial tomato or tomato wild relatives could be used as a source of resistance to *P. capsici*, (ii) whether or not infection and disease development were influenced by the *P. capsici* isolates selected, and (iii) the association between lineages of the cultivars and resistance to *P. capsici*. A preliminary report of these findings has been published (46).

MATERIALS AND METHODS

Tomato cultivars and wild relatives. Forty-two cultivars of tomato and wild relatives (referred collectively as host genotypes) were selected, which included cultivars for fresh market (17 cultivars), processing (17 cultivars), breeding lines (1 cultivar), and 3 wild species (7 accessions) provided by either Dr. D. Francis (The Ohio State University), Redgold, Inc. (Elwood, IN), or Seedway, LLC. (Hall, NY) (Table 1). Seed were planted in 72-square-cell plastic flats with cell depth of 5.7 cm and cell width of 4 cm (Hummert International, Earth City, MO), containing potting soilless media (Baccto Professional Planting Mix; Michigan Peat Company, Houston, TX) and a top layer of medium-grade

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vermiculite (Hummert International). Plugs were grown in a greenhouse at Michigan State University (MSU) for 4 weeks, under approximately 14 h of day illumination. They were transferred into 2.5-liter square plastic pots (Hummert International) containing the same potting soilless media once they reached an approximate height of 8 cm and developed three or four true leaves. Plants were allowed to recover from transplant stress for 2 weeks prior to inoculation. For the duration of the experiment, plants were fertilized weekly with Peter's 20-20-20 water-soluble fertilizer (The Scotts Company, Marysville, OH) at 200 ppm and irrigated as needed to maintain adequate moisture for plant growth and *P. capsici* disease development. Air temperature and relative humidity data (Table 2) were collected hourly with a Watchdog data logger (450 series; Spectrum Technologies, Inc., East-Plainfield, IL).

Isolate selection and maintenance. *P. capsici* isolates originating from diseased cucurbitaceous and solanaceous crops in Michigan were selected from the culture collection maintained in the laboratory of Dr. Hausbeck at MSU. The selected isolates have shown high virulence (defined as the degree of damage

caused to a host by a pathogen) and pathogenicity (defined as the capacity of a pathogen to cause disease on a host genotype) in different hosts (12,14,45,53). Isolates were phenotypically characterized according to mating type (MT) and sensitivity to mefenoxam, an oomycete-specific fungicide (30). Isolates OP97 (A1 MT) and SP98 (A2 MT), from pickling cucumber and pumpkin, respectively, were sensitive to mefenoxam, whereas isolates SFF3 (A2 MT) and 12889 (A1 MT) from pickling cucumber and pepper, respectively, were insensitive.

TABLE 2. Air temperature and relative humidity in the greenhouse during each of the three replicated experiments in which tomato plants were screened for resistance to *Phytophthora capsici*^z

Exp	Air temperature (°C)			Relative humidity (%)		
	Avg	Min	Max	Avg	Min	Max
1	18	8	28	75	50	100
2	19	2	36	83	63	103
3	18	1	35	77	64	90

^z Exp = experiment, Avg = average, Min = minimum, and Max = maximum.

TABLE 1. Disease reaction and mean area under the disease progress curve (AUDPC) and percent death values of three experiments in which tomato cultivars and wild relatives were screened for resistance to *Phytophthora capsici*

Accessions ^y	Disease reaction ^z				AUDPC mean				Death (%)			
	12889	OP97	SP98	SFF3	12889	OP97	SP98	SFF3	12889	OP97	SP98	SFF3
88 (Sl, P)	S	M	S	R	114	39.4	114	0	100	0	100	0
331 (Sl, P)	S	S	R	R	112	114	0	0	100	100	0	0
401 (Sl, P)	S	M	S	R	116	13.1	116	0	100	0	100	0
611 (Sl, P)	S	S	S	R	117	118	116	0	100	86.7	100	0
818 (Sl, P)	S	S	S	S	110	115	114	116	100	100	100	100
7983 (Sl, P)	S	S	S	R	123	107	126	0	100	73.3	100	0
9704 (Sl, P)	S	R	S	R	113	0	116	0	100	0	100	0
BHN444 (Sl, FM)	S	M	S	R	116	18.8	113	0	100	0	93.3	0
BHN591 (Sl, FM)	S	R	S	R	118	0	114	0	100	0	100	0
E6203 (Sl, P)	S	S	S	R	119	115	117	0	100	100	100	0
Fla7600 (Sl, FM)	M	R	R	R	45.4	0	0	0	0	0	0	0
Florida47 (Sl, FM)	S	S	M	R	115	119	22.9	0	100	100	6.67	0
Florida91 (Sl, FM)	S	M	M	R	110	35.6	20	0	100	0	6.67	0
Ha7998 (Sl, W)	M	R	M	R	37.9	0	20	0	6.67	0	6.67	0
Hunt100 (Sl, P)	S	S	R	R	122	120	0	0	100	100	0	0
Jolly Elf (Sl, FM)	M	M	M	R	30.4	25.9	18	0	6.67	0	6.67	0
LA1269 (Sp, W)	S	S	S	S	107	111	56.8	105	100	100	0	100
LA1589 (Sp, W)	S	S	S	S	104	110	112	119	100	73.3	93.3	100
LA407 (Sh, W)	R (C)	R (C)	R (A)	R	0	0	0	0	0	0	0	0
LA716 (Spe, W)	S	S	S	S	131	130	130	126	100	100	100	100
M82 (Sl, P)	S	S	S	S	114	105	113	117	100	80	100	100
Mountain fresh plus (Sl, FM)	S	R	R	R	110	0	0	0	100	0	0	0
Mountain spring (Sl, FM)	S	R	S	R	110	0	118	0	100	0	100	0
NC23E (Sl, FM)	S	S (B)	M	R	117	115	21.8	0	100	100	6.67	0
OH8245 (Sl, P)	S	S	S	R	115	122	51	0	100	100	6.67	0
OH88119 (Sl, P)	S (B)	S (B)	S	R	109	120	106	0	100	100	100	0
OH9241 (Sl, P)	S	S	S	S	116	110	117	120	100	100	100	100
OH9242 (Sl, P)	S	S	S	S	122	117	117	120	100	100	100	100
Peto95-43 (Sl, P)	S	S	S	S	119	122	121	118	100	100	100	100
PI114490 (Sl, W)	S	S	R	R	122	120	0	0	100	100	0	0
PI128216 (Sp, W)	S	S	S	R	121	122	109	0	100	100	100	0
PI365914 (Sp, W)	S (B)	S (B)	R	R	112	114	0	0	100	100	0	0
Plum crimson (Sl, FM)	S	S	M	R	113	120	45.2	0	100	100	13.3	0
Rio Grande (Sl, FM)	S	S	S	S	115	118	120	116	93.3	100	100	100
Sebring (Sl, FM)	S	S	S	R	116	115	118	0	100	100	100	0
Sunbeam (Sl, FM)	S	S	S	R	112	115	57.8	0	100	100	0	0
Sunleaper (Sl, FM)	S	S	S	M	74.7	118	114	26.7	33.3	100	100	0
Sunoma (Sl, FM)	S	S	S	R	107	117	112	0	100	100	100	0
Super sweet 100 (Sl, FM)	S	R	R	R	71.6	0	0	0	13.3	0	0	0
Talladega (Sl, FM)	M	M	M	R	44.4	29.4	38.2	0	40	6.67	13.3	0
TR12 (Sl, P)	S	S	S	S	117	108	111	119	100	100	100	100
TSH4 (Sl, P)	S (B)	S	R	R	115	115	0	0	100	100	0	0
Total	...	...	...	...	103	81.3	71.8	28.6	85.6	64.8	56	23.8

^y Letters in parentheses indicate species and type of the accession. Sl, *Solanum lycopersicon*; Sp, *S. pimpinellifolium*; Spe, *S. pennellii*; Sh, *S. habrochaites*; P, processing; FM, fresh market; W, wild.

^z Disease reaction. S, susceptible; M, moderately resistant; R, resistant. Letters in parentheses indicate tissue types from which *P. capsici* was recovered in the accession. *P. capsici* was recovered from root and crown tissue unless otherwise noted: A, roots; B, roots, crown, and secondary stems; and C, *P. capsici* not recovered.

Agar plugs from long-term stock cultures (stored at 20°C in sterilized microcentrifuge tubes containing 1 ml of sterile water and one sterile hemp seed) of each isolate were transferred onto unclarified V8 juice agar (UCV8; 16 g of agar, 3 g of CaCO₃, 160 ml of unfiltered V8 juice, and 840 ml of distilled water) to obtain actively growing cultures, which were maintained at room temperature (21 ± 2°C) under constant fluorescent lighting. To ensure that isolates could cause disease, cucumber fruit were disinfested for 5 min in a 5% sodium hypochlorite solution, dried at room temperature, and inoculated with each *P. capsici* isolate. A small, superficial wound was made with a sterile needle in the center of each cucumber. Agar plugs (7 mm in diameter) from the margins of actively growing *P. capsici* colonies were placed topside down over each wound. Sterile, capless microcentrifuge tubes were placed over each agar plug and were attached to the fruit with a ring of petroleum jelly. Control cucumber fruit were inoculated as described above using a sterile 7-mm-diameter plug of V8 agar. Cucumber fruit were placed in aluminum trays containing wet paper towels and covered with plastic wrap to maintain high relative humidity and incubated at room temperature (21 ± 2°C). Symptomatic cucumber tissue (0.5 cm) was excised and transferred to UCV8 and maintained under the same culture conditions as described above. Axenic cultures of each isolate were obtained from the infected cucumber tissue and these were transferred to new UCV8 plates weekly until use.

Inoculum preparation and root inoculation. *P. capsici*-infested millet seed was prepared as inoculum. Millet seed (100 g) was mixed with L-asparagine (0.08 g) and water (72 ml) in a 500-ml Erlenmeyer flask, capped with aluminum foil, autoclaved for two consecutive cycles, and shaken to homogenize the mixture. The sterilized millet seed was inoculated with four 7-mm-diameter agar plugs from actively growing *P. capsici* cultures. The inoculated millet seed was incubated at room temperature under constant fluorescent lighting for 4 weeks.

Inoculation of the plants was achieved by carefully inserting 1 g of millet seed infested with one of four *P. capsici* isolates (OP97, SFF3, SP98, and 12889) directly into the soilless media adjacent to each plant crown, avoiding root or crown injury. For each isolate, five replicate plants of each tomato genotype were inoculated. Five additional control plants of each tomato genotype were inoculated with uninfested millet seed containing sterile V8 agar plugs. This experiment was conducted three times.

Disease assessment. Disease assessment was conducted daily for 5 weeks and was initiated 1 day following inoculation. Disease progression was recorded for each plant using the following scale: 0 = no symptoms, 1 = 1 to 30% wilting, 2 = 31 to 50% wilting, 3 = 51 to 70% wilting, 4 = 71 to 90% wilting, and 5 = >90% wilting or dead plant (Fig. 1). The area under the disease progress curve (AUDPC) was calculated by inserting the disease score into the equation by Shaner and Finney (56) to describe the cumulative plant susceptibility throughout the experiments. The percentage of death and the incidence of *P. capsici* recovered from roots, crowns, secondary stems (equivalent of branches in a woody plant), and leaves were determined. The plant height was measured at the end of the experiment for host genotypes that appeared resistant based on AUDPC values. Tomato genotypes were considered to be resistant to *P. capsici* if disease caused by the four isolates used in our study did not result in serious damage to the plants, as indicated by mean AUDPC values of three replicated experiments. Resistant, moderately resistant, and susceptible genotypes to each *P. capsici* isolate included those with mean AUDPC values <10, 10 to 50, and >50, respectively.

Pathogen isolation. Control and inoculated plants were gently rinsed to remove the soilless medium and other residues, dipped into 5% sodium hypochlorite for 1 min to surface disinfect, rinsed with sterile water, and air dried. Three sections of root, crown, secondary stem, and leaf tissue were excised from each plant and

plated onto benomyl, ampicillin, rifampicin, and pentachloro-nitrobenzene (BARP)-amended UCV8 (32) under sterile conditions.

Colonies suspected to be *P. capsici* were transferred to new BARP-amended UCV8 plates. Axenic cultures were incubated for 7 days on UCV8 with constant fluorescent lighting under ambient laboratory conditions (21 ± 2°C). Cultures were positively identified as *P. capsici* based on morphological characteristics described by Waterhouse (64). Isolates were characterized for compatibility type and mefenoxam resistance as previously described (30) to compare the phenotype of the isolate obtained with the original inoculum.

DNA extraction and molecular confirmation of infection.

Samples of root and crown tissue from inoculated resistant and moderately resistant tomato genotypes were collected at the end of the experiment, gently rinsed to remove the soilless medium and other residues, dipped into 5% sodium hypochlorite for 1 min to surface disinfect, rinsed with sterile water, air dried, and ground in liquid nitrogen. Genomic DNA was extracted from approximately 1 g of ground tissue using the DNeasy DNA extraction kit (Qiagen, Valencia, CA) according to the manufacturer's instructions. DNA was quantified using the NanoDrop ND 1000 spectrophotometer and NanoDrop 2.4.7c software (NanoDrop Technologies Inc., Wilmington, DE). Two specific primers for *P. capsici* were used for polymerase chain reaction (PCR): one forward primer (CAFW; 5'TTTAGTTGGGGTCTTGTACC3') and one reverse primer (CAPRV2; 5'TACGGTTCACCAGC CCATCA3') (45). Reactions were performed in a total volume of 25 µl and contained 1 µl of DNA, 5 µl of 5× PCR reaction buffer, 1.25 µl of 25 µM MgCl₂, 0.5 µl of 10 µM dNTP mix, 0.7 µl of *Taq* DNA polymerase (Invitrogen, Carlsbad, CA), 1 µl each of 10 µM primer (MSU Macromolecular Structure Facility, East Lansing, MI), and 14.6 µl of sterile water. The PCR reaction was performed in a programmable thermal cycler (Eppendorf, Westbury, NY) starting with 3 min of denaturation at 94°C; followed by 45 cycles at 94°C for 30 s, annealing at 56°C for 30 s, and extension at 72°C for 60 s; with a final extension step of 10 min at 72°C. PCR products were analyzed by electrophoresis in 2% (wt/vol) agarose gel in 0.5× Tris-borate-EDTA buffer (35), stained with ethidium bromide (5 µg/ml) for visualization and compared with a 100-bp ladder (Invitrogen) to determine amplicon size. Controls with *P. capsici* DNA and tomato DNA were included.

Amplified fragment length polymorphism analysis. Samples (1 g) of 4-week-old healthy tomato tissue were collected and ground in liquid nitrogen. Genomic DNA was extracted and quantified as described above. All PCR reactions were performed with a programmable thermal cycler (Eppendorf). DNA (~500 ng) was subjected to a restriction/ligation reaction, preselective amplification, and selective amplifications using the amplified fragment length polymorphism (AFLP) kit for regular plant genomes (Applied Biosystems, Foster City, CA) according to the manufacturer's instructions. Selective amplifications with the selective primers *Mse*I-CAA and *Eco*RI-ACA, and *Mse*I-CAA and *Eco*RI-AAG, were performed. Selective PCR products were purified with ExoSAP-IT (Affymetrix, Inc., Santa Clara, CA) following the manufacturer's instructions. Products labeled with different colored dyes were analyzed at the MSU Research Technology Support Facility (RTSF) using the ABI PRISM 3130 Genetic Analyzer and compared with a 500-carboxy-X-rhodamine (ROX) size standard (Applied Biosystems) following the manufacturer's instructions. Results were prepared for analysis by the RTSF in the form of electropherograms using GeneScan 3.1 analysis software (Applied Biosystems). AFLP fragments were scored manually as present = 1 or absent = 0 using PeakScanner 1.0 (Applied Biosystems). Only DNA-labeled fragments with a size ≥70 bp were scored to obtain a binary character matrix. Once fragments of appropriate size were identified, only labeled fragments with a fluorescence signal ≥300 relative fluorescence units (rtf) in at

least one cultivar were scored for presence or absence in the rest of the cultivars. The binary matrix, consisting of combined data of both primer sets, was analyzed to generate an unweighted pair-group method with arithmetic mean (UPGMA) distance tree and a pairwise genetic distance matrix using PAUP*4.0b10 (60).

Statistical analyses. All statistical analyses were performed using the SAS statistical package (version 9.1; SAS Institute Inc., Cary, NC). The experiment was arranged in a split-plot with experiment as a blocking factor, isolates as whole plots, and host

genotypes as subplots arranged in a randomized complete block design. The AUDPC, incidence, and plant height values of the three replicated experiments were used for statistical analyses. AUDPC data from control plants were removed from the data set prior to statistical analyses because no infection occurred and no symptoms developed. AUDPC and plant height data were normalized by square root transformation, and the residuals followed the assumptions of all statistical tests performed. The percent death was not included for statistical analysis because the residuals did

Fig. 1. Symptoms of *Phytophthora capsici* root rot on tomato and wilting scale used in plant evaluations. A, 0 = no symptoms, healthy plant; B, 1 = 1 to 30% wilting; C, 2 = 31 to 50% wilting; D, 3 = 51 to 70% wilting; E, 4 = 71 to 90% wilting; and F, 5 = more than 90% wilting or dead plant. G, Stem canker. H, Water-soaked lesions in the stem, rotted crown, and rotted and discolored roots. I, Secondary roots. J, Girdling. K, Damping-off. L, Stunting.

not follow the assumptions required for statistical tests. Data were subjected to analysis of variance (ANOVA) using the PROC MIXED, PROC GLM, and PROC GLIMMIX procedures of SAS. Correlation between UPGMA cluster (defined by the genetic distance matrix) and resistance to *P. capsici* was determined using the PROC CORR procedure. Multiple comparisons among the means were conducted using *t* tests (least significant difference [LSD]) when effects were found to be statistically significant at $P = 0.05$ in ANOVA.

RESULTS

Initial disease symptoms occurred within 4 days following inoculation in susceptible and some moderately resistant tomato genotypes for each *P. capsici* isolate. In susceptible genotypes, wilting was observed in the lower leaves within the first week and progressed upward until the entire plant wilted. External, dark discoloration of the crowns and crown rot were observed on some plants and the root system was often discolored. Subsequently, stem cankers and girdling developed, and some plants shriveled at the soil level (Fig. 1). Brown, water-soaked lesions were visible on roots and crowns of symptomatic plants within 6 days. Plant death was observed as early as 7 to 8 days following inoculation in some host genotypes but was not visible until 4 weeks after inoculation for others. Moderately resistant cultivars showed mild wilting 7 to 8 days following inoculation but quickly recovered. Some plants continued to grow and maintain healthy tops by regenerating an extensive number of secondary roots (Fig. 1). Resistant genotypes did not show wilting after inoculation or production of secondary roots. Some resistant and moderately resistant host genotypes also presented significant stunting.

Disease severity differed significantly depending on the interaction effects between isolate and tomato genotype (ANOVA, $P < 0.0001$). Responses to individual isolates varied from no symptoms to 100% wilting and death of inoculated plants. Isolate 12889 was more pathogenic on tomato genotypes than other isolates used in plant inoculations according to trends in AUDPC and percent plant death (Table 1). Interaction effects were not found between repeated experiments and isolate (ANOVA, $P = 0.5178$). Effects due to differences between experiments were not significant (ANOVA, $P = 0.2926$). Only one of the genotypes (LA407) did not show wilting or any other symptoms other than stunting following inoculation with the four different *P. capsici* isolates (Table 1). Four of the host genotypes (Fla7600, Ha7998, Jolly Elf, and Talladega) developed moderate symptoms with <12% plant death (Table 1). LA716 was the most susceptible genotype in our study, exhibiting the highest AUDPC values and 100% plant death (Table 1).

The pathogen was isolated from all surviving susceptible, moderately resistant, and resistant genotypes at the end of the experiment and from plants that died over the course of the experiment. *P. capsici* was isolated from symptomatic plants of the moderately resistant genotypes Fla7600, Ha7998, Jolly Elf,

and Talladega and from the roots of the resistant genotype LA407 (Table 1). *P. capsici* infection in the moderately resistant and resistant genotypes was confirmed by performing *P. capsici*-specific PCR of DNA from surface-sterilized, inoculated root and crown tissue to confirm that the very low recovery of *P. capsici* from LA407 by culturing techniques was not due to an escape. All *P. capsici*-inoculated moderately resistant and resistant genotypes were positive for presence of *P. capsici* in root or crown tissue. Significant differences in pathogen incidence were found among different plant parts (PROC GLIMMIX and LSD, $P < 0.0001$). Interaction effects were found between the isolate used and tomato genotype for incidence data (ANOVA, $P < 0.0001$). Interaction effects were not found between experiments and isolate (ANOVA, $P = 0.9994$). Effects due to differences between repeated experiments were not significant (ANOVA, $P = 0.9442$). Although the secondary stems and leaves exhibited wilting in all experiments, the pathogen was rarely recovered from secondary stems and never recovered from leaves (Table 3). The phenotype (mating type and mefenoxam resistance) of recovered isolates was confirmed and matched the phenotype of the isolate used as inoculum (data not shown). The control plants remained asymptomatic and the pathogen was not isolated from any control plant tissue. Clean cultures were obtained from 94% of the original cultures isolated from tomato tissue.

Stunting was observed in several tomato genotypes, including some of the moderately resistant and resistant candidates (Fig. 1L). Plant height was measured for surviving inoculated moderately resistant and resistant genotypes and the corresponding control plants for those host genotypes at the end of the experiment. Generally, plants inoculated with one of the four *P. capsici* isolates were significantly shorter than the corresponding control plants, although the interaction between isolate and tomato genotype was significant (ANOVA, $P < 0.0001$). Pairwise comparisons between each tomato genotype inoculated with a particular isolate and the corresponding control plant indicated that genotypes Fla7600 and Jolly Elf did not show significant differences in plant height regardless of isolate (LSD, $P > 0.05$) (Table 4). Interaction effects were not found between repeated experiments and isolate (ANOVA, $P = 0.5478$). Effects due to differences between experiments were not significant (ANOVA, $P = 0.0600$).

TABLE 4. Probability (*P*) values for significance of differences in plant height between inoculated plants and their corresponding controls for tomato genotypes resistant and moderately resistant to *Phytophthora capsici*

Cultivar	<i>P</i> value ^z			
	12889	OP97	SP98	SFF3
Fla7600	0.8164	0.5798	0.0744	0.5324
Ha7998	0.0093*	0.0004*	<0.0001*	<0.0001*
LA407	0.0402*	0.0048*	<0.0001*	<0.0001*
Jolly Elf	0.0745	0.1681	0.1567	0.5653
Talladega	<0.0001*	0.0327*	0.0002*	0.8062

^z Asterisk (*) indicates means significantly different from the control (least significant difference); analyses included data from three experiments.

TABLE 3. Incidence of *Phytophthora capsici* isolates obtained from different parts of the symptomatic tomato plants sampled in each of three replicated experiments

Experiment	No. of plants ^v	No. of isolates ^w	Isolates obtained (%) ^{xy}		Incidence of <i>P. capsici</i> (%) ^{xz}			
			Root	Crown	Root	Crown	Secondary stem	Leaf
1	570	910	470 (51.6)	440 (48.4)	488 (50.7)	471 (48.8)	5 (0.5)	0 (0)
2	583	903	458 (50.7)	445 (49.3)	483 (50)	479 (49.6)	4 (0.4)	0 (0)
3	579	890	459 (51.6)	431 (48.4)	488 (51.3)	458 (48.1)	6 (0.6)	0 (0)

^v Number of symptomatic plants sampled.

^w Total number of isolates.

^x Numbers in parentheses correspond to percentage values and numbers not in parentheses are the raw values.

^y As determined by axenic pathogen isolation at the conclusion of the experiment.

^z As determined by observation of growth of the pathogen isolated from plant tissue.

The *Mse*I-CAA and *Eco*RI-ACA, and *Mse*I-CAA and *Eco*RI-AAG, primer pairs yielded a total of 104 and 120 AFLP markers, respectively. Of the total 224 AFLP markers obtained, 126 corresponded to labeled fragments with a size ≥ 70 bp and a signal ≥ 300 rtf that were scored for presence and absence in the tomato genotypes and included in the analyses. In all, 4 of these markers were polymorphic only among *Solanum* spp. (2% of interspecific polymorphism), 31 markers had variation among *Solanum* spp. and *S. lycopersicon* cultivars (14% of inter and intraspecific polymorphism), 11 markers were polymorphic only for *S. lycopersicon*

cultivars (5% of intraspecific polymorphism), and the other 80 markers were present in all tomato genotypes (79% monomorphic). Seven similarity groups, including four main clusters (A, B, C, and F) and three clades formed by single genotypes (D, E, and G) with $>80\%$ similarity, were obtained after genetic distance analysis of the AFLP data (Fig. 2). The UPGMA tree topology does not indicate a subdivision of clusters based on susceptibility to *P. capsici*. Statistical analysis found no significant correlation between cluster and susceptibility to *P. capsici* ($P > 0.05$).

Fig. 2. Amplified fragment length polymorphism distance tree of tomato cultivars and wild relatives screened for resistance to *Phytophthora capsici*. Susceptibility to *P. capsici* 12889, OP97, SP98, and SFF3 (S = susceptible, M = moderately resistant, and R = resistant), tomato species (SPP: SI = *Solanum lycopersicon*, Sp = *S. pinpinellifolium*, Spe = *S. pennellii*, and Sh = *S. habrochaites*), and type of cultivar (TYP: F = fresh market, P = processing, and W = wild) are indicated for each cultivar. A, B, C, D, E, F, and G, correspond to similarity groups obtained in unweighted pair-group method with arithmetic mean clustering analysis.

DISCUSSION

Early infections by *P. capsici* on tomato cause plant damping-off, whereas later infections cause root and crown rot on mature plants (21). Phytophthora root rot results in wilting with brown-to-black cankers on the lower stems, blackening of the vascular tissue, and root rot (29), which may initiate the development of secondary roots (4). Infected plants may become stunted and wilted (21), as they did in our study. Plants showing quantitative resistance to Phytophthora root rot have been reported to develop and maintain healthy canopies by regenerating an extensive number of secondary roots, a response that is most likely polygenic (4). In this experiment, several host genotypes produced secondary roots that emerged just above a stem canker or crown rot lesion but many of them still died, possibly because the response was not fast enough. The moderately resistant genotypes Talladega and Jolly Elf produced secondary roots in some cases and maintained healthy canopies throughout the duration of the experiment. The resistant cv. LA407 did not show this response. Symptoms developed more slowly on the moderately resistant genotypes Fla7600, Ha7998, Jolly Elf, and Talladega than on the other genotypes tested. In general, the response of moderately resistant tomato genotypes to Phytophthora root and crown rot was quantitative rather than qualitative, as observed in the *P. capsici*–pepper interaction (26). In some cases, such as the resistant genotype LA407 or the susceptible genotype LA716, the response is more qualitative because there appeared to be no disease in LA407 plants but all LA716 plants died.

The success of greenhouse screening depends on the plant's age and resistance level, inoculum quality or quantity, inoculation technique, and post inoculation environmental conditions. These factors may influence quantitative responses such as resistance to *P. capsici*. For this study, environmental conditions in the greenhouse were not always ideal for plant and pathogen growth due to low minimum air temperatures (1 to 2°C) and high maximum air temperatures (35 to 36°C) in experiments 2 and 3. The minimum, optimum, and maximum temperatures for growth of *P. capsici* are 10, 28, and >35°C, respectively (9). Tomato plants prefer warmer temperatures (18 to 26°C) for normal plant development, growth, and fruit set (24), and become sensitive to biotic and abiotic stress at temperatures <10°C or >35°C (24). Nonetheless, environmental conditions did not seem to alter disease development and average air temperatures were suitable for plant and pathogen growth. Furthermore, there were no statistically significant interactions with experiment; therefore, any effects on disease development appear to have been constant across isolates and host genotypes.

Resistance to *P. capsici* under greenhouse conditions may differ from the responses observed under field conditions due to the quantitative nature of the trait. The soilless medium used in this study may have influenced resistance to *P. capsici*, and the responses observed in the greenhouse may not be consistent in the field, depending on soil type, soil microbial community composition, pH, and other factors. Other studies on *P. capsici* (27) and other soilborne pathogens (16,39) have shown variable results, with observations of increased or decreased disease severity depending on the soil type, soilless substrates, or treatments used. To our knowledge, none of these studies have determined the influence of soilless medium on *P. capsici*. However, if soilless medium effects on *P. capsici* exist, they were consistent throughout the experiments and did not appear to inhibit disease development.

The type and quantity of inoculum used can also affect disease severity in greenhouse and field studies (14,15,45). The millet-seed inoculation technique was effective for establishing *P. capsici* infection on susceptible tomato genotypes in the greenhouse, as has been reported in previous studies with other host plants (12,45). The millet-seed inoculum for our experiments contained

only mycelia and sporangia, whereas Michigan fields infested with *P. capsici* have been found to contain oospores (30,31). Oospores allow the pathogen to overwinter, may persist for years (18,32), and are primary inocula (18). For our experiments, we used *P. capsici* propagules that constitute secondary inocula and cause tomato field epidemics (21). Studies replicating our greenhouse experiment in *P. capsici*-infested fields would help establish the influence of inoculum type on disease severity as well as the transferability of greenhouse results to field conditions.

All *P. capsici* isolates tested caused disease on some plants, although there was a significant isolate–tomato genotype interaction. Isolate 12889 produced symptoms in more host genotypes, followed by OP97, SP98, and SFF3. When these isolates were tested on pepper in an earlier study (15), 12889 was also found to be the most pathogenic; however, no differences were detected when the isolates were tested on cucumber fruit (14) and Fraser fir (45). As early as 1972, field populations of *P. capsici* from solanaceous and cucurbitaceous hosts were shown to differ in pathogenicity (44); other reports of this variation have been published for several hosts (5,26,40,41,48,50), including tomato, where an interaction between host and isolate was also reported (17,20). It has been suggested in previous studies that *P. capsici* isolates obtained from the family Solanaceae have increased pathogenicity on Solanaceous hosts compared with isolates obtained from cucurbits (19,34,50). Further studies that include more isolates are needed to clarify whether *P. capsici* 12889 has increased pathogenicity in tomato and pepper genotypes because it was isolated from another solanaceous host or whether the pathogenicity is due to an isolate-specific effect.

The differences in pathogenicity found between these Michigan isolates have significant implications for local plant breeding programs. Breeding for resistance in pepper against *P. capsici* has been challenging due to the diversity of pathogen populations, the existence of different physiological races within *P. capsici* in the United States (40,63), and the various complex modes of inheritance of resistance reported for pepper (47). Similarly, for tomato, studies are needed to characterize *P. capsici* populations, define races that infect tomato, and determine their spatial structure in order to intelligently deploy tomato resistance in Michigan. Nonetheless, in this study, tomato genotypes LA407, Fla7600, Ha7998, Jolly Elf, and Talladega showed high or moderate resistance to all of the isolates tested and could be used as a starting point to find resistance to Michigan *P. capsici* isolates.

Our data indicated that the four isolates tested probably represent different races. Nonetheless, the tomato genotypes should be tested against a larger number of isolates, particularly those obtained from tomato, to better understand this interaction and to eventually develop a system with host differentials for identifying races, as has been described for pepper (62). No resistant control was used in our study because, to the best of our knowledge, there is no standard tomato resistant to *P. capsici*. When screening pepper cultivars for resistance to *P. capsici*, the χ^2 test has been used to determine relative resistance of pepper lines by comparison with standard resistant peppers such as CM334 (62). Our experiments identified the resistant genotype LA407; thus, a similar approach to the one used in the *P. capsici*–pepper interaction can now be applied to future *P. capsici*–tomato interaction studies.

Significant differences in pathogen incidence were found among different plant parts, possibly indicating that more than one mechanism of host resistance may be involved. In pepper, it has been observed that resistance to *P. capsici* is due to different genetic controls depending on tissue type (61,63). In our study, the pathogen was successfully recovered from roots and crowns of all symptomatic, root-inoculated seedlings. *P. capsici* can be difficult to isolate from certain tissue types, such as mature pepper stems (M. K. Hausbeck, unpublished data), asparagus (55), or Fraser fir tissue (45), but was easily isolated from

symptomatic tomato plants in our experiments. However, PCR was useful because it confirmed that the inability to detect *P. capsici* in LA407 using culture techniques was not due to an escape. In previous studies, PCR was able to detect false negatives after a lack of *P. capsici* detection by culturing techniques in pepper plants when symptoms and signs were not obvious (51). Genotypes Ha7998 and Talladega were both stunted as a result of *P. capsici* infection; yet, when Talladega was inoculated with SFF3, the least pathogenic isolate in our study, the plants did not show stunting. Stunting in resistant and moderately resistant genotypes indicates that the pathogen was directly limiting plant growth. However, it is not known whether stunting would occur in nature on a resistant genotype or was the result of the relatively axenic conditions of the experiment.

Wild species of tomato can be a valuable source of economically important traits, including resistance to diverse pathogens for the improvement of cultivated tomato crops (10). In our study, we found that *S. habrochaites* LA407 was resistant to the four *P. capsici* isolates used. Interestingly, LA407 was resistant to bacterial canker caused by *Clavibacter michiganensis* subsp. *michiganensis* (13,25) and early blight caused by *Alternaria solani* (11). Other genotypes of *S. habrochaites* were resistant to late blight caused by *P. infestans* (6,7), whitefly infestations (38), whitefly-transmitted *Geminivirus* spp. (36), and *Bemisia argentifolli* (38). This wild species can be crossed with *S. lycopersicon* to generate progeny with relative ease (6,7,13), making the species a good candidate for improving tomato disease resistance.

When screening for economically important traits, breeders have performed host samplings under the assumption that taxonomically related plants, or those found in geographic proximity, are likely to share desirable characteristics such as disease resistance (22,23,57). The third objective of our study was to determine whether resistance to *P. capsici* could be found in diverse tomato lineages or whether it was only associated with one or more particular clades. If a breeder can use taxonomic or geographic information to identify the most likely sources of valuable traits, then the efficiency of the search may be improved (22). To determine the relatedness of the tomato genotypes used in this study, we relied on AFLP analysis. AFLP analysis is an appropriate tool to detect polymorphisms in tomato and establish genetic diversity and relatedness among tomato genotypes (42, 59). The obtained UPGMA tree topology agreed with clustering analysis of previous studies (1,37,42,59) and the statistical analysis found no correlation between UPGMA clade and resistance to the pathogen isolates used in this study, indicating that the tree topology obtained cannot be reliably used to predict where additional sources of *P. capsici* resistance may be found. Resistance to isolate SFF3 seems to be more widespread across tomato lineages than for the other *P. capsici* isolates used in our experiment. Spooner et al. (58) also found that resistance to *P. infestans* was not associated with lineages of wild potato. Our results suggests that tomato germplasm needs to be screened for resistance to diverse *P. capsici* isolates until other methods, such as marker-assisted selection, are developed to identify resistant genotypes. The association between *P. capsici* resistance and geographic origin of tomato genotypes was not analyzed in this study because the origin of most cultivars is unknown. Information on geographic origin is only available for LA407, that comes from Ecuador; Ha7998, that comes from Hawaii; and LA1269, LA1589, and LA716, that come from Peru. More accessions of the wild species used in this study need to be included in future experiments to establish whether the responses observed occur in other accessions of the same species, and to determine whether the lack of association between resistance to *P. capsici* and AFLP clades is due to the low number of *Solanum* accessions tested or to genetic effects.

P. capsici has been documented in many vegetable production regions in Michigan (18), a state that grows over 33,000 ha of

susceptible crops (3). In Michigan, a grower's ability to grow vegetables in uninfested land is diminished due to a general reduction in farmland caused by urban sprawl and an increase in the number of *P. capsici*-infested fields. In addition, fungicide resistance, an increasing list of susceptible hosts, and the phasing out of methyl bromide treatments make cost-effective management of *P. capsici* difficult. Disease management strategies that reduce fungicide use without increasing disease related losses, such as host resistance, are needed.

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