Survival of *Pyrenochaeta lycopersici* and the Influence of Temperature and Cultivar Resistance on the Development of Corky Root of Tomato

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ABSTRACT

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Behavior of *Pyrenochaeta lycopersici* in soil, root debris, and living roots was studied with the use of a semiselective medium and extraction of propagules by blending and wet-sieving. Abundant propagules (up to 54,000 per gram of tissue) were found in the cortex of tomato root debris 2–8 mo after a tomato crop was harvested. Propagules were recovered from pieces of root buried for 33 mo, the longest period tested. The fungus was unable to colonize tomato root tissue buried in naturally infested soil or in vermiculite infested with microsclerotia produced in vitro. Symptomless infection, which occurred on tomato and *Solanum* spp., may result in extensive invasion and formation of propagules by harvest. Thus, *P. lycopersici* is an ecologically obligate parasite that survives in root tissue as dormant propagules (microsclerotia). Effects of environment on infection and symptom expression were studied at constant temperatures in controlled environment chambers and in the field. The disease was more severe at constant temperatures of 16 or 21 C than at 27 C. Cool temperatures stimulated lesion expansion and symptom development, rather than the initial infection process, when plants were incubated under different combinations of temperatures. When resistant and susceptible tomatoes were planted in naturally infested soil in microplots at monthly intervals, disease progress was linear for all planting dates and both cultivars, but the rate of disease increase was greater for the susceptible than for the resistant cultivar and for the early planting (February) than for the late planting (May). Cool temperature, particularly during the first few weeks of growth of seedlings, probably has a significant effect on increasing disease severity, and this effect is not overcome as seasonal warming occurs later.

Pyrenochaeta lycopersici R. Schneider & Gerlach causes corky root of tomato and reduces yield of greenhouse tomatoes in Europe (7) and field-grown tomatoes in Florida (19) and California (4). Symptoms of the disease include cortical lesions on larger roots and decay of the fine rootlets. The fungus is an obligate primary parasite and a poor saprophytic competitor compared to species of *Fusarium*, *Colletotrichum coccodes* (Wallr.) S. J. Hughes, and *Rhizoctonla solani* Kühn (5,6). Microsclerotia of *P. lycopersici* are formed within infected cortical cells and in culture (2,3,14,20). Microsclerotia survive in root pieces buried in soil for 43 wk (9) and probably are the primary form in which the fungus survives in soil. In addition to tomato, *P. lycopersici* infects *Datura stramonium* L., spinach, safflower, pepper, eggplant, cucumber, squash, and melon (9,15,18) and has been isolated from symptomless beet roots (9). The potential contribution of these hosts to the long-term survival of the pathogen has not been investigated. A semiselective medium that permits the reliable recovery of the corky root fungus from both soil and plant tissue was developed by Grove and Campbell (9).

Soil temperature appears to play an important role in the development of corky root. Disease was severe at constant temperatures of 15–20 C (18). Further, early-planted tomatoes in California had more disease than late-planted tomatoes, perhaps because of cool soil temperature during the early stages of tomato growth (4). Detailed field studies relating natural temperature regimes to disease development have not been reported. Furthermore, the effects of temperature on infection and on lesion expansion have not been studied.

The present study evaluated a modification of the Grove and Campbell procedure for quantification of *P. lycopersici* in soil, in infected living roots, and in root debris. This procedure was used to study 1) the distribution and survival of the pathogen in soil and root tomato debris during the first year following tomato culture in an infested field, 2) the long-term survival of the corky root fungus in buried root pieces, 3) the role of solanaceous weeds and symptomless infections of tomato roots in maintenance of the pathogen in soil, and 4) the ability of the pathogen to colonize tomato roots saprophytically. Additionally, we evaluated the influence of temperature (in microplots and controlled environments) and of cultivar resistance on infection and rate of disease increase.

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MATERIALS AND METHODS

Naturally infested soil. A naturally infested field near Patterson, California, was the source of soil and tomato root debris for this study. Samples of soil and root debris from the 1987 tomato crop were collected randomly from November 1987 to October 1988 from an area of about 500 sq m in one corner of the field. In addition to the random soil sample, a rhizosphere sample of soil in which the tomato root debris was embedded was collected. Three replicate extractions of microsclerotia were done from each sample. The field was planted to dill in March 1988 and to celery in the fall of 1988.

Isolation from soil, debris, and plant tissue. The procedure for isolating microsclerotia of *P. lycopersici* from soil, root tissue, or debris was modified from Grove and Campbell (9). A sample was added to 200 ml of 0.525% NaOCl in a Waring blender and mixed at high speed for 2.5 min. The mixture was then poured into a 38-μm-diameter mesh sieve, washed for 3–5 min with tap water, and backwashed into 100 ml of sterile, distilled water. One-half millileter of this solution was spread on each of five to seven plates of semiselective corky root medium (CRM; 9) and incubated for 2 wk. Corky root colonies were identified on this medium as described (9) and expressed as the number of colonies per gram of substrate. Representative colonies were transferred to plates of sporulation medium that were incubated at 20 C with a 16-hr photoperiod to induce sporulation (9,13) and confirm the tentative identification of the fungus. Root debris was usually dissected into cortical and stelar tissue, and 0.2-g (air-dry weight) samples of each tissue were processed separately. Cortical tissue from living plants was dissected into diseased or symptomless segments that were processed separately. The air-dry weight of these samples was estimated from the moisture content of comparable material dried in an oven at 30 C for 3–4 days. The roots of some small seedlings were weighed and processed on a fresh weight basis. Soil samples were 5 g

(air-dry weight). Sometimes soil or debris extracts were washed in two nested sieves with openings of 38 and 250 μm. In this case, each sieve was backwashed into a separate beaker, plated onto CRM, and the final colony counts consisted of the total from both sieves.

Pathogenicity tests. Inoculations to test the pathogenicity of isolates or to compare the susceptibility of hosts were done in pot culture experiments. Microsclerotia of *P. lycopersici* (isolates 80-55 or 5MC) were prepared as described elsewhere (17) and used to infest vermiculite to give 1×10^4 microslerotia per milliliter of vermiculite. The control was vermiculite to which an equivalent volume of sterile, distilled water was added. Vermiculite was placed in 10-cm-diameter pots or in 270-ml micropots made from plastic cups with holes punched in the bottom. Seeds of tomato cv. VF 6203, susceptible to corky root, or Moboglan, a resistant cultivar (12), were sown and covered with additional vermiculite. Plants were grown for 8 wk in a greenhouse with temperatures ranging from 16–18 C to 27–35 C (minimum and maximum, respectively) or for 4–5 wk in controlled environment chambers. There were three to four replicate pots per treatment. For experiments in controlled environment chambers, repetitions were made over time, with one pot per treatment in each trial. Susceptibility of *Solanum nodiflorum* Jacq., *S. nigrum* L., and *S. sarachoides* Sendt. was also determined.

Disease severity was rated on a 0–4 scale modified from Campbell, Schweers, and Hall (4) where 0 = no symptoms, 1 = 1 to a few lesions on <10% of the taproot, 2 = small lesions that did not coalesce on 10–25% of the taproot, 3 = numerous lesions on 25–75% of the taproot, and 4 = >75% of taproot with lesions.

Saprophytic ability. The corky root fungus was tested for the ability to colonize dead tomato root tissue. The roots of healthy tomato plants grown in vermiculite in a greenhouse for 4 mo were washed free of vermiculite. These roots were buried in 10-cm-diameter clay pots filled with one of the following: sterile vermiculite, vermiculite infested with microsclerotia of *P. lycopersici* isolate 87-26, pasteurized Yolo clay loam, or naturally infested soil from Patterson. There were three pots per treatment. One replicate was dug up 1 mo after burial and two were dug up after 2 mo. The root debris was weighed and assayed for *P. lycopersici* as described.

Microplots. Microplots were installed in November 1987 at a site in the departmental experimental field on the Davis campus to study disease development in field conditions. Microplots consisted of plastic irrigation pipe 29 cm long × 30.5 cm in diameter and placed vertically in 26.5-cm-deep holes. There were 20 microplots spaced 1 m apart in each of two rows spaced 3 m apart. The experiment was a randomized block design in which the main treatment was infested vs. noninfested soil and the secondary treatment was planting date in 1988 (9 February, 8 March, 6 April, and 4 May) for each soil. Each of the five blocks consisted of four microplots from each row. After treatments were randomly assigned within blocks, microplots were filled to 2.5 cm below the top either with a naturally infested soil collected near Patterson or with noninfested soil from the site.

A split-plot treatment within each microplot was used to compare cvs. Moboglan and VF 6203. About 100 seeds of Moboglan or VF 6203 were sown in opposite halves of each microplot. The plots were watered by hand-sprinkling until the seedlings emerged and as needed thereafter. The plots were fertilized before and after the first bloom stage by drenching with 1.2 L of half-strength Hoagland's solution per plot. Tomato plants from each planting date were sampled for disease severity at the two-leaf stage, the four- to eight-leaf stage, at flowering, and at first fruit ripening. The third and fourth plantings were also sampled once between flowering and fruit ripening. Approximately equal numbers of plants were harvested from infested and noninfested soil at each sampling time; the numbers ranged from three to 15 plants with the higher numbers being taken at the early seedling stages. Root systems were rated individually for corky root severity and an average for the sample was calculated. A disease severity of 0 was assigned arbitrarily to the date of seedling emergence. Standard climatological air temperatures (mean = maximum + minimum/2) were obtained from the University of California Integrated Pest Management data base for the Davis weather station located approximately 2 km from the site. The weather station and the microplots were similarly situated in agricultural fields on level terrain. The threshold temperature for degree day calculations was 10 C (1) and the reference period for the 30-yr average was 1951–1980.

Statistical tests. Testing of data for normal distribution, analysis of variance, Duncan's multivariate analysis, analysis of linear and quadratic components of disease progress curves, and comparisons of slopes were done on the Statistical Analysis System (SAS; 16) program using the General Linear Models (GLM) procedure. Because disease severity was rated for each plant and average disease per pot was calculated, categorical data approximated random normal data, allowing use of analysis of variance.

RESULTS

Survival and distribution of *P. lycopersici* in tomato roots and soil. Tomatoes in a commercial crop growing in the infested field near Patterson had only a few lesions of corky root in June 1987 when the plants had set fruit even though severe symptoms were observed on tomatoes grown in this field in 1978 and 1982. The temperature in 1987 had warmed faster and earlier than the 30-yr average, which probably accounted for the reduced severity. A similar reduction in severity was noted in an experimental plot in another infested field in the same general area (*unpublished data*). The tomatoes were harvested in August and the plant residue was disked into the soil which was listed until February 1988 when beds were shaped and planted with dill. The root debris from the 1987 crop was clearly recognizable in November 1987 and in February 1988 at which time it was partially broken up by cultural practices. The tomato debris was harder to find but still recognizable in March and May 1988. Only small fragments (probably dill residue) were found in October 1988.

The abundance of *P. lycopersici* in material collected from November 1987 to May 1988 was greatest in the cortical tissue of tomato root debris, much lower in the stelar tissue of debris or the rhizosphere soil, and lowest in soil not associated with root debris (Table 1). Soil collected in October 1988 when tomato debris from 1987 was not recognizable gave 4.0 ± 4.0 colonies of *P. lycopersici* per gram of soil. The pathogen was isolated from necrotic lesions on the roots of volunteer tomato seedlings collected in November 1987, March 1988, and May 1988. It was not isolated from the root debris or volunteer seedlings of the dill crop in this field in

Table 1. Number of colonies of *Pyrenochaeta lycopersici* recovered from soil and root debris[a] of a 1987 tomato crop in an infested field near Patterson, CA

Month sampled	Field soil	Rhizosphere soil	Cortical tissue	Stelar tissue
November 1987	2.7 ± 2.3	NT[b]	4,400 ± 2,200[c]	48[d]
February 1988	2.7 ± 4.6	320 ± 330	21,000 ± 9,000	270 ± 230
March 1988	5.3 ± 9.2	64 ± 32	6,300 ± 500	200 ± 200
May 1988	10.7 ± 4.6	117 ± 84	17,500 ± 7,600	2,300 ± 830

[a] Number of colonies per gram of material. Average for three replicates ± standard deviation.
[b] NT = Not tested.
[c] Sample consisted of 4.5 g of tissue.
[d] Results from one replicate; sample consisted of 4.5 g of tissue.

October 1988. A melon crop was grown in an adjacent field in 1987. *P. lycopersici* was not isolated from melon root debris collected in February 1988, but 5.3 colonies per gram of soil were isolated from the soil.

Representative colonies were transferred from CRM and 113 of 128 were confirmed to be *P. lycopersici*. Fourteen colonies did not sporulate but had the colony characteristics of the corky root fungus; one colony sporulated but was not *P. lycopersici*.

Grove and Campbell (9) began a long-term study of the survival of *P. lycopersici* in infected tomato roots buried in fallowed soil. Their earlier data, converted to colonies per root piece, together with those obtained when we recovered sets of roots 20 and 33 mo after burial, are included in Table 2. Many colony-forming units of the pathogen survived for at least 33 mo. Colonies tentatively identified as *P. lycopersici* were transferred from CRM and 40 of 51 were confirmed to be *P. lycopersici*. The remaining 11 colonies had similar colony characteristics.

Table 2. Recovery of *Pyrenochaeta lycopersici* from infected tomato roots buried in soil at two depths[a]

Burial period	15 cm	30 cm
1.5 months[b]	4,190 ± 970	3,170 ± 670
6 months[b]	5,950 ± 1,670	7,900 ± 680
10 months[b]	2,123 ± 2,120	1,070 ± 1,190
20 months	3,200 ± 860	6,100 ± 3,900
33 months	1,030 ± 1,200	870 ± 800

[a] Number of colonies per root sample. Average of four samples ± standard deviation.
[b] Data from Grove and Campbell (9) converted to colonies per root sample.

Saprophytic ability. No colonies of *P. lycopersici* were isolated from healthy tomato roots buried for 1 or 2 mo in infested vermiculite, naturally infested soil, or noninfested control media.

Disease progress in microplots. The influences of environmental conditions and cultivar resistance on infection and disease severity were studied in microplots. Environmental conditions were varied by planting seeds at monthly intervals from February to May 1988. Disease development and isolation of lycopersici from roots of plants in the noninfested soil was rare; data presented here are only from plants grown in naturally infested soil.

The weekly mean air temperatures in 1988 were warmer than the 30-yr average after the first two plantings but colder after the last two plantings (Fig. 1). The accumulation of degree days (10 C threshold) during the first and fourth plantings are presented in Figure 2C; those for the second and third plantings were intermediate and have been omitted. The average number of degree days accumulated per day for plantings one to four was 6.6, 8.5, 10.4, and 12.3, respectively.

The first symptoms of corky root on plants in infested soil were small, dark, oval lesions on the taproots of plants at the two-leaf stage. By the four- to eight-leaf stage, the lesions had become medium-brown, slightly cracked bands encircling some of the roots, and by the flowering stage the bands had the characteristic dark brown, fissured appearance of corky root.

The final disease severity ratings were 3.4, 2.9, 2.4, and 1.5 for plantings one to four, respectively, of the susceptible cultivar and 2.2, 2.0, 1.8, and 0.8, respectively, of the resistant cultivar. Disease severity in infested soil increased linearly throughout the growing season for all planting dates and for both cultivars. When the effect of planting date on disease development was analyzed, the rate of increase in disease severity was significantly greater ($P < 0.001$) for the first planting than for the fourth planting of each cultivar (Fig. 2A, B). The rate of disease increase on the resistant cultivar was significantly less ($P = 0.04$) than on the susceptible cultivar only for the third and fourth plantings but in pair-wise comparisons between resistant and susceptible cultivars, the resistant cultivar had significantly less disease ($P < 0.01$) than the susceptible cultivar at each planting date. The rate of increase in disease severity for the second and third plantings of the susceptible cultivar was intermediate to and not significantly different from either the first or fourth plantings. For the resistant cultivar, however, the disease progress curves for the second and third plantings were similar to those for the first planting and differed significantly only from the fourth planting.

The roots from plants harvested at the flowering stage or later were rated for disease and cut into sections with or without corky root lesions. The cortex was removed from the root pieces in each group, combined, and processed as one or two samples, depending on the amount of tissue available. Many colonies were recovered from corky root lesions, particularly from the three earliest plantings (Table 3). Half of the samples of asymptomatic roots of both cultivars yielded *P. lycopersici*, although in lower numbers than those obtained from lesion tissue. Asymptomatic roots from seven samples of plants grown in noninfested soil did not yield *P. lycopersici*.

Temperature experiments. The effect of temperature on disease development was studied in controlled environment chambers. In one trial, resistant and susceptible tomatoes were grown at 16 C, 21 C, or 27 C for 3 wk in vermiculite infested with isolate 80-55. Disease severity ratings of 0.7 at 27 C were significantly ($P = 0.01$) lower than the ratings of 2.0 at the lower temperatures, but there was no difference in disease severity between the two cultivars.

Two experiments were done to separate the potential effect of temperature on initial infection from that on lesion expansion and symptom development. Seeds of the cultivars Moboglan (resistant) and VF 6203 (susceptible) were germinated and grown for 6 days in sterile vermiculite in 10-cm-diameter pots at 21 C. Six seedlings of each cultivar were transplanted into each of four micropots containing either infested or sterile vermiculite. The pots then were

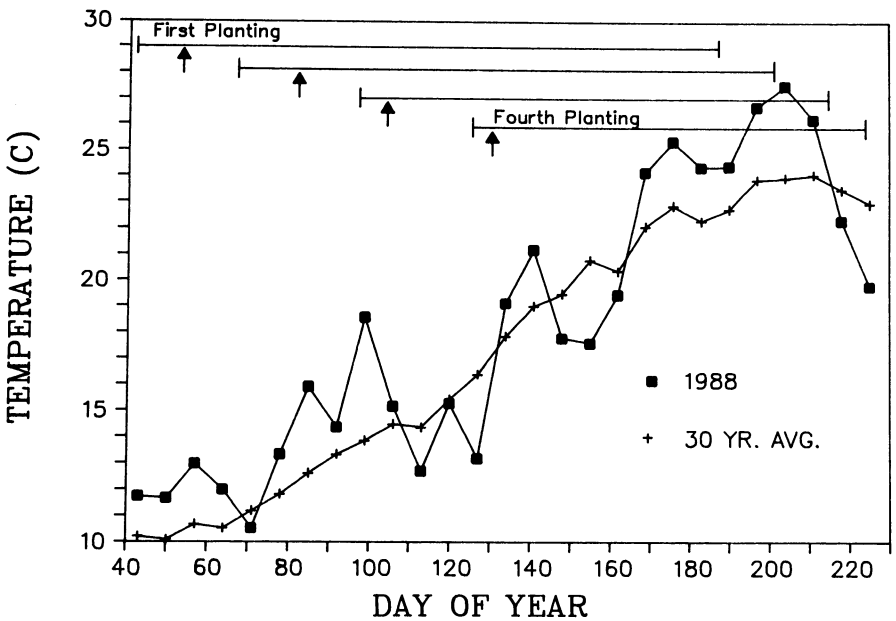

Fig. 1. Mean weekly temperature at Davis, CA, for 1988 and for 30-yr period (1951–1980) with growing periods of each planting in microplots. Vertical arrows indicate date of seedling emergence.

placed in chambers at 16 C or 27 C for an infection period. After the infection period, the vermiculite was washed from the roots, and groups of six seedlings were transplanted into new micropots of sterile vermiculite and returned to chambers for a postinfection period at either 16 C or 27 C. Thus, each replicate pot containing six plants was subjected to one of eight combinations of cultivar, infection, and postinfection treatments in each of the two trials. In the first trial, seedlings from the 21 C germination chamber were transplanted directly into vermiculite infested with isolate 5MC or noninfested vermiculite for a 2-wk infection period, and the postinfection period was 3 wk in duration, whereas in the second trial germinated seedlings were allowed to acclimate to the 16 C or 27 C infection temperatures for 3 days before the infection treatment began, isolate 80-55 of *P. lyopersici* was employed, and the postinfection period was 2.5 wk in duration.

In both trials, plants exposed to infested vermiculite contained small oval lesions on the hypocotyl and taproot at the end of the infection period. No noticeable difference in the size or number of lesions between the infection temperature treatments or between resistant and susceptible plants were observed. No disease was detected on roots of plants grown in the noninfested vermiculite; consequently, data from these controls were not included in the analysis. There were no significant interactions among infection and postinfection temperatures and cultivars. A significant ($P = 0.0001$) main effect of temperature during the postinfection period was detected (Table 4). Plants incubated at 16 C during the postinfection period were more severely diseased than those incubated at 27 C, regardless of the temperature during the infection period. Disease severity was significantly greater on the susceptible than on the resistant cultivar in the first, but not in the second trial, and the main effect of cultivar was not statistically significant.

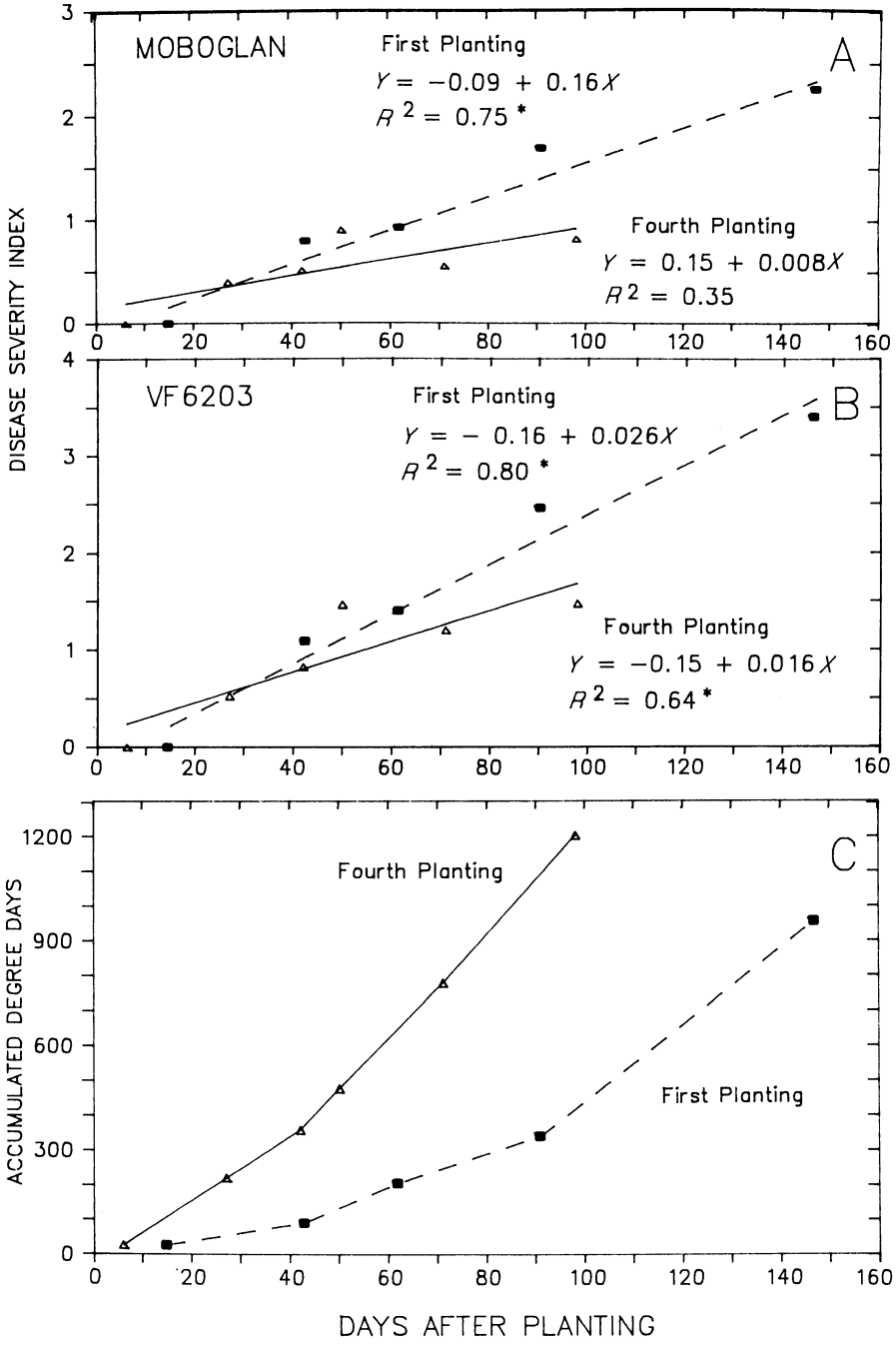

Fig. 2. Disease progress curves and accumulated degree days (10 C threshold) for two plantings of the tomato cultivars Moboglan (resistant) and VF 6203 (susceptible) in microplots with soil naturally infested by *Pyrenochaeta lycopersici*, Davis, CA, 1988. * = R^2 value significant at $P < 0.001$.

Susceptibility of *Solanum* spp. Three *Solanum* spp. were compared to tomato for their susceptibility to *P. lycopersici* (Table 5). Tomato was the most susceptible host with coalescing lesions on the taproot; *S. sarachoides* was intermediate with discrete lesions ≥1 mm in length; and *S. nigrum* and *S. nodiflorum* were resistant with lesions <1 mm long (Table 5). Colonies of *P. lycopersici* were isolated from triturated segments of roots with lesions of all species, as well as from symptomless segments of two *Solanum* spp., but not from controls.

Additional observations on the susceptibility of *Solanum* spp. growing in field conditions were made in conjunction with the experiments described above. Volunteer seedlings of *S. nodiflorum* that occurred in microplots with infested soil were collected when the tomatoes were sampled. The *S. nodiflorum* plants had no symptoms, but three of seven samples yielded *P. lycopersici* (Table 3). In October 1988, roots of *S. nodiflorum* and *S. sarachoides* plants growing in the naturally infested field near Patterson were collected. They had little or no discoloration but yielded *P. lycopersici* (134 colonies per gram of wet weight for *S. nodiflorum*, 551 colonies per gram of wet weight for *S. sarachoides*).

DISCUSSION

Our results strongly confirm and extend the conclusion that *P. lycopersici* is an ecologically obligate parasite with no competitive saprophytic ability (5,6). The fungus did not saprophytically colonize tomato roots buried for 1 to 2 mo in naturally infested soil or in pasteurized vermiculite with microsclerotia from pure culture. We recovered 1,000–53,000 microsclerotia per gram of cortex from corky lesions on roots of growing plants and similar numbers from random samples of roots collected 2–8 mo after harvest of a tomato crop in an infested field and from samples of roots with lesions buried in nylon bags for up to 33 mo. Although there was variation in numbers of microsclerotia recovered in various

trials, the numbers were consistent enough to argue that the fungus does not grow actively and increase the number of propagules in infected root debris for very long after the death of the host. The fungus is, therefore, in the category of those with dormant survival by resting propagules (8).

Symptomless infections have been described on some hosts (9,18), but they have not been evaluated for their impact on inoculum increase. We found propagules of the fungus in symptomless sections of roots of tomato or *Solanum* spp. growing in naturally infested soil in numbers exceeding those in random soil samples and in even greater abundance in the root debris samples from a crop that had a low disease severity at mid-season. We hypothesize that symptomless infection of host roots followed by extensive colonization and increase of microsclerotia near harvest time may play an important role in inoculum increase even in years when tomatoes are not grown or are not severely affected.

The technique of blending and sieving of roots or soil followed by plating onto a semiselective medium proved useful for quantitative studies of *P. lycopersici*. We have verified that 85% of 179 colonies putatively identified as *P. lycopersici* on the basis of colony characteristics were correctly identified. An additional 14% of these probably were *P. lycopersici* but failed to sporulate. Thus, we conclude that colonies of *P. lycopersici* can be reliably identified on CRM. It is reasonable to assume that the propagules being enumerated in these studies are microsclerotia. The fungus produces microsclerotia in roots and in culture (2,14,20), and they were the propagules recovered from roots buried in soil (9). The propagules extracted from living infected roots probably were microsclerotia because the brief NaOCl treatment in the protocol should have killed most vegetative mycelium and thin-walled spores.

The greatest concentration of microsclerotia was found in the cortical tissue of root debris. The low number of propagules recovered from the stelar tissue could have resulted from contamination of the samples by cortical tissue that was not removed completely or from limited colonization of the stele after harvest. The fungus is restricted to the cortex in lesions on large, living roots but can invade and kill entire small rootlets in the field or greenhouse (7,18). In our histological observations of early stages of infection of young seedling roots, the fungus was found only in the cortex of the taproot unless a lateral root had been killed (17). In this case, the vascular tissue of the lateral root was brown and the necrosis extended into the stele of the taproot. The number of microsclerotia in the rhizosphere soil was somewhat greater than in a general soil sample. Nevertheless, these microsclerotia probably originated from decay and sloughing of cortical tissue, not from saprophytic colonization or development of the fungus in soil. The number of colonies recovered from soil samples was comparable to the numbers from soil in other infested fields (*unpublished data*). Further study of the seasonal fluctuation of propagules in soil is warranted.

The sequential development of symptoms caused by *P. lycopersici* in field conditions was followed on plants grown in microplots. The pathogen infected young seedlings and caused small, necrotic lesions on the taproot. The lesions continued to enlarge as the host grew and developed into typical corky lesions. These observations apparently contradict those of Last et al (11), who considered corky lesions as a tolerant reaction that occurred when larger roots were invaded or the inoculum was less concentrated. For them, the brown root rot phase of the disease in which fine roots were killed represented the more severe symptom pattern. The brown root rot phase occurs on our field grown tomatoes, but it is usually observed as a lack of fine rootlets on the parts of the taproot or major lateral roots with corky lesions. The fine rootlets on field-grown, direct-seeded tomatoes are not as abundant as on transplanted tomatoes grown in glasshouses or in the field. Therefore, we put little emphasis on rating brown root rot as a separate phase of the disease. Until inoculum density and disease severity relationships in both systems are better understood and additional sequential sampling experiments are done, the interpretation of symptom expression is open.

The results of our experiments

Table 3. Number of colonies of *Pyrenochaeta lycopersici* recovered per gram of cortex of root pieces with or without corky root lesions

		Planting number[a]					
		1	2	3	4	4	4
		Days after planting[b]					
Host[c]	Tissue[d]	147	133	112	50	70	95
VF 6203	Diseased	47[e]	53	14 ± 14	2 ± 0	0.5	3 ± 3
VF 6203	Symptomless	···	···	0 ± 0	0.5	···	0.3 ± 0.1
Moboglan	Diseased	5	22	10 ± 11	0	4	0.2 ± 0.3
Moboglan	Symptomless	1	1	0	0	1	0 ± 0
Solanum nodiflorum	Symptomless	0	10 ± 6	0	···	1	0 ± 0

[a] Plantings one through four were planted at 4-wk intervals from 9 February to 4 May 1988.
[b] Root tissue was assayed at indicated days after planting.
[c] Tomato cultivar VF 6203 susceptible to corky root; Moboglan resistant.
[d] Cortex from root segments with symptoms of corky root or from symptomless segments.
[e] Results given as number of colonies per gram dry weight of cortex × 10^3. If enough tissue was available, two samples were assayed in which case the results are given as mean ± standard deviation; ··· = no samples assayed.

Table 4. Effects of temperature during infection and postinfection periods and of cultivar resistance on the mean severity of corky root lesions

		Cultivars[a]		Temperature
Period	Temperature (C)	Moboglan	VF 6203	mean
Infection	16	2.1 ± 0.3	2.4 ± 0.3	2.24
	27	1.7 ± 0.2	2.0 ± 0.3	1.87
Postinfection	16	2.5 ± 0.2	2.9 ± 0.2	2.69*[b]
	27	1.3 ± 0.2	1.6 ± 0.3	1.42*
Cultivar mean		1.91	2.21	

[a] Disease severity rated from 0 = no symptoms to 4 = >75% of tap root with lesion and given as mean ± standard error. Cultivar Moboglan resistant; VF 6203 susceptible.
[b] * = Means differed significantly from each other ($P = 0.0001$).

Table 5. Corky root disease severity on various hosts and recovery of *Pyrenochaeta lycopersici* from root cortex of inoculated plants

Host	Disease severity[z]	Symptoms on root pieces	Colonies/g dry wt. (no.)
Tomato	3.3 ± 0.0 a	+	5,500
Solanum sarachoides	2.3 ± 0.3 ab	+	3,100
S. nigrum	1.3 ± 0.3 b	+	5,800
		−	2,900
S. nodiflorum	1.3 ± 0.2 b	+	4,400
		−	800

[z] Average disease severity per pot and standard deviation for three replicates. Treatments followed by the same letter do not differ significantly at $P < 0.0005$.

examining the relationship of temperature to disease are consistent with the general observation that corky root is more severe in cool than in warm soil (4,18). In controlled environments with young seedlings, the prevalence of initial infections was the same at 16 or 27 C but the disease became more severe on plants placed at 16 C for the postinfection period than on those at 27 C. In microplots, the slope of the linear disease severity curves was greater for the first planting date with cooler soil temperatures than for the fourth planting date. This pattern occurred with both a resistant and a susceptible cultivar. The present linear disease progress curves contrast with the curvilinear disease progress curves for glasshouse tomatoes (10). The relationship between disease severity and temperature that fluctuates daily and changes seasonably is complex and needs to be resolved through further field experimentation to understand the epidemiology of corky root disease on field-grown tomatoes. Field trials may be necessary to screen cultivars or breeding lines for resistance because the difference between cvs. Moboglan and VF 6203 was more consistently detected in microplots than in short-term experiments in controlled conditions.

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