Light Brown Discoloration of Tomato Roots Caused by *Fusarium oxysporum*

NINA SHISHKOFF, Postgraduate Research Plant Pathologist, and R. N. CAMPBELL, Professor, Department of Plant Pathology, University of California, Davis 95616

ABSTRACT

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Light brown discoloration (LBD) of tomato is a distinct, minor disease caused by isolates of *Fusarium oxysporum* that are of the saprophytic type and do not cause wilt symptoms in tomato or other vegetable crops. No significant interactions with *Pyrenochaeta lycopersici*, causal agent of corky root, were observed in greenhouse trials.

A symptom that we propose to designate as light brown discoloration (LBD) has been observed frequently on roots of field-grown tomatoes (*Lycopersicon esculentum* Mill.) in California for several years. When LBD has been observed on young tomato plants, it has sometimes been diagnosed as the early stages in development of lesions of corky root, caused by *Pyrenochaeta lycopersici* R. Schneider & Gerlach. Isolations from LBD lesions, however, have yielded *Fusarium* spp. but not *P. lycopersici*. Other investigators have reported similar symptoms on tomato roots in the absence of *P. lycopersici* (16,20). The LBD symptoms are distinct from the chocolate brown lesions and vascular discoloration of crown and root rot of greenhouse tomatoes caused by *Fusarium oxysporum* Schlechtend.:Fr. f. sp. *radicis-lycopersici* W. R. Jarvis & Shoemaker (17). *Fusarium* spp. are common soil inhabitants associated with roots of plants and have been isolated by many workers attempting to isolate *P. lycopersici* (2,4,11,12,16,19,20). Interactions between *P. lycopersici*, *Fusarium solani* (Mart.) Sacc., and *F. oxysporum* Schlechtend.:Fr. were studied in tests, most of which used young seedlings and short (up to 1 wk) in vitro exposure to the fungi singly or in combination (4,5). Prior inoculations or coinoculations with *Fusarium* spp. reduced the severity of corky root.

The objectives of the present study were to identify the fungus that causes LBD, to compare early symptoms of LBD and corky root, and to test for possible interactions between the LBD fungus and the corky root fungus.

Present address of first author: U. S. Department of Agriculture, Agricultural Research Service, Ft. Detrick, Frederick, MD 21701.

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MATERIALS AND METHODS

Isolations and inoculum increase. Tissue with LBD symptoms was surface-disinfested for 30 sec in 0.525% NaOCl and rinsed in sterile, distilled water. Small chips of tissue from the edges of LBD lesions were aseptically transferred to water agar (WA), and the fungi that

grew out of the tissue were transferred to potato-dextrose agar (PDA). Three isolates of *Fusarium* sp. (LBD-A1, LBD-C1, LBD-E1) used in this study were obtained from LBD lesions on roots of tomatoes grown in a field in Yolo County, California, in 1987. Two isolates of *F. oxysporum* (FT 8803 and FT 8822) with colony morphology characteristic of nonpathogenic isolates (9) were obtained from T. Gordon. They had been isolated in 1988 from the surface of symptomless tomato roots (washed but not surface-disinfested) from Fresno County, California. An isolate of *F. oxysporum* f. sp. *lycopersici* W. C. Snyder & H. N. Hans. (race 2 [Fol 139]) isolated in 1987 from wilted tomatoes in Sutter County, California, was provided by R. M. Davis. All *Fusarium* isolates were grown on PDA in petri dishes. Conidia were harvested by adding sterile, distilled water and rubbing the colonies with a glass rod. The spore suspension was filtered through two layers of cheesecloth and the spores were counted in a hemacytometer.

Isolates of *P. lycopersici* (BL-1 and 80-55 isolated from corky root lesions on tomatoes in Fresno County and Sacramento County, respectively, or D-1 isolated from buried tomato root debris) were used. Conidia of *P. lycopersici* were produced as described (13). Microsclerotia of the corky root fungus were produced by culturing the fungus on a Difco Czapek's agar:quartz sand medium developed in this laboratory by D. M. McGrath (*personal communication*). Flint-shot sand (Ottawa Industrial Sand Co., Ottawa, IL) was placed in petri plates and sterilized on two consecutive days. Approximately 20 ml of molten sterile Czapek's agar was poured into each plate. A suspension of conidia of *P. lycopersici* was transferred to the medium and the plates were incubated at 25 C for 3 wk. Alternatively, conidia could be added to cooled medium just before being poured into the petri dishes. The microsclerotia were harvested by adding the contents of a petri dish to 200 ml of distilled water in a blender and mixing at high speed. After the sand settled, the supernatant was poured onto a 38-μm sieve and washed with tap water. The microsclerotia on the sieve were backwashed into sterile, distilled water and counted in a hemacytometer.

Identification of *Fusarium* isolates. *Fusarium* isolates were transferred to carnation leaf agar (8), illuminated with three or four 40-watt Sylvania cool-white fluorescent bulbs and one near-UV (black light) bulb suspended 40–50 cm above the plates, and grown at room temperature for 5 days. Thirty to 38 spores were measured from each isolate. Keys by Liddell and Davis (*personal communication*) and Booth (1) were used to identify the species.

Pathogenicity tests. Tests were done by growing plants from seeds sown in infested vermiculite or soil unless otherwise stated. The vermiculite was infested with suspensions of conidia to give a final concentration of 1×10^5 or 1×10^6 conidia per millileter of vermiculite and mixed in a plastic bag for 3–5 min. The same procedure was used for microsclerotia of *P. lycopersici*, but the final concentration of microsclerotia was 1×10^4 or 1×10^5 per millileter of vermiculite. The control treatment was vermiculite mixed with an equivalent volume of sterile, distilled water. Soil was infested in some experiments in the same manner except it was mixed for 5–10 min after the inoculum was added. The infested medium was dispensed into 10- or 15-cm-diameter plastic pots for most experiments but sometimes into 270-ml micropots made from plastic cups with holes punched in the bottom. There were three to five replicate pots per treatment. Seeds were sown in the pots and the seedlings were thinned to the desired density (four to six per pot, occasionally nine per pot) and grown for 4–6 wk. Plants were grown in a greenhouse at minimum night temperatures of 18–21 C. The maximum daytime temperatures were kept at <27 C during the winter (October–May) and <33 C during the summer by evaporative cooling. Unless otherwise stated, pathogenicity tests were done in the winter. Occasional trials were done in a growth chamber at 21 C. At the end of the trials, the plants were weighed and the extent of lesions on the upper 3 cm of taproot of each plant was graded using a 0–4 disease severity scale for corky root (2) and averaged for all of the seedlings in a replicate pot. The taproots were rated as follows: 0 = no symptoms, 1 = 1 to a few small lesions on <10% of the root, 2 = small lesions that did not coalesce on 10–25% of the root, 3 = numerous lesions on 25–75% of the root, and 4 = >75% of taproot with lesions.

For the root dip test, seedlings grown for 1–3 wk in sterile river sand were uprooted and their taproots were trimmed with scissors. The seedlings were placed for 15 min in beakers of spore suspensions adjusted to 1×10^6 to 1×10^7 conidia/ml or in beakers of sterile, distilled water. The seedlings were transplanted into 40 × 56 cm trays containing pasteurized potting soil and kept in a greenhouse during the summer. Plants were scored for wilt symptoms 2 wk later.

Host plants. VF 6203 or VF 145-B7879 (hereafter designated VF 145) were used interchangeably as corky root susceptible cultivars and Moboglan was used as a resistant cultivar (3) in the pathogenicity tests for tomato. Fusarium wilt differentials Earlypak, Pakmore, and Walter (7) were included in root dip tests.

Histology of infection. Infection and early stages of lesion formation were studied by destructive sampling every 2–3 days of one pot of seedlings grown in infested vermiculite for 16–25 days after sowing. Tomato roots were cleared in 10% KOH and stained with trypan blue using the procedure of Phillips and Hayman (15) and examined microscopically. Roots were sectioned transversely by hand and stained with phloroglucinol/HCl to detect lignin and with Sudan Red IV or Sudan Black B to detect suberin (14).

Statistical analysis. Duncan's multivariate analysis using the Statistical Analysis System (SAS) computer program (18) was used to analyze results for analysis of variance. Because disease severity was rated for each plant and averaged for each pot, categorical data approximated random normal data making analysis of variance applicable.

RESULTS

Description of LBD. On young plants in the field, LBD was characterized by light brown, slightly cracked bands <1 cm to 3 cm wide and encircling the taproot or lateral roots but only affecting a few outer layers of cortical tissue. These early lesions previously have been misdiagnosed as corky root. If these plants were examined later, however, the LBD lesions remained as superficial, light-colored lesions distinct from the advanced lesions of corky root that became darker, more fissured, and extended deeper into the cortex (Fig. 1). Furthermore, *P. lycopersici* was not recovered from young or advanced LBD lesions if they were plated onto a semiselective medium (10). Isolations on WA and PDA consistently yielded *Fusarium* spp.

Identification of *Fusarium* isolates. Isolates LBD-A1, LBD-C1, and LBD-E1 were similar except for the color on the underside of the colony. LBD-A1 was white to buff, LBD-C1 was pale orange, and LBD-E1 was purple. The average macroconidial dimensions were: 38 (32–44) × 3.3 (2.8–3.8) μm for LBD-A1, 37 (34–40) × 3.9 (3.5–4.3) μm for LBD-C1, and 34 (24–44) × 2.9 (2.5–3.3) μm for LBD-E1. Macroconidia of isolate FT 8803 were 32 (27–38) × 4.1 (3.7–4.5) μm, and those of isolate FT 8822 were 34 (26–43) × 4.1 (3.9–4.3) μm. All of these isolates were identified as *F. oxysporum* on the basis of the size of macroconidia and phialides, the presence of microconidia, macroconidia, and chlamydospores, and the colony characteristics.

Pathogenicity tests. Twelve, 11, and five of the 15 plants inoculated with isolates LBD-A1, LBD-C1, and LBD-E1, respectively, had one or two distinct bands of light brown discoloration on their taproots (Fig. 2). These bands of slightly cracked tissue were only three to eight cell layers deep. There was no vascular discoloration and no difference in fresh weight between inoculated and control plants. A *Fusarium* sp. with

conidial characteristics similar to the inoculated isolate was recovered from symptomatic tissue. Two of 45 control plants had lesions, but attempts to isolate *Fusarium* onto WA from them or from symptomless controls were not successful.

In another trial, dark brown lesions typical of corky root developed on the plants inoculated with *P. lycopersici* isolate 80-55 (Table 1). Some, but not all, of the plants inoculated with LBD-E1 developed the characteristic light brown banding of LBD on the taproot. The analysis of variance was done with log-transformed data omitting noninoculated controls that were symptomless. The severity of LBD was significantly less than that of corky root. Moboglan was slightly less diseased than VF 145. Disease severity did not change between 4 and 6 wk. Plants sampled at 4 wk were weighed but there was no significant difference in fresh weight among the treatments.

None of the LBD isolates caused wilt symptoms on the differential cultivars in a root dip test, whereas Fol 139 caused wilt of the cultivars susceptible to race 2. *F. oxysporum* was recovered from plants inoculated with each of the isolates. The controls were symptomless and did not yield *Fusarium* sp. colonies.

Isolates LBD-E1 and LBD-A1 were inoculated to 15 vegetables in a root dip test but did not cause wilt of any of the species tested. The vegetables tested included: *Allium cepa* L. 'Sweet Spanish'; *Apium graveolens* L. 'Tall Utah Improved'; *Brassica campestris* L. var. *chinensis* L. 'Wong Bok'; *B. oleracea* L. var. *botrytis* L. 'Snowball'; *B. oleracea* L. var. *capitata* L. 'Golden Acre'; *Capsicum annuum* L. VR-2564-5001; *Carthamus tinctorius* L. N-10; *Cucumis melo* L. PMR45; *Gossypium hirsutum* L. 'Acala'; *Phaseolus vulgaris* L. 'Tenderpod'; *Pisum sativum* L. 'Early Frosty'; *Raphanus sativus* L. 'Champion' and 'Icicle'; *Solanum melongena* L. Petoseed PSX6674; and *Spinacia oleracea* L. 'Improved Thick Leaf'. Plants were not rated for LBD because LBD symptoms were not expected in a 2-wk incubation period.

Histology of early stages of disease. The development of LBD by isolates LBD-A1 and LBD-E1 on VF 145 was observed in two experiments. Light brown lesions developed on the hypocotyls of plants more than on the taproots. Hyphae, sometimes producing macroconidia, were seen on roots at 7 days, and they had penetrated epidermal cells or stomates. The infected epidermal cells or guard cells had brown, granular contents and the adjacent cells were enlarged. By 9–14 days, the lesions usually consisted of three to 20 brown cells and had an irregular appearance because of sloughing of dead cells and hypertrophy of neighboring cells. Walls of the dead cells and of adjacent living cells stained positively for lignin and suberin. After 15 or 16 days, the lesions extended around the circumference of the root and formed bands that penetrated only a few cell layers into the cortex.

The development of corky root lesions by *P. lycopersici* (isolate BL-1) was studied in two experiments, one with VF 145 and inoculum of conidia and the other with VF 145, Moboglan, and inoculum of microsclerotia. Regardless of the type of inoculum, the fungal hyphae were first seen on the outside of the roots 5–6 days after planting. After 6 days, the fungus began to penetrate the epidermal cells of the taproot, but usually not of the hypocotyl, and prominent papillae formed at the penetration sites. Infected cells had brown, granular contents. Lesions grew from small groups of infected cells at 8–10 days to larger, deeper lesions with a rough, cracked appearance by 20–25 days. The walls of dead cells and of cells directly adjacent to brown cells stained positively for lignin and suberin. Lesions did not extend inward past the endodermis except at sites of lateral root development. Lateral roots were often killed, and limited vascular discoloration was observed. There was no visible difference in disease severity between resistant and susceptible cultivars in the trial with microsclerotia.

Early stages of infection by microsclerotia of *P. lycopersici* isolate 80-55 and by LBD-A1 on VF 6203 were compared in an experiment at 20 C in a growth chamber. *P. lycopersici* caused lesions that formed primarily on the taproot and invaded inward to the endodermis. The lesions caused by LBD-A1 formed mainly on the hypocotyl and remained superficial. Individual cells in either type of lesion had dark, granular contents, but the corky root lesions were darker brown than the LBD lesions because the corky root lesions were deeper.

LBD-A1 and saprophytic isolates FT 8822 and FT 8803 caused similar LBD lesions in a separate experiment at 20 C.

Interaction between *F. oxysporum* and *P. lycopersici*. Isolates LBD-A1 and *P. lycopersici* were inoculated to tomatoes singly or in combination and evaluated 12 wk later in four trials. Although severity of infection was greater in vermiculite than in soil, vermiculite was not satisfactory for plant growth for 12 wk. Different media or placements of infested medium were made to assure infection by both fungi and good growth of the host. Micro-

Fig. 1. Light brown discoloration **(left)** and corky root **(right)** symptoms on roots of tomatoes collected from different fields in the San Joaquin valley of California in July 1985.

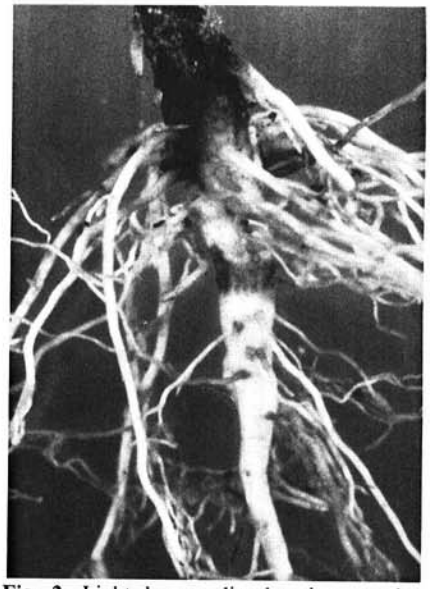

Fig. 2. Light brown discoloration on the hypocotyl of a tomato inoculated with *F. oxysporum* isolate LBD-C1 2 mo previously.

sclerotia of *P. lycopersici* isolate 80-55 were used in all trials, except microsclerotia of isolate D-1 were used in trial number 2. Trials 1, 2, and 4 were conducted during the winter and trial 3 was done in the summer. In trial 1, the medium was a 50:50 mixture of Yolo clay loam:sand and LBD-A1 and *P. lycopersici* were added individually at a concentration of 1.4×10^5 propagules per millileter or added together each at 0.7×10^5 propagules per millileter. In trial 2, pasteurized Yolo clay loam soil was placed in pots to a depth of 9 cm. Seeds were sown and covered with 3 cm of infested or control vermiculite. In trial 3, plants were germinated at 21 C in a growth chamber in infested or noninfested vermiculite in micropots. After a 12-day infection period, seedlings were transplanted into pots filled to a depth of 7 cm with pasteurized soil and topped with 4 cm of new vermiculite. The pots were placed in the greenhouse. In trial 4, the pots were filled to a depth of 7 cm with a 3:1 mixture of river sand:Yolo clay loam. Seeds were sown and topped with infested or control vermiculite.

Corky root was severe on plants inoculated with *P. lycopersici* alone, and LBD was less severe on plants inoculated with LBD-A1 (*F. oxysporum*) alone (Table 2). The severity of corky root on plants inoculated with both fungi was reduced significantly only in trial 4. Controls were symptomless except for one plant that developed LBD in trial 3. No attempt was made to isolate the causal agent from the lesion. There was no significant difference in fresh weight of plants in different treatments.

DISCUSSION

We have reproduced symptoms similar to LBD with *F. oxysporum* isolates from LBD lesions on tomato roots and have recovered the inoculated fungi, thus completing Koch's postulates. We propose that LBD should be recognized as a distinct disease of tomato. The importance of the disease, however, seems to be minor because it did not cause obvious growth reduction of plants in pathogenicity tests and significant yield reductions have not been associated with it in the field. The disease severity scale used in the pathogenicity trials was based on the area of the lesions on the host and therefore overestimated the severity of LBD lesions that are more superficial than corky root lesions. The chief reason to distinguish LBD in the field is to avoid confusing it with corky root, which can cause significant yield loss (2,12,16). A similar brown discoloration probably has been noted by others. Browning of the roots in the absence of *P. lycopersici* was attributed to low oxygen content in saturated soils or to salinity (20). Root lesions from which *Fusarium* and other fungi, but not *P. lycopersici*, were isolated were reported by Polley (16). Davet (6) noted some cortical lesions that were not described further when a saprophytic type of *F. oxysporum* was inoculated to cv. Marmande which was susceptible to Fusarium wilt but not when it was inoculated to two cultivars resistant to Fusarium wilt. In our experiments, we worked only with resistant cultivars and noted LBD on them. Thus, it is not certain that our LBD is the same as the lesions noted by Davet.

The lesions caused by *F. oxysporum* and *P. lycopersici* also differed in trials in the greenhouse or in growth chambers. The lesions of *P. lycopersici* penetrated more deeply into the cortex, were darker in color, and involved more of the surface of the root or hypocotyl than those of *F. oxysporum*. In these trials, *F. oxysporum* often invaded the cortex of the hypocotyl, the boundary of which with the taproot is difficult to observe. The predominance of LBD on the lower hypocotyl region of plants in greenhouse trials, in contrast to its prevalence on roots of plants in the field, is attributed to the differences in the environment.

We do not propose a subspecific name for the causal fungus of LBD. Our isolates did not cause vascular wilt and the symptoms of LBD are distinct from those of crown and root rot, caused by *F. oxysporum* f. sp. *radicis-lycopersici*. We believe that LBD can be caused by a number of the so-called saprophytic *F. oxysporum* that inhabit soil and colonize debris. This conclusion is supported by the inoculations done with saprophytic isolates of *F. oxysporum* provided by T. Gordon.

We tested the possibility that coinoculation of *F. oxysporum* with *P. lycopersici* might reduce the severity of corky root, but there was only a slight effect

Table 1. Pathogenicity of *Fusarium oxysporum* isolate LBD-E1 and *Pyrenochaeta lycopersici* isolate 80-55 to two cultivars of tomato

	Tomato cultivar				
	Moboglan		VF 145		
	Incubation period				
Inoculum[x]	4 wk	6 wk	4 wk	6 wk	Inoculum mean[y]
P. lycopersici	2.0 ± 0.7[z]	2.3 ± 0.05	2.6 ± 0.6	2.4 ± 0.8	1.19 a
F. oxysporum	0.2 ± 0.2	0.1 ± 0.2	0.5 ± 0.3	1.2 ± 1.2	0.32 b
Cultivar mean[y]	0.62 a		0.88 b		

Source of variation	df	Sums of squares	*F*	Probability > *F*
Treatments	7	6.91	17.43	0.001
Time	1	0.04	0.72	0.40
Cultivar	1	0.53	9.39	0.005
Inoculum	1	6.00	105.85	0.0001
Interactions	4	0.34	1.50	0.24
Error	24	1.36		
Total	31	8.27		

[x] Noninoculated controls were symptomless but have been omitted from the ANOVA test and therefore are not shown in this table.

[y] Means (transformed) for cultivar or inoculum followed by the same letter do not differ significantly ($P = 0.05$) by Duncan's multiple range test.

[z] Disease severity scale based on lesion extent on tap root or hypocotyl: 0 = no lesions; 1 = < 10% of tissue with lesions; 2 = 10–25% of tissue with lesions; 3 = 25–75% of tissue with lesions, 4 = >75% of tissue with lesions. The disease severity ratings are averages and standard deviations from four to five plants in four replicate pots.

Table 2. Interactions between *Pyrenochaeta lycopersici* and *Fusarium oxysporum* on tomato cultivar VF 6203

	Disease severity rating[y]			
Inoculum	Trial 1	Trial 2	Trial 3	Trial 4
P. lycopersici	2.1 ± 0.1 a	2.1 ± 0.2 a	2.5 ± 0.3 a	2.5 ± 0.2 a
P. lycopersici + *F. oxysporum*	1.8 ± 0.1 ab	2.0 ± 0.3 a	2.4 ± 0.1 a	1.9 ± 0.4 b
F. oxysporum	1.6 ± 0.4 b	1.1 ± 0.7 b	0.5 ± 0.4 b	0.4 ± 0.3 c
Control	0 c	0 c	0.4 ± 0.1 c[z]	0 d

[y] Disease severity scale based on lesion extent on tap root or hypocotyl: 0 = no lesions; 1 = <10% of tissue with lesions; 2 = 10–25% of tissue with lesions; 3 = 25–75% of tissue with lesions; and 4 = >75% of tissue with lesions. The disease severity ratings are averages from four to nine plants in four or five replicate pots followed by the standard deviation. Number of plants in each replicate pot/number of replicates pots were: trial 1 = five plants/four pots, trial 2 = nine plants/four pots, trial 3 = four plants/five pots, trial 4 = six plants/five pots. Treatments followed by the same letter in each column do not differ significantly ($P < 0.001$) by Duncan's multiple range test.

[z] One control plant with light brown discoloration.

in one of four trials. Davet observed a marked reduction in corky root severity in two similar trials with a 6-wk incubation period (6). The differences between our results may be attributable to the physical conditions of soil or environment or to biological differences between the fungi involved. The remainder of the tests that Davet used to study the interactions between *F. oxysporum* and *P. lycopersici* were done in vitro with agar block inoculations and 1-wk incubation periods (5,6).

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