# End Rot, Surface Rot, and Stem Lesions Caused on Sweet Potato by Fusarium solani

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#### ABSTRACT

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Fusarium solani isolates from sweet potato caused a surface rot on roots, a previously undescribed decay that progressed from the ends of the roots, and stem lesions on sprouts and vine cuttings. Isolates from Louisiana sweet potatoes with any one of the three different symptoms, produced all three types of symptoms after appropriate inoculations. Louisiana F. solani isolates from sweet potato differed from North Carolina isolates in colony

morphology and in an apparent inability to penetrate the vascular ring of infected roots. Wounding is a prerequisite for infection of sweet potato by *F. solani*, and deep wounds may allow penetration of the vascular ring by Louisiana isolates. Jasper was the only commercial cultivar of six tested which was susceptible to end rot.

Additional key words: Ipomoea batatas.

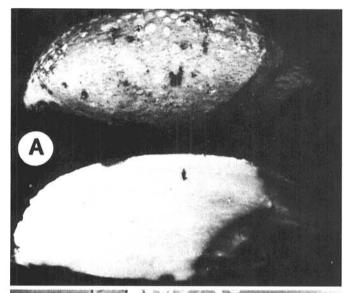
In January 1978, stored sweet potatoes (*Ipomoea batatas* L. 'Jasper') were observed with a dry rot progressing from either or both ends of the root. Margins of the lesions were dark brown to black and the lesion was tan to dark brown in the flesh while the periderm initially was smooth and dark brown. As the decay

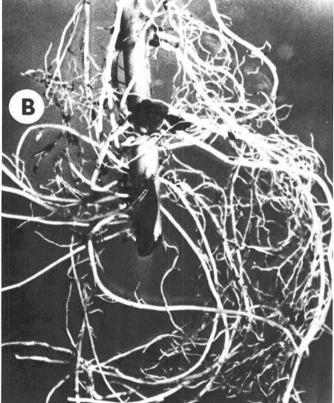
progressed, the lesion shrank and shrivelled and small cavities containing white mycelium developed within the lesion. When affected roots were bedded in the greenhouse, small black lesions developed on the sprouts below the soil line. Cultural morphologies of *Fusarium* sp. isolates from both end rot and from sprout lesions were similar to that of an isolate previously collected from surface rot lesions on tuberous roots.

Harter et al (3) consistently isolated Fusarium oxysporum from sweet potatoes with end rot, but they were unable to reproduce symptoms of the disease with this organism. F. oxysporum has

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caused surface rot of sweet potatoes in storage (2,5). McClure described stem lesions caused by an organism he called Fusarium solani f. sp. batatas (6). His isolates were obtained from roots with end rot, but he reported no attempt to reproduce end rot. F. solani also recently was reported to cause a storage rot of sweet potatoes which differs from surface rot (7). Other Fusarium and Gibberella spp. were reported on sweet potatoes stored at low temperatures (3).

This study was conducted to determine the causes of the end rot, stem lesions, and surface rot of sweet potatoes in Louisiana and to compare these with those of other Fusarium diseases of sweet potato. A preliminary account was published previously (1).

## MATERIALS AND METHODS

Isolations were made by surface sterilizing (0.525% sodium hypochlorite for 10 min) pieces of tissue from the margins of lesions and plating them on potato dextrose agar (PDA). After 5 to 10 days of incubation at room temperature, mycelial transfers were made from the margins of developing colonies to fresh plates of PDA. Single conidium transfers of each isolate were made before it was used for further study. Isolates 77-29 and 78-7 were obtained from surface rot lesions on tuberous roots of sweet potato cultivars Centennial and Jasper, respectively. Isolates 78-30 and 78-32 were obtained from tuberous roots of cultivar Jasper with end rot and isolate 78-36 was obtained from a stem lesion on a sprout produced from the same sweet potatoes bedded in the greenhouse. Isolate 78-47 was obtained from a tuberous root of sweet potato cultivar Porto Rico 198 with a deep rot that superficially resembled surface rot. The above sweet potato roots came from several locations in

TABLE 1. Effects of proximal and distal end inoculations of wounded tuberous roots of sweet potatoes of cultivar Jasper with different isolates of Fusarium solani from sweet potato

Isolate		Experiment II b,c		
	Experiment I-L Proximal	Distal	Lesion length (mm)	Lesion weight (g)
Control	0	0	0	0
NC-M-10	***	***	34	30.6
NC-N-I	***	***	20	7.7
77-29			25	18.0
78-7	9	10	•••	•••
78-25	***	***	21	10.2
78-30	2	3		
78-32	15	15	9	3.4
78-36	10	4	15	7.5
78-47			23	9.8

alnoisions were made at each end of the root, the roots were dipped in propagule suspensions of each isolate ( $1 \times 10^4$  propagules per milliliter) and incubated in moist chambers at room temperature for 43 days.

<sup>b</sup>Freshly cut proximal ends were pressed on the surface of actively sporulating cultures on PDA and incubated in moist chambers at room temperature for 24 days.

Differences among lesion lengths or weights for four replicate sweet potatoes were not significant at P = 0.05 for either inoculation method by Duncan's multiple range test.

Fig. 1. A, A tuberous root of sweet potato cultivar Jasper inoculated with Fusarium solani showing symptoms of Fusarium end rot (upper half, external view; lower half, internal view of the same root). B, Terminal vine cuttings of sweet potato of the cultivar Jasper 4 wk after inoculation with Fusarium solani. Note necrosis at the basal cut and the leaf scar callus. C, An inoculated tuberous root of sweet potato cultivar Centennial showing surface rot symptoms produced by Louisiana isolates of F. solani (left and center lesions) and the penetrating rot produced by North Carolina isolates (right lesion). (Upper half, external view; lower half, internal view of the same root).

Louisiana. Isolates NC-M-10 and NC-N-1 were provided by J. W. Moyer (Department of Plant Pathology, North Carolina State University, Raleigh) and were representative of those reported to cause a deep rot of stored sweet potatoes (7).

Propagule suspensions were prepared by growing the isolate (from mycelial transfers) in Czapek Dox broth on a platform shaker at room temperature (~25 C) for 7 days. The cultures were filtered through four layers of cheesecloth and the propagule density in the filtrate was adjusted on the basis of hemacytometer counts. Seven-day-old PDA plate cultures were used to prepare inocula in the other experiments.

In one experiment, inoculations were made by making an incision with a flamed scalpel, at each end of the tuberous root and then dipping the whole root into a propagule suspension containing 100,000 propagules per milliliter. To compare cultivar susceptibilities to end rot, inoculations also were made by pressing the freshly wounded proximal ends of tuberous roots onto PDA plate cultures of F. solani. To compare the effects of different types of wounds on predisposition of Centennial and Jewel roots to infection, roots were first wounded by either gently scraping away only the periderm, or by scraping and making shallow cuts (1-2 mm) into the cortex or deep cuts (4-5 mm) through the vascular ring. Disks of agar, aseptically removed from the margins of colonies on PDA with a no. 2 cork borer, were inverted onto the surface of the wound.

Inoculated sweet potatoes were incubated at room temperature (20-24 C) in moist chambers made of closed plastic storage boxes lined with moistened paper towels. Measurements of lesion depth, diameter, and fresh weight were made. Fresh weights of lesions were determined by gently scraping off visibly necrotic tissue and weighing it. Four replicate roots were used for each treatment.

To determine their host specificity isolates NC-M-10, NC-N-1, 77-29, 78-25, 78-32, 78-36, and 78-47 of F. solani from sweet potatoes were inoculated to wounded fruits of apple (Malus sylvestris Mill.), cucumber (Cucumis sativus L.), eggplant (Solanum melongena L.), bell pepper (Capsicum frutescens L.), squash (Cucumis pepo L.), tomato (Lycopersicon esculentum Mill.), and white potato tubers (Solanum tuberosum L.) in a manner identical to that used for the wounding experiments.

#### RESULTS

Isolates from Louisiana sweet potatoes with symptoms of surface rot or end rot or sprouts with stem lesions all consistently produced cultures tentatively identified as F. solani.

TABLE 2. Lesion length on tuberous roots of commercial sweet potato cultivars following proximal end inoculation with Louisiana isolate 78-7 of Fusarium solania

Cultivar	Lesion length (mm) <sup>b</sup>			
Goldrush	1 A			
Jewel	2 A			
Heartogold	2 A			
Porto Rico 198	3 A			
Centennial	5 A			
Jasper	20 B			

<sup>&</sup>lt;sup>a</sup>Roots were incubated at 20-24 C for 41 days following inoculation.

TABLE 3. Effect of the type of wounding of sweet potatoes on the occurrence, nature, and extent of decay by isolates of Fusarium solani<sup>alb</sup>

		Cultivar Centennial		Cultivar Jewel			
Isolate	Wound	Lesion diam. (mm)	Lesion depth (mm)	Lesion wt. (g)	Lesion diam. (mm)	Lesion depth (mm)	Lesion wt. (g)
NC-M-10	none	0 A	0 A	0 A	***	****	***
	scrape	26 HI	3 D	1.5 FGH	34 FG	7 CD	7.1 E
	scrape, shallow cut	33 J	4 E	3.6 1	***	***	•••
	scrape, deep cut	33 J	4 E	3.3 I	40 G	13 E	5.4 D
NC-N-I	none	0 A	0 A	0 A		•••	•••
	scrape	15 C	2 C	0.9 CDEF	14 CDE	5 BC	1.5 ABC
	scrape, shallow cut	18 EF	2 C	1.1 DEFG	•••		***
	scrape, deep cut	16 C	2 C	1.1 DEFG	33 FG	10 D	3.0 C
77-29	none	0 A	0 A	0 A	***	***	
	scrape	14 CD	1 B	0.6 ABCD	12 CDE	2 AB	0.4 A
	scrape, shallow cut	16 CDEF	2 C	0.8 CDE	***	***	***
	scrape, deep cut	16 CDEF	4 E	1.1 DEFG	17 E	6 C	1.3 ABC
78-25	none	0 A	0 A	0 A	***	***	500
	scrape	23 GH	2 C	1.1 DEFG	31 F	2 AB	1.8 ABC
	scrape, shallow cut	25 HI	2 C	1.5 FGH			
	scrape, deep cut	28 I	2 C	1.9 H	34 FG	6 C	2.9 BC
78-32	none	0 A	0 A	0 A		···	***
	scrape	16 CDEF	2 C	1.0 CDEFG	2 AB	1 A	0.1 A
	scrape, shallow cut	7 B	1 B	0.4 ABC	***		***
	scrape, deep cut	20 FG	2 C	1.4 EFGH	1 A	1 A	0.1 A
78-36	none	0 A	0 A	0 A		····	***
	scrape	13 C	2 C	0.7 BCD	10 CD	1 A	0.5 A
	scrape, shallow cut	15 CDE	2 C	1.0 CDEFG		***	
	scrape, deep cut	12 C	3 D	0.7 BCD	17 DE	1 A	1.1 AB
78-47	none	0 A	0 A	0 A	***	225	
	scrape	2 AB	1 B	0.1 AB	8 BC	1 A	0.2 A
	scrape, shallow cut	7 B	1 B	0.5 ABCD	•••	***	•••
	scrape, deep cut	18 DEF	2 C	1.6 GH	9 CD	2 AB	0.5 A

Numbers in the same column followed by the same letter are not significantly different at P = 0.05 by Duncan's multiple range test.

Numbers followed by the same letter are not significantly different at P= 0.01 by Duncan's multiple range test. Data are the averages for four replicate sweet potato roots per treatment.

Inoculations of Centennial and Jewel were conducted on separate occasions. Inoculated roots were incubated in moist chambers at 20-24 C for 30 days. Data are the averages for four replicate sweet potato roots per treatment.

Representative isolates were confirmed as F. solani (Sacc.) Mart. emend. Snyd. and Hans. by P. E. Nelson (Fusarium Research Center, The Pennsylvania State University, University Park). The cultural and morphological characteristics of the isolates from sweet potatoes with the different symptoms described above were indistinguishable. On PDA, the Louisiana isolates produced sparse aerial mycelia, abundant microconidia and macroconidia, and a blue to blue-green pigment in the agar. They differed in these respects from the North Carolina isolates of F. solani, which produced abundant aerial mycelia, fewer conidia, and orange to maroon pigments in the agar.

Symptoms of end rot developed slowly at room temperature following inoculation with each isolate of F. solani, whether it originally was isolated from plants showing stem lesions, surface rot, or end rot; Louisiana and North Carolina isolates reacted similarly (Table 1). The end rot of the tuberous root consisted of a dry rot of the cortex and internal storage tissue which was tan to very dark brown (Fig. 1A). In some cases, elliptical cavities with associated macroscopic white mycelial growth developed within the infected tissue. Although they have not been observed on naturally infected samples, some artificially inoculated roots had compact, white tufts of extramatrical growth on the surface. F. solani cultures indistinguishable from the original isolates were reisolated from surface-sterilized, artificially inoculated tissue. Decay that developed at inoculated proximal ends of tuberous roots of cultivar Jasper was significantly greater than that on any of the other cultivars tested (Table 2).

None of the isolates of F. solani tested (78-7, 78-30, 78-32, and 78-36) appeared to affect growth of inoculated terminal vine cuttings; most of the cuttings were free of observable symptoms. Necrosis appeared in some cuttings at the wounded basal end and in the callus tissue at leaf scars (Fig. 1B). This necrosis progressed slowly and its extent apparently was limited. F. solani was recovered only from necrotic tissue.

In preliminary inoculation experiments, Louisiana isolates of F. solani produced symptoms identical to those previously described for surface rot caused by F. oxysporum (2). Lesions were confined to the cortex of the root and were dark brown and mealy. Louisiana and North Carolina isolates produced different symptoms when inouclated side-by-side on the same roots. In all cases decay caused by the Louisiana isolates was confined to the cortex while in many cases that caused by the North Carolina isolates rotted tissue in the cortex, penetrated the vascular ring, and progressed toward the center of the root (Fig. 1C). Results of inoculations to test different isolates and types of wounds indicated that all isolates required wounding of the periderm for initial infection (Table 3). North Carolina isolates penetrated the vascular ring regardless of the depth of the wound while Louisiana isolates required deep wounds that penetrated the cortex before they could invade the pith. Even with deep wounds, only two of the five Louisiana isolates penetrated the vascular ring. F. solani was reisolated from surface rot lesions.

All F. solani isloates tested partially rotted fruits of apple, cucumber, eggplant, bell pepper, squash, tomato, and white potato after 3-4 days of incubation.

### DISCUSSION

F. solani caused a surface rot, sprout lesions, and a previously undescribed end rot of sweet potato. The pathogen required a wound for infection and, in contrast to McClure's isolates (6), it was nonspecialized because it also caused rots on several other unrelated fruits and vegetables.

We suggest the name Fusarium end rot for the end rot disease. Initial observations indicated that end rot was more prevalent on the proximal end of the root, but inoculations of proximal and distal ends resulted in similar amounts of decay (1). This disease has been produced only on cultivar Jasper. Jasper is a recent release, which may account for the disease not being described previously. However, the fact that F. solani isolates from surface rot lesions on other cultivars of sweet potatoes caused end rot indicates that the pathogen was likely present before Jasper was released. Nielsen and Moyer (7) found that their isolates of F. solani were most active at 24-28 C, while Harter et al (3) conducted unsuccessful inoculations with their isolates of F. oxysporum at below 10 C. In this study Fusarium end rot developed well at temperatures of ~20-24 C, but no data were recorded at other temperatures. Harter et al (3) may have been unable to reproduce end rot because incubation temperatures were unfavorable for disease development. Fusarium end rot differs from the "shrivelled end" symptom found on Jasper, which does not progress down the root and for which no cause is known.

The Louisiana isolates of F. solani used in this study differed from the North Carolina isolates in colony appearance and in symptoms produced. Decay caused by the Louisiana isolates did not penetrate beyond the vascular ring unless it was breached by the wound made prior to inoculation. North Carolina isolates readily penetrated the vascular ring of the cultivar Jewel regardless of the depth of wounding, but this occurred less frequently on cultivar Centennial.

Because F. solani is a wound pathogen, proper curing of sweet potatoes immediately after harvest should limit the incidence of surface rot and Fusarium end rot (4,7). F. solani appeared to be a weak pathogen on sprouts and did not appear to affect subsequent growth of vine cuttings. Thus, this pathogen may not be an economic threat to sweet potato production. However, if roots with surface rot or end rot are used for propagation, the decay may become an important factor during presprouting or in seed beds as indicated by Nielsen and Moyer (7).

Storage rots of sweet potato cuased by F. solani and F. oxysporum apparently include a complex of rots which may vary with the sweet potato cultivar, the part of the plant infected, and genotypes of the pathogen.

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