Stem Canker of Sweet Potato Induced by *Fusarium solani*

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ABSTRACT

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A new disease of sweet potato (*Ipomoea batatas*) stems and roots observed on two farms in North Carolina in 1978 had spread more extensively by 1980. Stems were swollen and ruptured near the soil line with a firm, dark brown to black lesion extending from the stem into the pith of tuberous roots. Inoculation of stems and tuberous roots with single conidial isolates of *Fusarium solani* obtained from naturally infected stems resulted in symptoms similar to those observed in the field. Cankers at the soil line extended up to 7.5 cm from the base of the stem (point of attachment to the root). Bioassays demonstrated that colonization occurred in advance of the canker. Necrotic lesions were present on 20 of 60 sprouts produced from infected roots; *F. solani* was isolated from all symptomatic sprouts, from 26 of 40 symptomless sprouts, and from all symptomatic fibrous roots. A survey 4–5 mo after harvest revealed the presence of the *Fusarium* stem-rotting pathogen in tuberous roots at seven of 17 storage facilities. "Seed" roots may be a source of initial inoculum for *F. solani* infection.

Root diseases of sweet potato (*Ipomoea batatas* (L.) Lam.) induced by nonspecific, cortical-rotting *Fusarium oxysporum* Schlecht. and *F. solani* (Mart.) Sacc. emend. Snyd. & Hans. have been considered postharvest diseases (6). These *Fusarium* spp. inhabit most soils where sweet potatoes are grown, and infection enters through wounds occurring at harvest. Symptoms become evident in storage a few weeks after harvest (2,5). "Curing" (2) immediately after harvest is the primary means of preventing decay by *F. solani* and *F. oxysporum* in sweet potato (2,5,6). Stem infections by *F. solani* have been reported under greenhouse conditions following artificial inoculation; however, lesions were limited to tissue below the first node (1,3).

Observations of tuberous roots of the sweet potato cultivar Jewel on two North Carolina farms during the 1978 harvest and on other farms during the 1979 and 1980 harvests revealed the sporadic occurrence of roots with symptoms characteristic of *F. solani* infection. The basal portions of stems from plants with infected roots were necrotic, larger than normal in diameter, and with longitudinal ruptures frequently extending above the soil line. These stem symptoms have not previously been associated with *F. solani* infection of sweet potato; however, they are similar to symptoms incited by *F. oxysporum* f. sp. *batatas* (Wollenw.) Snyd. & Hans. The following investigation describes the etiology of this disease and presents preliminary evidence for a previously undescribed inoculum source of *F. solani*.

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MATERIALS AND METHODS

Isolates. Symptomatic stems, collected during September 1980 from a field being harvested, provided the source of *F. solani* isolates used in these studies except when other sources are indicated. Diseased tissues were washed 15 min in running tap water and surface disinfested in 0.5% sodium hypochlorite for 10 min. Tissue from the lesion margin was incubated on acidified potato-dextrose agar. Single conidial isolates were then selected and maintained on potato-dextrose agar (PDA). An isolate of *F. oxysporum* f. sp. *batatas* used to evaluate sweet potato breeding lines for resistance to the pathogen was also used in this investigation. The identity of all isolates used in this study was graciously confirmed by P. E. Nelson, Fusarium Research Center, Pennsylvania State University, University Park 16802.

Pathogenicity. Inoculum consisted of a suspension of conidia and mycelial fragments of the isolates prepared by homogenizing 7- to 10-day-old PDA cultures in sterile distilled water (100 ml/9-cm petri plate) and straining them through two layers of cheesecloth. Isolates were assayed on tuberous roots and stems of Jewel and Porto Rico 198, cultivars tolerant and susceptible to the pathogen, respectively, for comparison of disease reactions. Roots were inoculated by filling 3-mm-diameter wells in the root cortex with inoculum. The roots were incubated for at least 2 wk in a moist chamber at 20–25 C for symptom development.

Stem cuttings were inoculated by dipping the basal 3 cm in the inoculum, followed by incubation for at least 1 mo in a 10-cm clay pot filled with a pasteurized mixture of sand and Norfolk sandy loam soil (2:1, v/v). Colonization of stem tissue was assayed by excising 2-mm-thick cross sections at 2-cm intervals beginning at the apical end of the stem. Sections were surface sterilized and incubated on PDA at 30 C to observe growth of the pathogen.

Transmission. We surveyed storage facilities during February 1981 to determine whether stem-infecting isolates of *F. solani* were present in roots late in the storage season (4–5 mo after harvest) and to estimate the distribution and prevalence of this pathogen in North Carolina. Jewel roots with symptoms of *F. solani* infection were collected from 17 storage facilities located in Columbus, Duplin, Johnston, Nash, Sampson, and Wilson counties in eastern North Carolina. Initial isolations were made on acidified PDA and cultures were maintained on PDA. Inoculated roots were incubated about 1 mo in a moist chamber and then planted for sprout production in flats 35 × 51 × 8 cm containing a pasteurized mixture of Norfolk sandy loam soil, sand, and peat (3:2:1, v/v). Similarly, symptomless tuberous roots were planted to serve as uninoculated controls. Sprouts were pulled when 30–60 cm long, lesion formation was recorded, and the basal 14 cm of each sprout was assayed for *F. solani*.

RESULTS

The isolates of *F. solani* obtained from symptomatic stem tissue at harvest were pathogenic on tuberous roots and on stem cuttings of Jewel and Porto Rico 198. Necrotic lesions on tuberous roots were circular, radiating from the inoculation site. Lesions had a surface

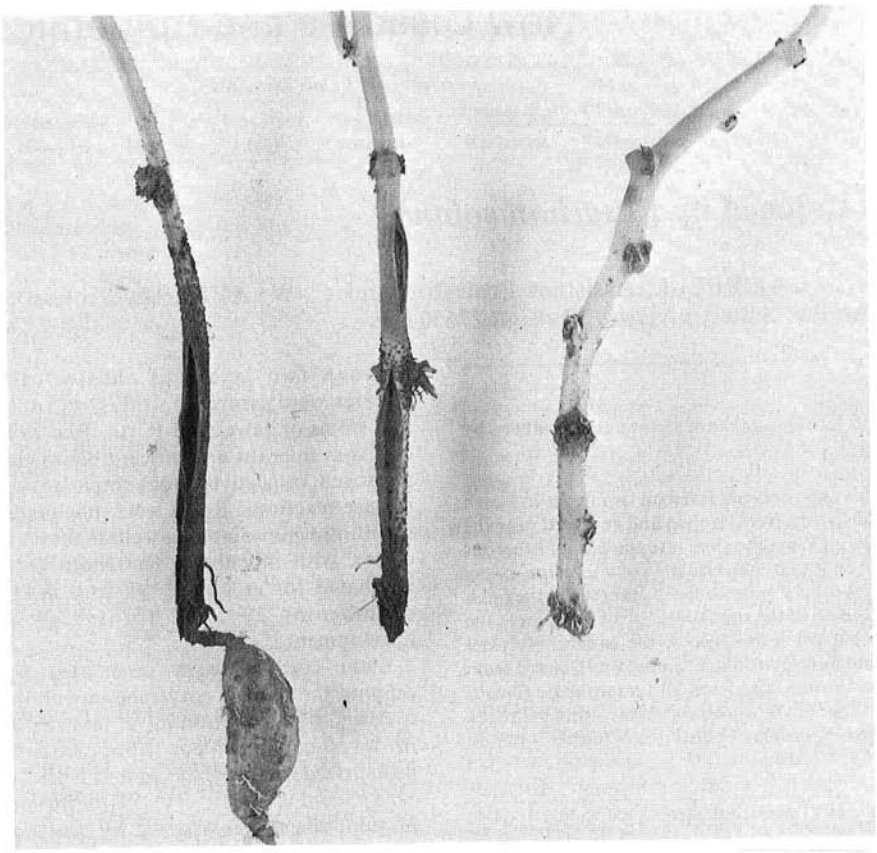

Fig. 1. Sweet potato stems of Jewel about 1 mo after inoculation with *Fusarium solani* isolated from symptomatic stem tissue. Necrosis extending apically and basipetally into tuberous root, and stem with rupture (left); limited necrosis with stem ruptures (center); healthy, uninoculated stem (right).

diameter of 2–3 cm in about 7 days; some isolates were limited by the vascular ring, and some were not. Inoculated stem cuttings exhibited necrotic lesions extending one to two nodes from the base of the cutting (Fig. 1). Longitudinal ruptures of the stems in advance of the lesions were frequently observed about 1 mo after inoculation. These isolates of *F. solani* produced fine, aerial mycelia on PDA with red to pink pigmentation. This response was distinct from that of the *F. oxysporum* f. sp. *batatas* isolate, which had coarse, depressed mycelia with deep purple pigmentation.

Stem symptoms induced on Jewel by isolates of *F. solani* and *F. oxysporum* f. sp. *batatas* were similar, with the necrosis induced by each species extending 4–5 cm from the base of Jewel stems. Six of 40 Jewel plants inoculated with *F. oxysporum* f. sp. *batatas* died before the experiment was ended. Similar symptoms were induced by *F. solani* on Porto Rico; however, 28 of 30 Porto Rico plants were killed by *F. oxysporum* f. sp. *batatas*. Serial bioassays demonstrated that both organisms colonized Jewel sprouts 4–16 cm in advance of the necrosis. Separate cultures of *F. solani* were collected from four serial sections immediately distal to the canker from one Jewel and one Porto Rico plant. Each isolate of *F. solani* caused typical cortical decay when assayed on tuberous roots. Roots inoculated with *F. oxysporum* f. sp. *batatas* remained symptomless.

We collected 68 isolates of *F. solani* pathogenic on storage roots during the survey. Most isolates induced lesions limited by the vascular ring; however, some isolates freely penetrated into the pith. Nineteen of 68 isolates induced stem infections above the first node; the remaining isolates caused limited necrosis at the base of the stem. At least one isolate of *F. solani* was obtained from each county surveyed and from seven of the 17 storage facilities. The 68 isolates collected from storage roots had cultural morphologies similar to the stem isolate. Pigmentation varied among isolates, with a few being pink to red, but most isolates were tan. Stem-infecting isolates were not confined to one pigmentation.

Tuberous roots infected with *F. solani* produced sprouts that were also infected. Necrotic lesions on sprouts extended up to 7.5 cm from the base (point of attachment to root) on 20 of 60 sprouts, and colonization by *F. solani* was detected up to 4 cm in advance of the lesions. *F. solani* was also detected in symptomless sprouts; 46 of 60 sprouts were found to be infected. The organism was also isolated from necrotic lesions on fibrous roots produced by the sprouts, and these isolates also produced typical cortical necrosis when assayed on tuberous roots. No necrosis was observed on sprouts produced from uninoculated roots, nor was there evidence of systemic infection by *F. solani*.

DISCUSSION

Infection of sweet potato stems by *F. solani* has not been previously recognized under field conditions. Such infections may suppress yield through reduced root production and rotting of tuberous roots. Furthermore, infected roots in the field are a potential inoculum source for additional root infections at harvest that may not become apparent until after roots are stored. About 28% of the *F. solani* isolates obtained from storage facilities caused significant stem infections when infected roots were used for propagation.

The remaining *F. solani* isolates were similar to those reported by others (1,3,5) and could infect tuberous roots. Some were limited to the cortex, while others colonized the pith and eventually decayed the entire root (1,5). Stem necrosis, if present, was limited to tissue below the first node. Pathogenicity tests with the 68 isolates from the survey did not suggest any correlation between the type of root rot and the degree of stem necrosis. Colony morphology and pigmentation of the isolates were consistent with previous reports of *F. solani* isolates obtained from sweet potato (1,5).

Systemic colonization of stems from mother roots suggests that "seed" roots can serve as a source of initial inoculum. Propagation material has been reported as a source of *F. solani* inoculum for other crops. The organism is carried on true seed (7) as well as vegetative tissues (4). This may explain reports by farmers of the sporadic occurrence of this disease in fields not previously planted to sweet potato. Thus, growers should avoid using infected roots as a propagation source and should continue present sanitation practices at harvest to prevent new infections.

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