Reactions of Sweet Potato Selections to Fusarium Root and Stem Canker Caused by *Fusarium solani*

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ABSTRACT

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Sweet potato (*Ipomoea batatas*) cultivars, advanced selections, and breeding lines were evaluated for reactions of fleshy storage roots and vine cuttings to Fusarium root and stem canker. Four root rot reaction types were observed that ranged from a small, restricted lesion type limited by the vascular ring to a large lesion type that extended beyond the vascular ring into the center of the root. Reactions of most selections to vine inoculations consisted of decay restricted to the leaf scars and basal internodes immediately above the cuts. Stem canker reactions for two sweet potato selections were variable and included some instances where decay extended into internode tissue.

Fusarium root and stem canker (FRSC) has recently been reported as a problem in sweet potato (*Ipomoea batatas* L.) production in the southeastern United States (1,6,9). External symptoms on fleshy storage roots are similar to those of surface rot, a disease caused by strains of *Fusarium oxysporum* and *F. solani* (Sacc.) Mart. emend. Snyd. & Hans. that has been a minor storage problem in sweet potatoes for many years (2,4). In contrast, the root rot phase of FRSC develops to a greater extent in storage by decaying the root beyond the vascular ring into the center of the root (9). In addition, the FRSC pathogen, *F. solani*, has been observed to infect the sprouts produced on infected "seed" roots and cause a stem canker that may continue to develop after "slips" are transplanted to the field (6). FRSC differs from the previously reported Fusarium foot rot in that it has not been observed to affect the fibrous roots (5). Crop losses may result from either reduced plant production by infected seed roots, reduced growth and yield in the field caused by stem canker, or decay of sweet potatoes in the field and in storage.

Previous work indicated that sweet potato germ plasm varied in reaction to the surface rot pathogen (3,7,8,10). This was not pursued as a means of disease control, however, because surface rot could be managed by reducing physical injury during harvest and by properly curing the sweet potatoes immediately after harvest. Although the same measures also reduce FRSC, the report that the pathogen may be transmitted on infected slips makes it possible for the pathogen to circumvent these controls to a limited extent. Reports also suggest that commercial sweet potato cultivars vary in their reactions to the root rot phase of the disease (1,9). This work was undertaken to refine screening methods for FRSC and to assess the range of reactions to both phases of the disease in germ plasm currently used in sweet potato breeding programs in the United States.

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MATERIALS AND METHODS

Isolates of *F. solani* were obtained from naturally infected roots or vines of sweet potato from Louisiana (77-29, 78-7, 78-32, 78-36, 78-47, 83-1, 83-3, 84-50, 84-51, 84-52, 84-53, 84-54, 84-56, 84-58, 84-59, 84-69, 84-71, 84-76, 84-77, and 84-78) or Alabama (85-1 and 85-4); P. E. Nelson, Fusarium Research Center, Pennsylvania State University (S-128, S-564, S-565, S-566, S-567, S-662, S-663, S-664, and S-665); or J. W. Moyer, Department of Plant Pathology, North Carolina State University (N-1, M-10, B2R, and DB-85). Fifteen isolates that produced lesions on inoculated Jewel sweet potato storage root slices in petri dish moist chambers were then compared for relative root rot and stem canker virulence by inoculating fleshy storage roots of the cultivar Jewel and vine cuttings of Jewel and selection L82-527.

Fleshy storage roots were inoculated by gently scraping the periderm from a spot on the median of the root, cutting a 5-mm-diameter plug with a cork borer from the margin of a 5-day-old potato-dextrose agar (PDA) culture, and placing the plug (mycelium side down) on the wound. Inoculated roots were incubated at 16–20 C for 2 days in plastic bags containing a moistened paper towel and then transferred to paper bags for 4–6 wk. Evaluations were made by measuring the external diameter and depth of each lesion and determining whether it extended beyond the vascular ring. Vine cuttings were inoculated by briefly touching the basal end of the freshly cut vine to the surface of a 5-day-old PDA culture of the isolate. The cuttings were planted in the greenhouse in 15-cm-diameter clay pots containing 1:1 sand-soil mix and evaluated for lesion length after 6 wk. Isolate M-10, originally from North Carolina, and isolate 85-4, originally from an infected stem from Alabama, were selected as the most virulent isolates for evaluating host reactions to root rot and stem canker, respectively.

Two tests were conducted to evaluate the reactions of different sweet potato genotypes to root rot, using isolate M-10 with the storage root inoculation procedure described before. The first was inoculated on 5 April 1984, using six stored roots per entry, and was evaluated on 17 May 1984. The second test was inoculated on 6 November 1984, using 10 roots per entry harvested the same day, and was evaluated on 26 February 1985.

Vine cuttings were inoculated differently for comparisons of host reactions than for isolate comparisons. All expanded leaves and petioles were removed and the freshly cut vine cuttings were dipped in a conidial suspension of isolate 85-4 (10^6 propagules per milliliter) prepared by rinsing 5-day-old PDA cultures with sterile distilled water and filtering through four layers of cheesecloth. Five inoculated cuttings were planted per 15-cm clay pot containing a 1:1 sand-soil mix, and the plants were grown in the greenhouse for 6 wk. They were then washed and rated for necrosis using a scale of 0–5, where 0 = no necrosis and 5 = plant dead. Two tests were conducted, one inoculated on 26 March 1985 and the other on 20 August 1985.

RESULTS

Root rot development was more rapid in the first test than in the second, but for most genotypes, the reactions were similar in both tests. In the first test, mean lesion diameters for different genotypes ranged from 19 to 113 mm and mean lesion depth ranged from 1 to 37 mm. Lesion diameter in the second test ranged

from 13 to 85 mm and lesion depth from 1 to 19 mm. Four reaction categories were defined in each test on the basis of lesion diameter and depth and frequency of penetration of the vascular ring, where I = small-diameter (less than 20 mm), shallow (less than 3 mm) lesions that did not penetrate the vascular ring; II = intermediate diameter (20–40 mm), shallow lesions that did not penetrate the vascular ring; III = lesions of intermediate diameter and depth (7–15 mm) that frequently penetrated the vascular ring; and IV = large-diameter (greater than 60 mm), deep (greater than 15 mm) lesions that frequently penetrated the vascular ring and often had extramatrical growth of the pathogen. Genotypes in category I were L0-93, L81-6, L81-8, L82-502, L82-505, L82-506, W-184, and W-193. Genotypes in category II were Centennial, Jasper, Porto Rico, L8-10, L8-68, L79-36, L0-22, L0-106, L82-504, L82-514, L82-521, L82-524, T-17-2, T-17-3, T-30-6, W-151, W-191, W-196, W-199, W-220, and W-81-54. Genotypes in category III were Goldrush, Heartogold, Jewel, L8-21, L0-28, L81-10, L81-11, L82-165, W-179, and W-192. Genotypes in category IV were Travis, L81-3, T-25-30-3, and 8W2641. Table 1 gives the average lesion measurements combined from both tests of the named cultivars that were evaluated.

Table 1. Average lesion diameter, depth, and percent frequency of vascular cambium penetration after inoculation of storage roots with *Fusarium solani* isolate M-10 for the commercial sweet potato cultivars evaluated in two tests for root rot reaction

Cultivar	Lesion diameter (mm)	Lesion depth (mm)	Penetration of vascular cambium (%)
Jasper	25	3	10
Porto Rico	30	7	28
Centennial	53	10	35
Goldrush	39	6	55
Jewel	38	15	50
Heartogold	52	13	58
Travis	85	29	80

Two sources of variability in reactions were noted for various genotypes after storage root inoculations with isolate M-10. Although the reactions for most genotypes were comparable, cultivar Porto Rico differed in reactions between the two tests. Lesion depth was 2 mm in the first test but 11 mm in the second. Cultivar Jewel varied considerably in reactions among individual roots within each test. These reactions were studied further by inoculating 144 Jewel fleshy storage roots on 10 November 1984 with isolate M-10 and measuring lesion diameter on 11 March 1985. Figure 1 shows the bimodal distribution of lesion diameters for those roots, with peaks at 5–10 and 85–90 mm in diameter. The rate of enlargement in lesion diameter was measured on an additional 18 roots inoculated on 26 April 1985. Diameters of the lesions were measured at 3- to 4-day intervals until 3 June 1985. Lesion enlargement on five of the roots ceased within 4–17 days of inoculation, with a final average lesion diameter of 10 mm. Lesion enlargement continued on the remaining 13 roots throughout the period, with a final average lesion diameter of 47 mm.

Two of the 20 entries screened in the first stem canker test, L82-527 and L83-514, had significantly higher severity ratings than the other entries (Table 2). Entry L81-9 had a significantly higher severity rating than the other entries in the second test. Necrosis was restricted to wounded tissue (the callus that developed on leaf scars and the tissue from the cut to the first node) on most of the entries in both tests, and results for such entries were consistent between the two tests. However, reactions for selections L81-9 and L82-527 varied between the tests. In test 2, L81-9 had additional necrosis in the internodal tissue not found in test 1, and the converse was observed for L82-527.

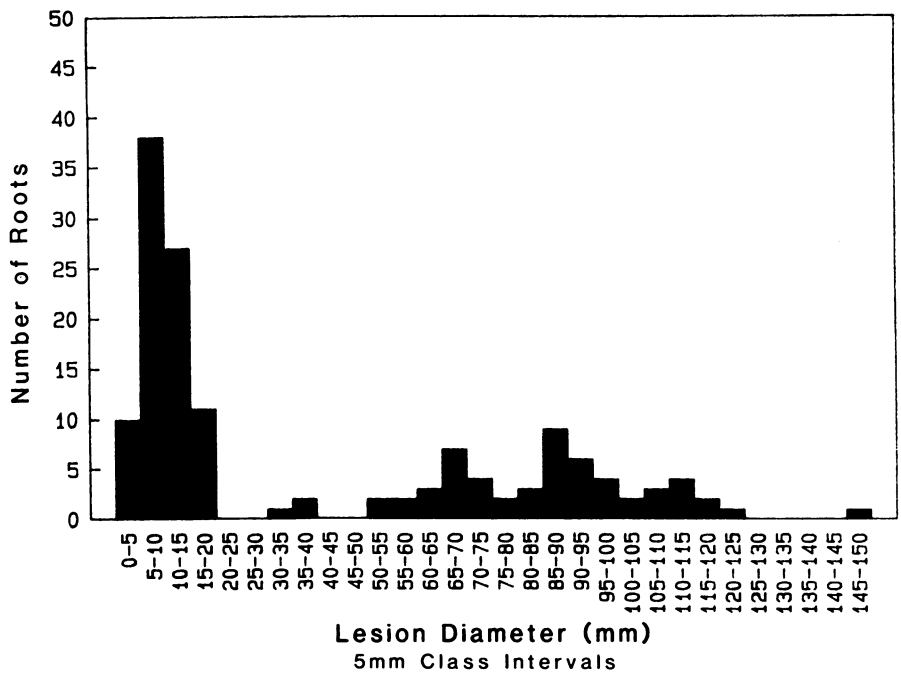

Fig. 1. Histogram showing the distribution of lesion diameters on fleshy storage roots of sweet potato cultivar Jewel after inoculation with *Fusarium solani* isolate M-10 by 5-mm class intervals.

DISCUSSION

Sweet potato germ plasm varies significantly in reaction to the root rot phase of FRSC, and although none of the genotypes appeared immune to the

Table 2. Mean stem canker severity ratings of sweet potato genotypes 6 wk after inoculation of vine cuttings in the greenhouse with *Fusarium solani* isolate 85-4

Selection	Mean severity[y]
Test 1	
L82-531	0.0 f[z]
L82-19	0.2 f
L82-508	0.2 f
L83-510	0.4 ef
L83-521	0.4 ef
L83-523	0.4 ef
Centennial	0.8 de
Jasper	0.8 de
L82-66	0.8 de
Jewel	1.0 cd
L81-9	1.0 cd
L83-502	1.0 cd
L83-504	1.0 cd
L83-523	1.0 cd
L80-62	1.4 c
L80-15	1.4 c
L81-10	1.4 c
L82-509	1.4 c
L83-514	2.2 b
L82-527	3.0 a
Test 2	
W-151	0.6 g
L82-527	0.6 g
W-192	0.7 g
L80-15	1.0 efg
L82-66	1.0 efg
W-220	1.0 efg
Ti80-130	1.0 efg
L81-8	1.1 defg
L81-10	1.1 defg
Jasper	1.2 def
Jewel	1.2 def
MD-708	1.3 cde
Heartogold	1.3 cde
Porto Rico	1.3 cde
Centennial	1.5 bcde
L81-3	1.5 bcde
Travis	1.6 bcd
Goldrush	1.8 bc
W-191	1.9 b
L80-62	1.9 b
L81-9	2.7 a

[y]Severity was rated using a 0–5 scale for stem necrosis where 0 = no necrosis and 5 = plant dead.

[z]Numbers in the same column followed by a common letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

pathogen, some selections showed a relatively high degree of resistance to this phase of the disease. As suggested previously, the increased occurrence of FRSC during recent years may be related to the increased use of sweet potato cultivars such as Travis and Jewel, which are more susceptible to the root rot phase of the disease than some of the older cultivars such as Porto Rico (1,9).

Most genotypes evaluated for reactions to the stem canker phase of the disease appeared relatively resistant. This may account for the less frequent observation of the stem canker phase of the disease under field conditions, even when significant root rot has been observed (1,6). The variable reactions of some entries may indicate that environmental factors not controlled in these experiments, such as physiology of the vines at the time they were cut and inoculated, may affect the severity of stem canker development.

Differences in reactions among selections to FRSC were qualitative as well as quantitative. Apparently, sweet potato can restrict development of the pathogen at specific sites within the plant. Development of stem canker was restricted at the nodes of the stem. In some instances, root rot was restricted by the vascular ring to superficial lesions within the cortex of the root. Even where the vascular ring was eventually penetrated, lesion enlargement was apparently delayed at the vascular ring. Because the vascular ring of the sweet potato fleshy storage root is a zone of meristematic activity, it may be that active cell division is involved in the restriction of the pathogen at this site. However, this region also differs from the rest of the root in that it contains more laticifers and fibers.

Several major sources of variation have been noted with respect to FRSC development. Some isolates of *F. solani* induce only small, restricted lesions, some cause a surface rot, others an aggressive root rot, and still others an aggressive root rot and stem canker (1,6). Differences in reactions to the more aggressive isolates of *F. solani* among sweet potato genotypes, among roots within an individual genotype, or between roots of a cultivar such as Porto Rico from different harvests resulted in similar differences in symptomatology. These sources of variation should prove useful in studying mechanisms of resistance of sweet potato to FRSC. The pattern of overall variation suggests the possibility that FRSC reaction is determined by the relative rate of pathogen development in the tissue in relation to the rate of response by the sweet potato resulting in the development of either a restricted lesion or a lesion that continues to expand.

Although this paper and others indicate the existence of resistance to FRSC, further work is needed to assess the significance of the available levels of resistance to efforts to control the disease (1,9). The severity of disease development may be less important than the incidence of disease under natural conditions, because any blemish may reduce the marketability of the sweet potatoes. It will be especially important to determine the frequency of transmission of the pathogen through the propagating cycle and the incidence of the root rot phase under field conditions for the different reaction categories.

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