Effect of Resistance to *Streptomyces ipomoeae* on Disease, Yield, and Dry Matter Partitioning in Sweetpotato

JEAN B. RISTAINO, Assistant Professor, Department of Plant Pathology, North Carolina State University, Raleigh 27695-7616

ABSTRACT

Ristaino, J. B. 1993. Effect of resistance to *Streptomyces ipomoeae* on disease, yield, and dry matter partitioning in sweetpotato. Plant Dis. 77:193-196.

The effect of resistance to *Streptomyces ipomoeae* on disease, yield, and dry matter partitioning in two sweetpotato (*Ipomoea batatas*) cultivars commonly grown in North Carolina was evaluated in field studies conducted over 3 yr. Cultivar Jewel, which is susceptible to *S. ipomoeae,* and cultivar Beauregard, which is resistant, were planted in field plots that were not infested or infested with *S. ipomoeae.* The percentage of diseased storage roots produced per plot and the severity of disease on fibrous roots were greater on Jewel than on Beauregard in each of the 3 yr. Jewel plants produced significantly more storage roots and more diseased storage roots per plant than Beauregard plants. Marketable yield was not affected by disease because disease incidence on storage roots was less than 20%. Dry matter production at harvest was greater in both shoots and fibrous roots of Jewel than of Beauregard. Beauregard partitioned less dry matter to both shoots and fibrous roots over the season than Jewel. Disease did not affect dry matter partitioning to shoots or fibrous roots over time because incidence was low. However, disease significantly reduced dry weights of storage roots of Beauregard at harvest compared to noninoculated controls in 2 of 3 yr. Roots of Beauregard plants may have contacted less inoculum in soil because their fibrous root systems were smaller than those of Jewel plants. In addition, *S. ipomoeae* colonized fibrous roots of Beauregard less extensively than it colonized those of Jewel. These results may be useful in understanding resistance to *S. ipomoeae* in Beauregard. Further studies with more sweetpotato genotypes are needed to critically evaluate the role of fibrous root development in resistance to Streptomyces soil rot. Although disease incidence was low, the techniques described may be useful to plant breeders interested in selecting for resistance to Streptomyces soil rot when field nurseries with uniform natural inoculum are not available.

Additional keywords: pox, actinomycete

Streptomyces soil rot (pox), caused by *Streptomyces ipomoeae* (Person & W. J. Martin) Waksman & Henrici), is an important disease in many sweetpotato (*Ipomoea batatas*) production areas in the United States (4). The pathogen infects both fibrous roots and storage roots and can reduce the yield and quality of storage roots (7,14,15). Management of the disease primarily involves the use of resistant cultivars; however, integrated approaches including soil fumigation, crop rotation, and reduction of soil pH with sulfur are also recommended (5,9,11,15).

Cultivars are available with resistance to Streptomyces soil rot (6,8,10,13,16); however, the disease continues to cause losses in North Carolina because the cultivar Jewel, which is susceptible to the disease, is extensively grown there. The cultivar Beauregard, with intermediate resistance to *S. ipomoea,* was released by the Louisiana Agricultural Experiment Station in 1987 (16) and has been more widely grown in recent years, especially in fields known to be infested with *S. ipomoeae.* The level of resistance to Streptomyces soil rot described in Beauregard is similar to that of the cultivar Travis but less than that of Jasper (6,10,16).

Little is known about the effect of resistance on the components of disease or about differences between Jewel and Beauregard in dry matter partitioning. In addition, only one study has quantitatively compared the yield characteristics of resistant Beauregard and susceptible Jewel in the field (16).

The objective of this work was to evaluate the effect of resistance to *S. ipomoeae* on disease, yield, and dry matter partitioning in sweetpotato. Jewel and Beauregard were compared in order to quantify possible components of resistance to disease.

Accepted for publication 24 September 1992.

MATERIALS AND METHODS

Inoculum preparation. Stock cultures of *S. ipomoeae* were maintained on silica gel at 5 C (17). An isolate of *S. ipomoeae* (78-57) pathogenic to sweetpotato and originally isolated from North Carolina was obtained from C. A. Clark (Louisiana State University) and used to infest field plots. Cultures were grown on *Streptomyces* growth medium at 32 C for 8–10 days before transfer (3). Inoculum for use in field experiments was prepared by culturing the pathogen at 32 C for 4 wk in 500 cm^3 of vermiculite and 375 ml of broth (5 g of mannitol, 1 g of sodium propionate, 0.2 g of K_2HPO_4, 0.2 g of $MgSO_4 \cdot 7H_2O$, 1 g of yeast extract, 2.0 g of $CaCO_3$, 0.11 mg of $CoCl_2$, and 1.0 L of distilled water) in 1.0-L mason jars (C. A. Clark, *personal communication*). Inoculum consisted of aerial mycelia and spores of the pathogen in a vermiculite carrier.

Field experiments. Sweetpotatoes were grown following recommended cultural practices (18). To produce transplants for use in the field, storage roots of Jewel and Beauregard were dipped in Mertect 340 F (Merck and Co., Rahway, NJ) at the labeled rate of 8.36 ml/L of water (0.46 kg a.i./L) and planted in early April in beds previously fumigated with methyl bromide-chloropicrin (MB-33, 3.6 kg per 83.6 m^2) (Great Lakes Chemical Co., West Lafayette, IN). After the roots were planted, beds were covered with clear polyethylene (0.025 mm thick) that was punctured for ventilation. The polyethylene was removed after plant emergence.

Experiments were conducted on sandy loam soils at the Central Crops Research Station in Clayton, NC. The field in Clayton was fumigated with methyl bromide-chloropicrin (MB-33, 3.6 kg per 83.6 m^2) in the spring before planting. Plots were 9.1 m long in 1989 and 12.2 m long in 1990 and 1991. In each year, plots consisted of two single-row beds that were each 1.1 m wide. The experiment was arranged in a split-plot design, and all treatments were replicated four times.

Main plots were infested with inoculum of *S. ipomoeae* grown on the vermiculite-broth growth medium at a rate of 212 cm^3 per meter of row. A furrow was dug in the center of each row, and inoculum was buried approximately 10 cm below the soil surface. Beds were reshaped with a disk hiller after infestation. Plots were infested at the time of transplanting.

Subplots were planted with sweetpotato sprouts of Jewel or Beauregard that were cut from the plant beds in June (8 wk after beds were planted). Plants within the rows were spaced 30.5 cm apart. Border plots of the same size as experimental plots were also planted. Immediately after transplantation, all plots were irrigated with overhead irrigation to establish plants.

Data collection. Five soil cores (1.9 cm in diameter and 20 cm deep) were sampled from each plot twice during the season to measure soil pH in each field. The top 5 cm of soil from each core was discarded, and cores from each plot were combined into a composite sample. Standard methods were used to measure soil pH (12). Mean soil pH in the three fields ranged from 5.7 to 6.3 during the season.

Three to nine plants were sampled periodically and at harvest from one of the two rows in each experimental unit. Roots were dug to a depth of 30 cm below each sampled plant. Disease severity was evaluated visually on fibrous roots of individually sampled plants and was rated on a scale of 0 to 4, where 0 = no lesions, 1 = <25% of the fibrous root system with lesions, 2 = 26–50%, 3 = 51–75%, and 4 = >75% of the fibrous root system with lesions. The total number of storage roots and diseased storage roots and the dry weight of fibrous roots, storage roots, and shoots (vines and leaves) were measured on each sampled plant. Weights were determined after drying tissue at 60 C for about 1 wk. Storage roots were sliced to facilitate drying. At least two plants were left between sample locations to avoid problems with plant growth compensation resulting from removal of previously sampled plants.

Yield was measured in the second row of each experimental unit at harvest. Total yield (U.S. #1s, jumbos, and canners), yield of marketable storage roots (free of lesions), and yield of diseased storage roots were determined by fresh weight. The percentage by weight of the total yield of storage roots affected by disease was calculated.

Statistical analysis. Separate analyses of variance were conducted for data from each field experiment with the general linear models program from the Statistical Analysis System (SAS Institute, Cary, NC). Means were separated with least significant difference tests when appropriate.

RESULTS

Jewel plants had a significantly greater percentage of diseased storage roots per plot than Beauregard plants in 1989 and 1990 (inoculum × cultivar effect significant at $P < 0.01$) (Fig. 1A). The severity of disease on fibrous roots also was significantly higher for Jewel than for Beauregard in 1990 and 1991 (inoculum × cultivar interaction significant at $P < 0.05$) (Fig. 1B), whereas the inoculum main effect was significant in 1989 ($P < 0.05$). The percentage of diseased storage roots was highest and fibrous root disease was most severe in the susceptible cultivar Jewel in 1990 (Fig. 1).

In each year, individual plants of Jewel produced significantly more storage roots than plants of Beauregard (Fig. 2A) (cultivar main effect significant at $P < 0.01$). In addition, Jewel plants produced significantly more diseased storage roots than Beauregard plants (Fig. 2B) (inoculum × cultivar effect significant at $P < 0.01$).

Disease did not significantly affect marketable yield in any year (Table 1). However, in 1989, marketable yield of Beauregard was significantly lower in noninfested plots than marketable yield of Jewel in infested or noninfested plots.

Dry matter production at harvest in

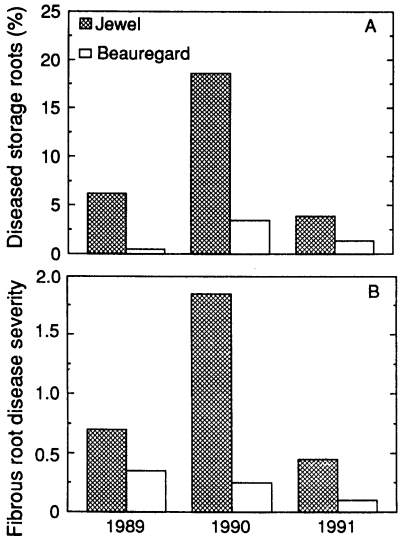

Fig. 1. The effect of sweetpotato cultivar on the percentage of diseased storage roots infected with *Streptomyces ipomoeae* **(A)** and on the severity of disease on fibrous roots **(B)** in 1989, 1990, and 1991. The inoculum × cultivar interactions were significant at $P < 0.01$ in 1989 and 1990 and $P < 0.05$ in 1990 and 1991 for the percentage of diseased storage roots and the severity of disease on fibrous roots, respectively.

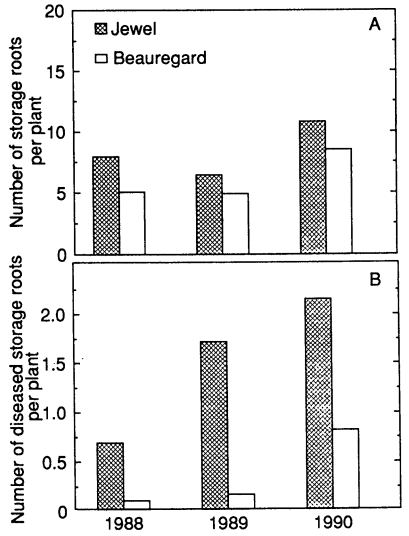

Fig. 2. The effect of sweetpotato cultivar on the number of storage roots **(A)** and diseased storage roots **(B)** produced per plant in 1989, 1990, and 1991. The cultivar main effect was significant at $P < 0.01$ for the number of storage roots per plant, and the inoculum × cultivar interaction effect was significant at $P < 0.01$ for the number of diseased storage roots produced per plant in each year.

both shoots and fibrous roots was greater in Jewel than in Beauregard in each year (cultivar effect significant at $P < 0.01$ and 0.0001, respectively) (Fig. 3). Disease did not reduce dry weights of shoots or fibrous roots in any year. However, in both 1990 and 1991, disease significantly reduced dry weights of storage roots of Beauregard plants but not of Jewel plants at harvest (cultivar × inoculum interaction effect significant at $P < 0.05$) (Fig. 4).

Beauregard partitioned less dry matter to shoots than Jewel between 63 and 118 days after planting (Fig. 5A) (cultivar main effect significant at $P < 0.01$). Similarly, Beauregard partitioned significantly less dry matter to fibrous roots than Jewel from 42 to 118 days after planting (Fig. 5B) (cultivar main effect significant at $P < 0.01$). Partitioning of dry matter to storage roots was greater in Beauregard than in Jewel at 42 and 63 days after planting (cultivar main effect significant at $P < 0.05$) but was similar at other sample times (Fig. 5C). Disease did not affect partitioning of dry matter over time to shoots or fibrous roots of either cultivar.

DISCUSSION

The percentage of diseased storage roots per plot and the number of diseased storage roots per plant were greater and disease on fibrous roots was more severe on Jewel than on Beauregard in 3 yr of field evaluation. These results support the work of others and confirm that storage roots and fibrous roots of Beauregard are more resistant to disease than those of Jewel (16). Storage roots of Jewel generally contained more lesions than those of Beauregard, and the lesions were more severe than those on Beauregard (J. B. Ristaino, *unpublished*).

Using the infestation methods described in this and previous research (15), we were able to obtain low levels of disease in plots. The data also demonstrate statistical differences between the cultivars in the components of disease measured on storage and fibrous roots.

Table 1. Marketable yield (kg/ha) of sweetpotato cultivars Jewel (susceptible to *Streptomyces ipomoeae*) and Beauregard (intermediate in resistance to *S. ipomoeae*) in three field tests

Cultivar	Year[b]		
Inoculated[a]	1989	1990	1991
Jewel			
Yes	27,308	18,314	27,040
No	23,137	18,933	30,458
Beauregard			
Yes	21,293	19,098	26,177
No	14,708	18,521	26,836

[a] Inoculated plots were infested with inoculum of *S. ipomoeae* at the rate of 212 cm^3 per meter of row.

[b] $LSD_{0.05}$ = 9,264 in 1989. Means were not significantly different in 1990 and 1991.

These techniques may be useful to plant breeders interested in selecting for resistance to *S. ipomoeae* when field nurseries with uniform natural inoculum of the pathogen are not readily available. However, use of more virulent isolates of the pathogen and repeated infestation of soil with the pathogen over several seasons may be necessary to obtain higher levels of disease than those reported here.

Disease did not reduce the marketable yield of either cultivar examined. These results are not surprising, because in our previous work we did not observe reductions in marketable yield in Jewel until the percentage of diseased storage roots exceeded 20% (15). High disease incidence in fields with natural inoculum of *S. ipomoeae* can have negative effects on yield of Jewel and other susceptible cultivars (7,15).

The two cultivars differed in dry matter accumulation over the season and at harvest. Plants of Beauregard partitioned less dry matter to both shoots and fibrous roots than plants of Jewel over time, and fibrous root systems of Beauregard were smaller than those of Jewel at harvest. Because fibrous root growth through soil was less extensive in Beauregard than in Jewel, fibrous roots of Beauregard may have contacted less inoculum in soil. This may partly explain the level of disease resistance observed in the fibrous roots of Beauregard. In addition, *S. ipomoeae* did not colonize the fibrous roots of Beauregard as extensively as it did the fibrous roots of Jewel. In contrast, others (2,8) have proposed that sweetpotato breeding lines with faster root growth are more tolerant to disease than plants with slower root growth and thus escape inoculum in the soil. However, previous researchers used different genotypes of sweetpotato than those evaluated here. Increased root growth could enable Jewel to come into contact with more inoculum in the soil and thus to sustain higher levels of disease than Beauregard.

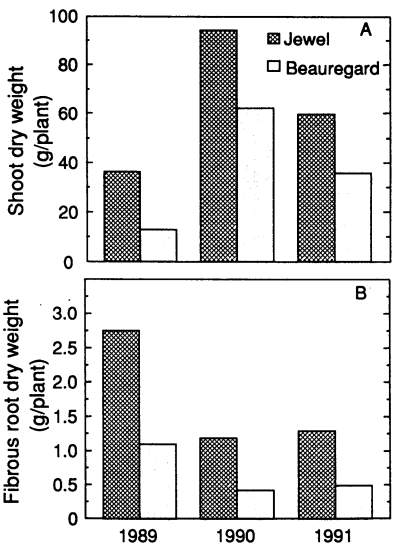

Fig. 3. The effect of sweetpotato cultivar on the dry weight at harvest of shoots **(A)** and fibrous roots **(B)**. The cultivar main effect was significant for shoots and fibrous roots at $P < 0.01$ and 0.0001, respectively, in each year.

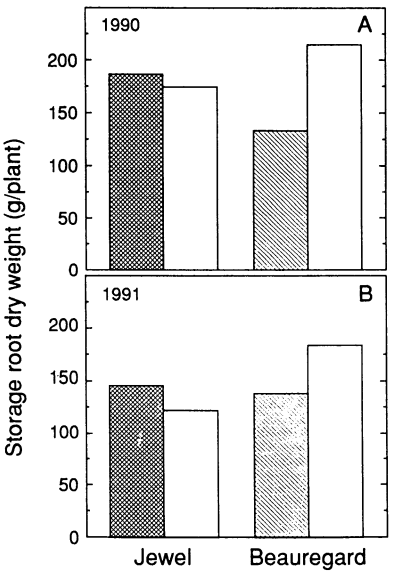

Fig. 4. The effect of sweetpotato cultivar on the dry weight of storage roots in infested (shaded bars) and noninfested (open bars) plots in 1990 **(A)** and 1991 **(B)**. The cultivar × inoculum interaction was significant in each year at $P < 0.05$.

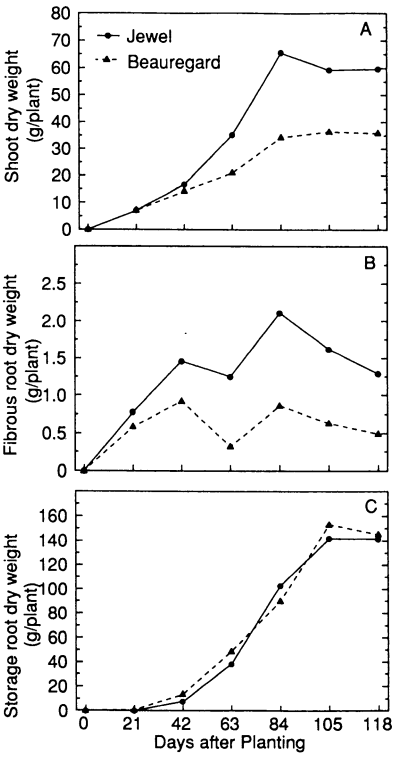

Fig. 5. The effect of sweetpotato cultivar on the accumulation of dry matter in shoots **(A)**, fibrous roots **(B)**, and storage roots **(C)** over time. The cultivar main effect was significant in shoots between 63 and 118 days after planting, in fibrous roots between 42 and 118 days after planting, and in storage roots at 42 and 63 days after planting.

The ability of diseased plants to regenerate roots in soil also may affect resistance to this and other soilborne pathogens (1) but was not examined in this study. In addition, separate forms of resistance to *S. ipomoeae* may occur in fibrous roots and storage roots (13), and if so, growth characteristics of fibrous roots may only partly explain resistance in Beauregard. Because studies of dry matter partitioning of roots in the field are very labor-intensive, these initial studies were done with the two sweetpotato cultivars most commonly grown in North Carolina. However, further studies in the field with more sweetpotato genotypes are needed to critically evaluate the role of fibrous root development in resistance to Streptomyces soil rot.

Accumulation of dry matter in storage roots was similar in both cultivars in the absence of disease. In the presence of disease, however, storage roots of resistant Beauregard were smaller than those of susceptible Jewel in 2 of 3 yr. The apparent response of storage root growth to low levels of disease differed between the cultivars and needs further examination.

Disease incidence in this study was lower than that reported by others (5,7,10,13,15). Studies are in progress to more closely examine the effect of higher levels of disease caused by *S. ipomoeae* in artificially infested plots on fibrous root growth, storage root development, and water extraction by these cultivars in the field. Because Streptomyces soil rot is probably a monocyclic disease in the field, initial growth of roots to contact inoculum in soil and the spatial pattern of initial inoculum in naturally infested fields could have large effects on subsequent epidemic development. Disease is often spatially aggregated in grower fields with natural inoculum in North Carolina. This spatial aggregation of disease could make evaluation of host resistance in such fields difficult unless factors affecting spatial patterns of inoculum occurrence are understood.

ACKNOWLEDGMENTS

I am grateful for the technical assistance of G. Parra, M. Hord, and A. Schepps. This work was funded in part by the North Carolina Agricultural Research Service and in part by the North Carolina Certified Sweetpotato Seed Growers Association and the North Carolina Sweetpotato Commission.

LITERATURE CITED

1. Blaker, N. S., and Hewitt, J. D. 1987. A comparison of resistance to *Phytophthora parasitica* in tomato. Phytopathology 77:1113-1116.
2. Carlson, L. W., and Struble, F. B. 1960. Methods for determining the reaction of sweetpotato lines to soil rot. Phytopathology 50:822-826.
3. Clark, C. A., and Lawrence, A. 1981. Morphology of spore-bearing structures in *Streptomyces ipomoeae*. Can. J. Microbiol. 27:575-579.
4. Clark, C. A., and Moyer, J. W. 1988. Compendium of Sweet Potato Diseases. American Phytopathological Society, St. Paul, MN.
5. Clark, C. A., Watson, B., and Hammett, H. L. 1981. Control of sweet potato soil rot with varietal resistance and chloropicrin. Fungic. Nematic. Tests 36:77.
6. Hernandez, Teme P., Constantin, R. J., Hammett, H., Martin, W. J., Clark, C. A., and Rolston, L. 1981. 'Travis' sweet potato. HortScience 16:574.
7. Lorbeer, J. W. 1960. The biology and control of *Streptomyces ipomoea* on sweet potato. Ph.D. thesis. University of California, Berkeley.
8. Martin, W. J. 1958. Reaction of sweet potato varieties and seedlings to soil rot. Phytopathology 48:445-448.
9. Martin, W. J. 1969. Control of soil rot of sweetpotatoes by preplant, in-row application of fungicidal fumigants. (Abstr.) Phytopathology 59:1040.
10. Martin, W. J., Hernandez, Travis P., and Hernandez, Teme P. 1975. Development and disease reaction of Jasper, a new soil rot-resistant sweet potato variety from Louisiana. Plant Dis. Rep. 59:388-391.
11. Martin, W. J., Jones, L. G., and Hernandez, Travis P. 1967. Sweet potato soil rot development in Olivier silt loam soil as affected by annual applications of lime or sulfur over a seven-year period. Plant Dis. Rep. 51:271-275.
12. McLean, E. O. 1982. Soil pH and lime requirement. Pages 199-224 in: Methods of Soil Analysis, Part 2: Chemical and Microbial Properties. A. L. Page, R. H. Miller, and D. R. Keeney, eds. American Society of Agronomy, Madison, WI.
13. Moyer, J. W., Campbell, C. L., Echandi, E., and Collins, W. W. 1984. Improved methodology for evaluating resistance in sweet potato to *Streptomyces ipomoea*. Phytopathology 74:494-497.
14. Person, L. H., and Martin, W. J. 1940. Soil rot of sweet potatoes in Louisiana. Phytopathology 30:913-926.
15. Ristaino, J. B., and Averre, C. W. 1992. Effects of irrigation, sulfur, and fumigation on Streptomyces soil rot and yield components in sweetpotato. Phytopathology 82:670-676.
16. Rolston, L. H., Clark, C. A., Cannon, J. M., Randle, W. M., Riley, E. G., Wilson, P. W., and Robbins, M. L. 1987. 'Beauregard' sweet potato. HortScience 22:1338-1339.
17. Sleesman, J. P., and Leben, C. 1978. Preserving phytopathogenic bacteria at −70 C or with silica gel. Plant Dis. Rep. 62:910-913.
18. Wilson, L. G., and Averre, C. W. 1989. Growing and marketing quality sweetpotatoes. N.C. Agric. Ext. Serv. Pub. AG-9, Raleigh, NC.