Pathogenicity of *Fusarium* spp. from Diseased Sugar Beets and Variation Among Sugar Beet Isolates of *F. oxysporum*

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ABSTRACT

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Sugar beets (*Beta vulgaris*) with symptoms of Fusarium yellows were collected from fields in California, Colorado, Montana, Oregon, Texas, Wyoming, and Manitoba, Canada, during 1982–1988. Forty-eight isolates of seven species of *Fusarium* and two nonsporulating, unidentifiable "Roseum" types were obtained from root tissue on a medium selective for *Fusarium* spp. Three isolates of *F. oxysporum* (the reported causal agent of Fusarium yellows) from California, one each from Colorado and Texas, and two each from Montana, Oregon, and Canada, a Colorado isolate of *F. acuminatum,* two isolates of *F. avenaceum* from Texas, and one "Roseum" type from Colorado all caused foliar yellowing, wilt, root necrosis, and in some cases eventual death of sugar beet seedlings in greenhouse tests. Two isolates of *F. solani* (from California and Colorado) induced mild to moderate root rot of seedling taproots and secondary roots but no typical yellows symptoms. Of the isolates that were pathogenic to seedlings, only isolates of *F. oxysporum* and the Colorado isolate of *F. acuminatum* induced typical Fusarium yellows symptoms in wound-inoculated 3-mo-old sugar beets in the greenhouse. Other isolates pathogenic on seedlings induced some necrosis of secondary roots and arrested, necrotic lesions on the taproot of older sugar beets but no wilting, foliar yellowing, or plant death. All virulent and avirulent isolates were reisolated from surface-disinfested roots 30 days after planting in infested soil or 2 mo after inoculation of taproots. Isolates of *F. oxysporum* varied in growth, pigmentation, and conidial production on potato-dextrose agar. A significant isolate × cultivar disease interaction occurred when two sugar beet breeding lines, one susceptible and one resistant, were tested with four isolates of *F. oxysporum* from diverse geographic areas. In separate analyses of data from the resistant and susceptible breeding lines, the four isolates varied in virulence toward the susceptible but not the resistant line. Thus, the existence of physiological specialization among isolates of *F. oxysporum* from sugar beet remains an unresolved question.

Additional keywords: *Fusarium equiseti, F. proliferatum, F. sambucinum*

Fusarium yellows of sugar beet (*Beta vulgaris* L.) was described first by Stewart (28) in 1931 from southeastern Colorado. Symptoms were slight to moderate wilting of the foliage, interveinal chlorosis, and vascular discoloration of the taproot; no external necroses of the taproot or secondary roots were reported. Although macroconidia were not observed, the causal organism was identified as *Fusarium conglutinans* var. *betae* and later reclassified as *F. oxysporum* f. sp. *betae* (27). Several other species of *Fusarium* are associated with various rots of sugar beet seedlings and taproots (8,11,16,18,20), but these diseases are not as widespread or as economically important as Fusarium yellows.

Fusarium yellows was limited in occurrence and importance in the United States until the 1970s, when the causal organism induced a serious stalk blight in sugar beet seed crops in Oregon (7). Agriculturists of beet sugar companies in Colorado, Montana, Texas, and Wyoming also noted increased incidence of the vascular wilt phase of the disease in the root crop (*personal communications*). The severity of stalk blight in the seed crop prompted the U.S. Department of Agriculture to initiate a breeding program in California to select for resistance to the fungus (19). Breeders at the Great Western Sugar Company (now defunct) in Colorado began a similar program.

Because many *Fusarium* spp. exist as various physiological races or biotypes, the question arose whether the two breeding programs indeed were directed toward developing resistance to the same pathogen. Sugar beet isolates of *F. oxysporum* from different geographic areas are quite variable in culture (E. G. Ruppel, *unpublished*), and some sugar beet lines with field resistance to stalk blight in Oregon were susceptible to Fusarium yellows when planted in Colorado (13).

The research reported herein was undertaken to study variability and pathogenicity of sugar beet isolates of *F. oxysporum* from diverse sugar beet-growing regions of the United States and Canada. Other *Fusarium* spp. isolated from diseased sugar beets during this study also were tested for pathogenicity on sugar beet.

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MATERIALS AND METHODS

Isolations. Sugar beets with symptoms of Fusarium yellows (2,24,28) were collected from fields in California, Colorado, Montana, Oregon, Texas, and Wyoming and from Manitoba, Canada, between 1982 and 1988. None of the plants had external disease symptoms on the roots. Tops were removed from the plants, and roots were washed to remove adhering soil. A vertical slit was made in the taproot crown, and the taproot was forcibly split lengthwise. Tissue from necrotic vascular strands was transferred to Komada's medium (KM) (12), which is selective for *F. oxysporum* and other *Fusarium* spp. Single-spore transfers were made from developing colonies, and individual microconidia or macroconidia (or hyphal tips for two nonsporulating isolates) were transferred to both carnation leaf agar (CLA) and freshly made potato-dextrose agar (PDA) for species identification following Nelson et al (21). Single-spore and hyphal tip isolates were maintained in soil in a refrigerator at 4 C (4).

Pathogenicity tests. To prepare inocula, oatmeal (noninstant) was triturated with a mortar and pestle then mixed with sieved, fine sand to a final oatmeal concentration of 1%. The mixture was placed in 125-ml Erlenmeyer flasks (50 cm^3 per flask), sufficient distilled water was added to wet the sand-oatmeal mixture, and the flasks were autoclaved for 2 hr on each of two consecutive days. A 4-mm-diam mycelial agar disk from a 4-day-old PDA culture of each isolate grown from the stock soil culture was transferred to a flask of sand-oatmeal medium. After 2 wk, inocula were removed from the flasks and assayed on KM to determine the number of colony-forming units (cfu) per gram.

For seedling pathogenicity tests, sand-oatmeal inocula were added to pasteurized greenhouse soil at the rate of 2 mg of inoculum per gram of soil; in an additional treatment, autoclaved inocula were added at the same rate to uninfested soil. This rate of inoculum provided initial population densities of between 2×10^4 and 4×10^5 cfu per gram of soil, depending on the isolate of

Fusarium tested. Because the objective in these initial tests was to determine pathogenicity of the isolates and not to compare their virulence or aggressiveness, inoculum concentrations were not standardized. However, in a subsequent experiment to compare isolates of *F. oxysporum* on seedlings of two sugar beet breeding lines, initial soil population densities of the isolates were adjusted to 10^5 cfu per gram of soil, and comparable amounts of sterilized sand-oatmeal mix were added to uninfested soil as an additional treatment. Soil and inoculum were mixed in a twin-shell blender for 30 sec, then immediately potted in 10-cm-diam clay pots. Sand-oatmeal inocula also were used for pathogenicity tests on older sugar beet plants.

For seedling pathogenicity tests, seeds of the susceptible sugar beet cultivar MonoHy D2 were surface-disinfested in 1% NaOCl for 10 min, rinsed three times with sterile distilled water, and planted in pans of autoclaved sand. Pans were placed on a greenhouse bench and irrigated immediately with water and thereafter with Hoaglund's solution as needed.

When seedlings were 10 days old, they were removed from the sand and rinsed in running tap water, and half of the lower taproot was excised. One seedling was transplanted to each pot of infested soil, and pots were arranged on the greenhouse bench in a randomized complete block design with three replicates of each isolate. Ample space was left between pots to prevent cross-contamination during irrigations. The greenhouse temperature was maintained at 27 ± 4 C during the day and 22 ± 4 C at night, and supplemental fluorescent light was provided at night.

Plants were observed daily for disease symptoms until testing was terminated 30 days after transplanting. As seedlings developed incipient wilt or died, reisolations were made on selective medium as described below. Because of the large number of isolates and limitations on space and time, eight isolates were tested at a time. In each test, however, a known pathogenic isolate of *F. o.* f. sp. *betae* (isolate SSB-4 [ATCC 34296], provided by J. D. MacDonald, University of California, Davis) was included as an additional treatment.

Isolates that induced wilt or death of seedlings were tested on older plants of the cultivar MonoHy A1 (susceptible to Fusarium yellows) grown individually in pasteurized greenhouse soil in 15-cm-diam clay pots. When plants were 3 mo old, a sterile spatula was used to wound secondary roots around each taproot and to open a space between the taproot and soil on opposite sides of each root. Sand-oatmeal inoculum (approximately 0.5 g) was placed in each opening and covered with soil. Controls were handled similarly except that they were treated with autoclaved inocula. Plants were observed daily for symptoms until the tests were terminated 60 days after inoculation. A randomized complete block design with three replicates was used in each of two trials.

When pathogenicity tests were terminated, surviving plants were harvested, tops were removed, and roots were washed in running tap water and blotted dry with paper towels. Hypocotyl taproot sections about 3 cm long were excised, immersed for 3 sec in 70% ethanol, surface-disinfested in 1% NaOCl for 10 min, and placed on KM in 9-cm-diam petri dishes. Taproot sections of older sugar beets were aseptically split longitudinally before they were placed on the medium. Stock cultures of each test isolate also were transferred to separate dishes of KM for comparative purposes.

Cultural comparison of isolates of *F. oxysporum*. A sterile cork borer 4 mm in diameter was used to transfer 10 isolates of *F. oxysporum* to 9-cm-diam petri dishes, each containing 20 ml of freshly made PDA. Dishes were arranged in a completely randomized design with three replicates and incubated under fluorescent light as described by Nelson et al (21). Colony diameter at the widest point was measured after 4 days, and pigmentation of aerial hyphae and of the colony undersurface was recorded after 10 days' growth. Cultures then were flooded with 10 ml of sterile distilled water and brushed gently to free conidia, and concentrations of microconidia and macroconidia were determined with a hemacytometer. Eight hemacytometer counts were made of each replicate and averaged. This test was repeated once. Analysis of variance (ANOVA) was performed separately on all data from both trials; because *F*-ratio tests indicated that error variances were homogeneous, sums of squares were pooled for overall ANOVA. Duncan's multiple range test was used for mean separations.

Isolate × cultivar interaction. Seedlings of two sugar beet breeding lines were tested for their response to four pathogenic isolates of *F. oxysporum.* Line 75MSH194 is a resistant selection from susceptible line 75MSH3; both lines were provided by Akio Suzuki of the Great Western Sugar Co. (now defunct) Agricultural Research Center, Longmont, CO.

The four sugar beet isolates of *F. oxysporum* were WP and FO from California, GWF-2 from Montana, and SSB-4 from Oregon. Experimental procedures were identical to those used for the seedling pathogenicity tests, except inocula were standardized as previously described and seedlings were harvested 3 mo after they were transplanted to infested soil. An additional treatment consisted of seedlings grown in uninfested soil to which autoclaved inocula were added.

At harvest, plants were rated on a scale from 0 to 5, where 0 = clean, healthy roots; 1 = some foliar chlorosis and slight discoloration of feeder roots; 2 = mild wilting and discoloration of taproot and feeder roots; 3 = severe wilting, with taproot discoloration and rotting of feeder roots; 4 = incipient wilt, with rot of taproot and feeder roots; and 5 = plant dead. A 2 × 4 factorial experiment was arranged in a randomized complete block design with three replicates in each of two trials. ANOVA was performed on data from each trial. Because error variances were homogeneous between the two trials (*F*-ratio test), an overall ANOVA was performed on pooled sums of squares from both trials.

RESULTS

Isolations. A total of 48 isolates of *Fusarium* were obtained from sugar beets. These included three isolates each of *F. acuminatum* Ellis & Everh., *F. avenaceum* (Fr.:Fr.) Sacc., and *F. sambucinum* Fuckel, one each of *F. equiseti* (Corda) Sacc. and *F. proliferatum* (T. Matsushima) Nirenberg, eight of *F. solani* (Mart.) Sacc., two nonsporulating "Roseum" types that produced chlamydospores in chains and clumps and had carmine red to dark brown colony undersurfaces, and 27 isolates of *F. oxysporum.*

Pathogenicity tests. Eleven isolates of *F. oxysporum,* including three from California, one each from Colorado and Texas, and two each from Montana, Oregon, and Canada, a Colorado isolate of *F. acuminatum,* two Texas isolates of *F. avenaceum* (one isolated in 1982 and the other in 1985), and one Colorado "Roseum" type induced varied degrees of wilting, leaf chlorosis, necroses of taproots and secondary roots, and vascular discoloration of seedling sugar beets in both trials. Differing inoculum densities precluded comparisons of virulence and aggressiveness of the test isolates; however, disease severity did not seem to be related to initial inoculum density (*data not shown*), and all isolates induced similar symptoms. All pathogenic isolates rapidly killed some, but not all, seedlings before wilted seedlings were harvested for reisolations. Two isolates of *F. solani* induced some mild to moderate necrosis of seedling taproots and secondary roots but no wilting or foliar chlorosis. Seedlings in the uninfested treatments remained symptomless.

When the isolates that induced symptoms in seedlings were tested on 3-mo-old sugar beets, only the Colorado isolate of *F. acuminatum* and the 11 isolates of *F. oxysporum* induced symptoms typical of Fusarium yellows, namely varied degrees of interveinal foliar chlorosis, wilting, necrosis of secondary roots, and some vascular discoloration of the

taproot. None of the plants died during the 60-day tests, and disease severity was similar for all isolates. Taproots were symptomless; no tip rot (17) was seen on taproots. All uninoculated plants remained symptomless.

Reisolations and cultural characteristics. All isolates, whether pathogenic or not, were reisolated from seedlings and older sugar beets. Distinct cultural appearances and comparisons with stock culture isolates grown in separate dishes of selective medium permitted easy identification of types or species used for soil infestation or inoculation. No *Fusarium* spp. were isolated from seedlings grown in uninfested soil or from older uninoculated plants.

Cultural characteristics on PDA of all species (except some isolates of *F. oxysporum*) and the "Roseum" types were as described by Nelson et al (21). Although microconidia are reported to be scarce or absent in most isolates of *F. acuminatum, F. avenaceum,* and *F. sambucinum,* all sugar beet isolates of these species produced some, albeit few, one- to three-celled microconidia on CLA.

Isolates of *F. oxysporum* from different geographic areas showed significant ($P = 0.05$) variability in colony growth and production of conidia (*data not shown*). Known pathogenic isolate SSB-4, one pathogenic isolate from Montana, and two Canadian isolates developed pale, purple-tinged aerial growth and dark purplish undersurfaces on PDA. Most pathogenic isolates from California, Colorado, and Texas produced very pale, salmon-tinged or pinkish white aerial growth and pale salmon to pale yellow undersurfaces, whereas one isolate produced almost white aerial growth with a pale yellowish undersurface. Microconidia, macroconidia, and chlamydospores produced by all isolates of *F. oxysporum* generally were typical for the species and were morphologically indistinguishable (21); however, the number of conidia produced in culture differed among isolates ($P = 0.05$). One Colorado isolate resembled Stewart's (28) original description except that a few macroconidia were observed on CLA and chlamydospores were single or double rather than in long chains.

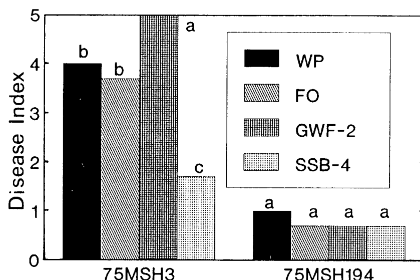

Fig. 1. Disease indexes of sugar beet seedlings resistant (cultivar 75MS194) or susceptible (cultivar 75MSH3) to *Fusarium oxysporum* 30 days after transplanting to soil infested with isolate WP or FO (from California), GWF-2 (from Montana), or SSB-4 (from stalk-blighted seed beets in Oregon). The disease index was based on a scale of 0–5, with 0 = healthy and 5 = dead. The initial population density of each isolate was 10^5 cfu per gram of pasteurized soil. Data are the combined means of two trials, each with three replicates. Within cultivars, bars labeled with the same letter do not differ significantly ($P = 0.05$) according to Duncan's multiple range test.

Isolate × line interaction. Means of the combined isolate × line tests are shown graphically in Figure 1. The susceptible parent line 75MSH3 was more severely affected by all isolates than was the resistant selection 75MSH194; however, a significant ($P = 0.05$) isolate × breeding line interaction precluded conclusions about main-effect treatments. A combined ANOVA on pooled data from the susceptible cultivar across trials indicated a significant ($P = 0.05$) difference among isolates. According to Duncan's multiple range test, isolate GWF-2 induced more severe disease than isolates WP, FO, and SSB-4. Isolates WP and FO did not differ significantly, but both induced more severe disease than isolate SSB-4. Isolates did not differ significantly in virulence in a combined ANOVA of data from the resistant breeding line. No *Fusarium* spp. were isolated from plants grown in uninfested soil.

DISCUSSION

F. avenaceum induces wilt of sugar beet seedlings in India (20), and Kocková-Kratochvilová et al (11) associated *F. culmorum, F. sambucinum, F. solani,* and *F. coeruleum* (syn. *F. solani* var. *coeruleum* [3]) with "heart rot" of older beets. *F. solani* (formerly *F. radicicola*) causes "tip rot" in the United States and Yugoslavia (16,18), and *F. culmorum* induces a crown rot of sugar beet under drought conditions in England (8). My isolates of *F. equiseti, F. proliferatum,* and *F. sambucinum* failed to induce disease in seedlings. To my knowledge, *F. acuminatum* has not been reported previously as a pathogen of sugar beet, although it has been reported to cause foot or root rot of many plants (3,5). Fusarium yellows, caused by *F. oxysporum,* has not been reported on sugar beet in California. However, Martyn et al (17) reported typical symptoms of the disease in one greenhouse test using two California isolates of the fungus supplied by our laboratory.

Two isolates were identified arbitrarily as "Roseum" types based on in vitro growth characteristics and the formation of smooth-walled chlamydospores in clumps and chains typical of this section of *Fusarium;* however, definite identification of these isolates as species of *Fusarium* is not possible without production of conidia. Cultural appearance of these isolates was similar to that of *Epicoccum* spp. (25), but chlamydospores definitely differed from the multicelled conidia of *Epicoccum.*

Why the isolates of *F. avenaceum* that induced disease in seedlings failed to cause symptoms in older sugar beets is not known. Older plants may have a type of mature-plant resistance to this species, inoculum concentrations may have been too low, or cultural conditions may have been unfavorable for infection by what may be a less virulent or aggressive sugar beet pathogen. Additional tests with *F. avenaceum* are warranted.

Although seedlings and older sugar beets differed in their response to *F. avenaceum,* the seedling assay was effective in identifying pathogenic isolates of *F. oxysporum* that induced symptoms of Fusarium yellows in older plants. The seedling assay also discriminated between sugar beet cultivars resistant and susceptible to *F. oxysporum* and thus may be suitable for preliminary screening of breeding lines for resistance to the pathogen. In addition, the assay provided a measure of isolate virulence in the susceptible cultivar when inoculum concentrations were standardized.

All pathogenic and nonpathogenic isolates of *Fusarium* tested in this study were recovered from surface-disinfested roots of inoculated sugar beet. Colonization of living plant roots by nonpathogenic *Fusarium* spp. and other nonpathogenic soilborne fungi has been reported (6,22,26). As suggested by Gordon et al (6), such colonization of nonsusceptible crops may be important in inoculum persistence and the epidemiology of diseases of other crops.

F. oxysporum was the species most frequently isolated from sugar beets with symptoms of Fusarium yellows. Only one Colorado isolate, however, matched the original description of the pathogen by Stewart (28). According to his description, sugar beet isolates produced terminal or intercalary chlamydospores, the latter in long chains. Stewart was unable to induce macroconidial production on several media. Similarly, Texas isolates used by Martyn et al (17) rarely produced macroconidia but did form microconidia and chlamydospores typical of *F. oxysporum.* Most of my pathogenic isolates produced abundant macroconidia on CLA and PDA and single or paired chlamydospores on PDA that were typical of the species (21). The differences between my isolates and those of Stewart (28) or the Texas isolates of Martyn et al (17) might be the result of variations in cultural conditions employed in our laboratories.

Although *F. oxysporum* was the most prevalent species isolated from beets with symptoms of Fusarium yellows, induction of the same symptoms by an isolate of *F. acuminatum* may be important to

sugar beet breeders. It is possible that resistance developed for one species will be ineffective against another species. Thus, when breeding nurseries are established in fields naturally infested with *Fusarium,* the species of the resident pathogen should be determined.

The question of the existence of races of *F. o.* f. sp. *betae* remains unresolved. Armstrong and Armstrong (1) proposed that *F. o.* f. sp. *betae* should be a separate race of *F. o.* f. sp. *spinaciae,* based on pathogenicity to common hosts and priority of the latter forma epithet. Martyn et al (17) suggested that differences in isozymes and induced symptoms between Texas isolates and those from other areas may warrant the designation of a new forma specialis for Texas isolates.

In the present study, variation among sugar beet isolates of *F. oxysporum* in growth in vitro, sporulation, and cultural pigmentation, and a significant isolate × cultivar interaction also may indicate that the pathogen exists as multiple strains or races. However, cultural variability is not a valid criterion for the designation of races or formae, and the observed interaction was primarily a change in rate rather than a complete reversal; four isolates induced the same degree of disease severity in a resistant breeding line.

To clarify the existence of physiological specialization among pathogenic isolates of *F. oxysporum* from sugar beet, sugar beet cultivars with varied degrees of resistance (15) need to be challenged with many diverse isolates of the pathogen, and isozyme patterns of a large sample of isolates should be compared for additional enzymes (17). Finally, cross-inoculation tests of various plants with several isolates of *F. o.* f. sp. *spinaciae* and *F. o.* f. sp. *betae* (1,14), coupled with isozyme analyses of the isolates (17), vegetative compatibility tests (9,23), or enzyme-linked immunosorbent assays described for *Fusarium* spp. (10), should be conducted to determine whether races or new formae speciales are justified.

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