Occurrence of Fusarium Yellows of Celery Caused by *Fusarium oxysporum* f. sp. *apii* Race 2 in New York and Its Control

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ABSTRACT

Awuah, R. T., Lorbeer, J. W., and Ellerbrock, L. A. 1986. Occurrence of Fusarium yellows of celery caused by *Fusarium oxysporum* f. sp. *apii* race 2 in New York and its control. Plant Disease 70: 1154-1158.

An important celery disease in New York was proven to be Fusarium yellows, caused by *Fusarium oxysporum* f. sp. *apii* (*F. o.* f. sp. *apii*) race 2. The pathogen was isolated from roots, crowns, and petioles of diseased celery plants collected from commercial fields, and typical symptoms of the disease were reproduced in greenhouse pathogenicity studies. Production of celery seedlings in seedbeds of organic soil infested with *F. o.* f. sp. *apii* race 2 and transplantation to commercial celery fields was the factor most responsible for the rapid spread of the fungus in commercial fields in New York. Fumigation of celery seedbed soil with methyl bromide eliminated this source of the pathogen. Several celeriac plant introductions (169001, 169004, and 171500) were highly resistant to the disease in greenhouse and field experiments. Attempts to control the disease by fungicides were ineffective.

A new disease of celery (*Apium graveolens* L. var. *dulce* DC.) in Orange County, New York, has resulted in the rapid decline in celery production in a number of commercial fields since 1980. The disease causes the foliage of infected plants to become chlorotic and later blanched and brittle. Such leaves normally curl inward. At this stage, plants are stunted and vascular tissue becomes discolored. The brick-red to brown discoloration of internal tissues generally starts from the remnant of the taproot (if present), extends into the crown, and usually progresses into the petiole. The root systems of diseased plants are reduced as a result of extensive decay. Severely affected plants normally die before harvest. Most infected plants usually are affected less extensively and generally survive. Most of these, however, are not harvested either because they are stunted or their petioles taste slightly bitter. When pulled from the soil, severely stunted plants usually break off at the crown and soft rot is apparent.

This paper reports investigations carried out to determine the cause of the disease and to identify the factors that resulted in its rapid buildup. Measures studied to control the disease are detailed and discussed.

Accepted for publication 19 June 1986.

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MATERIALS AND METHODS

Isolation of the pathogen. Diseased celery plants were collected from several fields on the Bierstine farm in Orange County during the summers of 1980 and 1981. From 1982 to 1985, diseased plants were obtained from the Bierstine farm as well as an additional farm (Gratz and Utter). Isolations were made from the roots, crowns, and petioles by placing small sections of diseased tissue on either acidified potato-dextrose agar (APDA, Difco) or chloramphenicol-amended potato-dextrose agar (CPDA) in petri plates. The plates were incubated at 24–25 C under fluorescent light with a 12-hr photoperiod for 5–7 days. Monoconidial isolates were obtained from the predominantly *Fusarium* colonies growing out of the plated tissues and maintained on CPDA slants. Identification of *Fusarium* isolates to species was made by morphological distinctions (3,8). *Fusarium oxysporum* f. sp. *apii* (*F. o.* f. sp. *apii*) race 2 was identified by pathogenicity tests.

Pathogenicity tests. Inoculum for pathogenicity studies was produced on oat kernel/potato-dextrose broth medium. Coarsely ground oat seeds were placed in petri dishes 7.5 cm (2.9 in.) deep and 9.5 cm (3.7 in.) across at 30 g/dish. Forty milliliters of 1.2% potato-dextrose broth (PDB, Difco) was added to each dish and autoclaved for 20 min at 0.98 kg/cm^2 (14 psi) and 121 C. Ten- to 14-day-old CPDA slants of each isolate tested were flooded with 10 ml of sterile water. A conidial-mycelial suspension loosened with a sterile loop from each culture was used to seed each dish of the oat kernel-PDB medium, which then was incubated at 25 C with a 12-hr photoperiod. The dish was shaken frequently during incubation to prevent caking. After 1 wk, the contents of the dish were mixed thoroughly with 250 g of steam-sterilized Cornell University Plant Pathology Department greenhouse mix (16.6% sand, 41.7% compost, and 41.7% peat moss) in a large plastic bag. This inoculum-soil mixture was transferred to a plastic tray (20 × 13 × 10 cm, 7.8 × 5.1 × 3.9 in.) and incubated for 1 wk, during which it was stirred to maintain a loosened state.

Three-week-old seedlings of the celery cultivar Florida 683 were transplanted into the infested soil at a rate of 12 per tray (17 trays, one tray for each isolate). A control tray of the Cornell soil mix with uninfested, autoclaved oat-PDB medium also was included. Naturally infested organic soil and naturally infested organic soil autoclaved at 121 C for 3 hr at 0.98 kg/cm^2 (14 psi) were used in these pathogenicity studies. Two pathogenic isolates of *F. o.* f. sp. *apii* race 2 from California (H171 and 40) also were included in the study. Isolates H171 and 40 originated from Ventura and Orange counties (California), respectively, and were supplied by John Puhalla.

Temperatures during an experiment ranged between 25–30 C and the photoperiod was 14–16 hr. Plants were watered with tap water when necessary. They were grown for 5 wk in a greenhouse at Ithaca, removed from the soil, and rated for disease severity.

Disease rating scheme. The following system, using a disease rating scale of 0–5, was used to determine a Fusarium yellows index: 0 = clean roots, healthy top, no disease; 1 = slight vascular discoloration limited primarily to secondary and tertiary roots, healthy top; 2 = pronounced vascular discoloration of secondary and tertiary roots, healthy top; 3 = general chlorosis, at times slight blanching of leaves but without leaf curling and brittleness, severe discoloration of secondary and tertiary roots but no vascular discoloration of the primary root (or remnant), crown, and petiole; 4 = blanching, brittleness and curling of leaves, stunted growth, vascular discoloration of the primary root (or remnant) and crown, discoloration occasionally extending into the petiole; and 5 = dead plant.

Rapid spread of the pathogen. To understand the rapid spread of the pathogen, cultural practices used at the Bierstine farm as well as the Gratz and Utter farm, especially methods for growing seedlings for transplanting, were studied. The root systems and internal tissues of transplants were examined for symptoms, and isolations were attempted from diseased tissues. Experiments using different sources of seedbed medium for growing seedlings were then conducted.

Seeds of the celery cultivar Florida 683 were planted in different soil media in 30-cm plastic pots in the greenhouse at Ithaca. The greenhouse temperatures averaged 25 C and the photoperiod was 14 hr. Five soil media were used: 1) autoclaved organic soil (obtained from a celery field heavily infested with *F. o.* f. sp. *apii* race 2 at the Bierstine farm and autoclaved twice for 3-hr periods), 2) Bierstine seedbed soil (organic soil obtained from one of the Bierstine farm greenhouses), 3) organic soil obtained from the Bierstine farm celery field heavily infested with the pathogen, 4) organic soil not previously planted to celery (obtained from a field in Orange County planted to turfgrass), and 5) Cornell greenhouse mix steam-sterilized for 3 hr.

Plants were grown in the various soil media for 4.5 wk. Twenty plants from each medium then were removed and their root systems examined and rated for disease severity. The remaining plants from each medium were transplanted to a field plot. Six-week-old transplants raised in one of the Bierstine farm greenhouses also were included in the study. A completely randomized design was used. Each of the treatments (four replicates) involved setting the plants in 12.2-m-long (40-ft) rows at a spacing of 21.6 cm (8.5 in.) between plants and 0.5 m (1.5 ft) between rows. The experiment was repeated at another location in the same field. Normal commercial cultural practices were followed for growing the plants. Seven weeks after transplanting, 20 plants from the middle of each row were rated for incidence and severity of Fusarium yellows.

Cultivar reaction. Trials to determine resistant and susceptible reactions to the pathogen among celery and celeriac cultivars and plant introductions were conducted both in a greenhouse at Ithaca and in a commercial celery field in Orange County. Artificially infested greenhouse soil and naturally infested organic soil from the Bierstine farm were used in the greenhouse studies. The New York isolate P-3 of *F. o.* f. sp. *apii* race 2 was used to artificially infest the greenhouse soil.

Inoculum for infesting the soil was prepared in the following manner. Three-hundred-gram lots of wheat kernels were soaked overnight in 300-ml distilled water in 1-L Erlenmeyer flasks. The medium was autoclaved twice for 25 min. Each flask was seeded with five plugs from 3-wk-old cultures of isolate P-3 of the pathogen grown on potato-dextrose agar (PDA) at 24–25 C with 12-hr photoperiods. During the incubation period, flasks were shaken periodically to prevent the kernels from sticking together. The soil used was Cornell greenhouse mix. Twenty-four kilograms of the greenhouse mix was autoclaved for 9 hr and mixed thoroughly with 7.8 kg of inoculum in a cement mixer for 1 hr. The infested soil was distributed equally into 117 plastic pots (12.7 cm in diameter) and incubated in the greenhouse at 24–26 C for 12 days. During this period, the potted soil was maintained in a loose state by periodic stirring.

Three-week-old seedlings of 39 cultivars and plant introductions were transplanted (six per pot) into the naturally infested soil. Eighteen plants of each cultivar and plant introduction were tested (three replicates per cultivar or plant introduction). After 8 wk, the plants were uprooted and rated for disease severity.

The greenhouse study with the naturally infested field soil was divided into two parts. In the first, organic soil naturally infested with the pathogen from the Bierstine farm was used for growing the same celery and celeriac selections tested in the artificially infested field soil. In the second, 11 of the entries from the first greenhouse study using the naturally infested soil were subjected to a further test but with a modified source of inoculum in that the naturally infested soil used had been planted once to celery in the greenhouse to increase the inoculum level. Five of the 11 selections, Large Smooth Prague, Alabaster, Giant Prague, 352 Marble Ball, and PI 171500 (all celeriac), had been rated moderately resistant in the initial greenhouse trial. Three other entries, Tendercrisp, Surepak, and Burpee's Fordhook (all celery), were moderately susceptible in the same trial. PI 169001 (celeriac), which was highly resistant in the initial trial, also was included.

The 18 cultivars and plant introductions used in the field experiment had demonstrated different degrees of resistance in the greenhouse studies. The field used for the trial consisted of a soil heavily infested with the pathogen at the Bierstine farm. Previous observations of the disease in the field suggested a relatively uniform distribution of the pathogen. Six-week-old transplants of each entry were produced in a greenhouse at the Bierstine farm and transplanted to the field. A completely randomized design experiment was used. Each cultivar or plant introduction was planted in 4.6-m-long (15-ft) rows at a spacing of 21.6 cm between plants and 0.5 m between rows. Treatments were replicated four times. The experiment was repeated twice at two other locations in the same field. Regular cultural practices were followed. Twelve weeks after transplanting, the plants were rated for disease severity.

Chemical control. Fourteen fungicides applied as drenches were tested for control of the disease in a completely randomized design field trial. In one subset of the experiment (replicated three times), the 14 fungicides were applied as drenches to the roots of celery plants at the time of transplanting. Four weeks later, half of these treated plants received additional side-drenches of the same fungicide. In another subset of the experiment, previously untreated plants were side-drenched with each of the 14 fungicides (three replicates for each treatment) 4 wk after transplanting. Appropriate water checks were included in all experiments. Fungicides were applied by drench equipment, which delivered 0.95 L (1 qt) of fungicide mixture per 6.1 m (20 ft) of row length. Seven-week-old transplants of the celery cultivar Florida 683 were transplanted to each one-row plot at a spacing of 21.6 cm between plants and 0.5 m between rows. The plants were grown following conventional commercial cultural practices. Nine weeks after transplanting, 15 plants in the middle of each treatment row were rated for disease severity.

Each fungicide used was also applied to 18.6-m^2 (200-ft^2) plots with a CO_2-powered hand sprayer at a spray pressure of 1.1 kg/cm^2 (15.6 lb/$in.^2$) with four replicates per treatment. The soil then was thoroughly harrowed and the plots drenched with water by overhead irrigation for 30 min. The herbicide trifluralin (Treflan) also was used because of its reported effectiveness in controlling root rot of pea. A water check was included. Seven-week-old plants of the celery cultivar Florida 683 were transplanted to 6.1-m-long rows (four rows per plot) at a spacing of 21.6 cm between plants and 0.5 m between rows. After 7 wk, 30 randomly selected plants from the two innermost rows of each plot were rated for disease severity.

RESULTS

Isolation of the pathogen. *F. o.* f. sp. *apii* race 2 was isolated frequently from roots, crowns, and petioles of celery plants showing symptoms of the disease. On CPDA, all isolates were strongly mycelial and pale salmon in color according to *Color Standards and Color Nomenclature*, published by R. Ridgeway in 1912. Two-week-old cultures of the fungus growing on CPDA under a 12-hr photoperiod at 24–25 C produced abundant microconidia ranging from 4.8 to 7.4 × 1.5 to 3.7 μm. Only a few macroconidia were produced and were mostly septate (four cells) ranging from 18.7 to 29.6 × 3.7 to 4.8 μm. Chlamydospores, though formed only occasionally, were globose and borne

singly or in pairs in an intercalary or terminal manner.

All isolates of *F. o.* f. sp. *apii* race 2 from Orange County and the two isolates from California were pathogenic in greenhouse studies (Table 1). Symptoms of Fusarium yellows (general chlorosis, leaf branching, brittleness and curling, stunting, vascular discoloration, and death of plants) were reproduced in greenhouse pathogenicity studies. As the disease progressed, leaf blanching, curling, and brittleness gradually became apparent. By the fifth week, severely infected plants showed vascular discoloration of the primary root (or its remnant) and crown, and the plants were stunted. At this stage, plants usually died. Six-week-old transplants from the Bierstine farm greenhouse often had extensive root decay, especially in the secondary and tertiary roots. *F. o.* f. sp. *apii* race 2 consistently was isolated from roots of these transplants. Internal discoloration (crown, petiole, and primary root), general chlorosis, leaf curling and brittleness, however, were not observed on these transplants.

Rapid spread of the pathogen. Transplants produced in Bierstine farm field soil naturally infested with *F. o.* f. sp. *apii* race 2 had the greatest root necrosis and the highest disease levels. Transplants produced in Cornell greenhouse mix were disease-free. In the autoclaved, naturally infested organic soil, all of the transplants rated for disease were free of Fusarium yellows symptoms and no damping-off was observed. Transplants produced in the natural organic soil not previously planted to celery also were free of Fusarium yellows symptoms. In the Bierstine farm seedbed soil, root necrosis caused by *F. o.* f. sp. *apii* race 2 was prevalent in transplants.

In the field study, transplants, regardless of the soil medium in which they were produced, were heavily infected by the pathogen and severely diseased. Differences in the rate and level of symptom development of Fusarium yellows among the treatments were not detected, and final disease indices for the various treatments did not differ significantly ($P = 0.05$).

Cultivar reaction. In both naturally infested organic field soil and greenhouse soil artificially infested with isolate P-3 of *F. o.* f. sp. *apii* race 2, celeriac cultivars and plant introductions showed a higher level of resistance than those of celery (Table 2). In the artificially infested soil, 14 of the celery cultivars and plant introductions were highly susceptible, five were moderately susceptible, and one was moderately resistant. In the artificially infested soil, 17 celeriac cultivars and plant introductions were moderately resistant, one was moderately susceptible, and one was highly resistant. In naturally infested field soil, a similar trend was observed. Sixteen of the celery cultivars and plant introductions were highly susceptible, four were moderately susceptible, and none was moderately resistant. Sixteen of the celeriac cultivars and plant introductions were moderately resistant, two were moderately susceptible, and one was highly resistant. In the second greenhouse experiment, using the naturally infested organic field soil cropped once to celery to increase the inoculum level, the higher level of resistance possessed by celeriac was confirmed (Table 3). Of the six celeriac cultivars evaluated, three were moderately susceptible and the other three showed moderately resistant reactions. All but one of the celery entries tested were highly susceptible.

In the field experiment, nine of the 10 celeriac entries were rated highly resistant and one was moderately resistant (Table 4). Only two of the eight celery entries, Burpee's Fordhook and Tendercrisp, were rated moderately susceptible. All the others showed a highly susceptible reaction. Some plants of the yellow celery cultivars showed typical symptoms of the disease (brittleness and cupping of leaves) as early as 3–4 wk after transplanting. The green cultivars, however, remained healthy until about the fifth week, when typical symptoms became apparent.

In all of the tests (greenhouse and field), the yellow cultivars of celery (Stokes Golden Plume, Golden Detroit, Golden Self-blanching, Burpee's Golden Self-blanching, and Cornell 619) and the green cultivar (Tall Green Light) were the most susceptible on the basis of their mean disease indices.

Chemical control. None of the 14 fungicides applied as a drench at planting or as a side-drench 4 wk after planting significantly reduced the incidence and severity of Fusarium yellows. The fungicides tested included benomyl (Benlate 50WP), chlorothalonil (Bravo 500 4.17F), mancozeb (Dithane M-45 80WP), thiabendazole (Mertect 42.28F), dicloran (Botran 75WP), propiconazole (CGA-64250 3.6EC), captan (Captan 50WP), vinclozolin (Ronilan 50WP), captafol (Difolatan 4F), triadimefon (Bayleton 50WP), fenaminsulf (Dexon 25WP), PCNB (Terrachlor 75WP), chloroneb (Demosan 65WP), and triadimenol (Baytan 150 1.25FS). Plants that received two drenches of a fungicide (drench at planting and side-drench 4 wk later) showed disease symptoms similar to those on plants that received a single application. Many of the treated plants had vascular discoloration in their crown tissue and a number were dead 9 wk after transplanting.

Incorporating fungicides and the herbicide trifluralin into the soil before transplanting also was ineffective in controlling the disease. Of the seven chemicals tested in this manner, none was able to reduce the severity of the disease. Phytotoxicity was not observed with any of the fungicides in either trial.

Table 1. Pathogenicity of representative New York isolates of *Fusarium oxysporum* f. sp. *apii* race 2 to celery (*Apium graveolens* var. *dulce*)[a]

Treatment	Fusarium yellows index[b]
P-13	3.17
P-3	3.50
R-1	3.17
40[c]	4.10
H171[c]	3.42
Check	0.00
Autoclaved field soil	0.00
Naturally infested field soil	3.93

[a] Soil infested with oat kernels colonized by *F. oxysporum* f. sp. *apii* race 2.

[b] On a scale of 0–5, where 0 = clean roots, healthy top, no disease; 1 = slight vascular discoloration limited primarily to secondary and tertiary roots, healthy top; 3 = general chlorosis, at times slight blanching of leaves but without leaf curling and brittleness, severe discoloration of secondary and tertiary roots but no vascular discoloration of the primary root (or remnant), crown, and petiole; 4 = blanching, brittleness and curling of leaves, stunted growth, vascular discoloration of the primary root (or remnant) and crown, discoloration occasionally extending into the petiole; and 5 = dead plant. Fusarium yellows index formula: (no. plants in class 0×0 + no. plants in class 1×1 + . . . no. plants in class 5×5)/total no. plants in each treatment.

[c] California isolates that originated from Ventura and Orange counties (supplied by John E. Puhalla).

DISCUSSION

The new disease of celery in Orange County, New York, is Fusarium yellows, caused by *F. o.* f. sp. *apii* race 2. Almost all field symptoms of the disease were reproduced consistently in the greenhouse by either growing celery seedlings in steam-sterilized greenhouse soil artificially infested with pure cultures of the pathogen isolated from Orange County celery plants or by growing celery seedlings in naturally infested field soil. Reisolations from infected tissue yielded a high percentage of *F. o.* f. sp. *apii* race 2. Soft rot in the crown region was not reproduced in the greenhouse, and this observation is consistent with the report of Hart and Endo (7). Inability to produce this symptom, which is caused by secondary bacteria, even with the naturally infested field soil, probably was due to the short experimental period of 5 wk. Under field conditions, soft rot of the crown normally is observed near harvesttime, which varies from 11 to 13 wk after planting. This is believed to be the first occurrence of Fusarium yellows in Orange County, although the disease caused by race 1 of the pathogen occurred in western New York in the

1920s (4).

All Orange County isolates of *F. o.* f. sp. *apii* obtained and tested for pathogenicity during the present study have been race 2. The Orange County isolates have caused disease symptoms (severity and type) identical to those caused by race 2 isolates from California on the green cultivar Florida 683. The Orange County isolate P-3 caused disease on both the green and yellow celery cultivars. When the Orange County isolate JC-1 and a known isolate of race 1 (A8 from France) were compared, the former caused severe disease on both green and yellow celery cultivars, whereas the race 1 isolate was pathogenic to only the yellow celery cultivar. Elmer's study in Michigan (6) with three Orange County isolates (JC-1, P-13, and NR-1) indicated that these were race 2, because they caused disease on both the green and yellow celery cultivars.

Whether *F. o.* f. sp. *apii* race 2 evolved in New York or was introduced from another celery-producing region is not known. Its introduction from another region, however, seems likely because Fusarium yellows of celery caused by race 1 of the pathogen has never been reported in Orange County, although the disease has been reported previously in western New York in Niagara County (4) and also Wayne County (A. G. Newhall, *personal communication*). Orange County celery growers have frequently used transplants from Florida to augment their own stock (1). That Fusarium yellows has been a problem in nurseries in Florida is well documented. In a comprehensive study of celery diseases in Florida, Cox (5) listed *F. o.* f. sp. *apii* as the fungus most commonly associated with Fusarium yellows in celery nurseries involving green cultivars, which suggests race 2 was the pathogenic type.

Use of celery transplants grown in seedbeds naturally infested with the pathogen appears to be the factor most responsible for the rapid spread of the pathogen to disease-free fields in the recent New York outbreaks of the disease. Celery growers in Orange County for years have produced their own transplants in seedbeds of organic soil from commercial celery fields. Soil placed in seedbeds generally was fumigated until recently with D-D (1,3-dichloropropene plus 1,2-dichloropropane) (1), but this fumigant is not effective in controlling Fusarium yellows. Extensive secondary and tertiary root decay has been observed on transplants produced in such seedbeds, and *F. o.* f. sp. *apii* race 2 has been isolated from the decayed tissues. Use of infected transplants, therefore, contributed significantly to the dissemination of the pathogen to commercial celery fields that had previously been free of infestation.

Several control measures for the disease have been tested but without

Table 2. Reactions of 39 celery and celeriac cultivars and plant introductions (PIs) to New York isolate (P-3) of *Fusarium oxysporum* f. sp. *apii* race 2 in artificially infested and naturally infested soil

Cultivar or PI	Mean disease index[v,w]		Cultivar (PI) reaction[x,y]
	Artificially infested soil	Naturally infested soil	
Stoke's Golden Plume	4.67 ab	4.55 ab	HS
Golden Detroit	4.44 abc	4.55 ab	HS
Golden Self-blanching	4.33 abc	4.77 a	HS
Cornell 619	4.80 a	4.11 abcde	HS
Burpee's Golden Self-blanching	4.44 abc	4.16 abcd	HS
Surepak	3.94 bc	3.72 cdef	MS
Tall Utah 52-70R Improved	4.39 abc	4.16 abcd	HS
Transgreen	4.16 abc	4.17 abcd	HS
Giant Pascal	4.16 abc	4.00 bcde	HS, MS
Florida 683	4.16 abc	4.10 abcde	HS
Tall Green Light	4.66 ab	4.78 a	HS
Tendercrisp	3.77 c	3.56 def	MS
Burpee's Fordhook	3.72 c	3.78 cde	MS
Stoke's Improved Utah 52-70	3.66 c	4.11 abcde	MS, HS
352 Marble Ball[z]	2.94 d	2.72 h	MR
F-1 Green Giant	4.10 abc	4.39 abc	HS
Clean Cut	4.21 abc	4.28 abcd	HS
Utah 52-70	4.11 abc	4.22 abcd	HS
Giant Prague[z]	2.89 de	2.78 gh	MR
Prague[z]	2.94 d	2.83 gh	MR
Large Smooth Prague[z]	2.27 def	2.39 hi	MR
Balder LD[z]	2.44 def	2.78 gh	MR
Alabaster[z]	2.11 ef	2.67 h	MR
Tall Utah 52-70 Strain 213	3.72 c	4.28 abcd	MS, HS
Japanese Green Giant	4.39 abc	4.44 abc	HS
PI 196831[z]	3.94 bc	3.72 cdef	MS
PI 204557[z]	2.39 def	2.61 h	MR
PI 174054[z]	2.27 def	2.50 hi	MR
PI 171500[z]	2.77 def	2.33 hi	MR
PI 176869[z]	2.83 de	2.33 hi	MR
PI 320994[z]	2.94 d	3.44 efg	MR, MS
PI 169007[z]	2.72 def	2.61 h	MR
PI 193454[z]	2.33 def	3.00 fgh	MR
PI 176419[z]	2.55 def	2.83 gh	MR
PI 169001[z]	1.94 f	1.83 i	HR
PI 169004[z]	2.50 def	2.55 h	MR
PI 254539[z]	2.27 def	2.61 h	MR
PI 289691[z]	2.83 de	2.55 h	MR
PI 418955	2.22 def	4.33 abc	MR, HS

[v] Average of three replicates. Disease index for each replicate was calculated for six plants, using a disease rating scale of 0–5, where 0 = healthy plant and 5 = dead plant.

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

[x] Based on mean disease index: 0–2.00 = highly resistant (HR), 2.01–3.00 = moderately resistant (MR), 3.01–4.00 = moderately susceptible (MS), and 4.01–5.00 = highly susceptible (HS). This is an arbitrary grouping scheme and may not be in agreement with the statistical groupings.

[y] Reaction identical in artificially infested soil and naturally infested soil unless indicated by both reactions.

[z] Celeriac.

Table 3. Reactions of 11 celery and celeriac cultivars and plant introductions (PIs) to *Fusarium oxysporum* f. sp. *apii* race 2 in naturally infested organic field soil[v]

Cultivar or PI	Mean disease index[w,x]	Cultivar (PI) reaction[y]
Giant Prague[z]	3.60 cde	MS
Tendercrisp	3.73 bcd	MS
Surepak	4.35 abc	HS
352 Marble Ball[z]	3.53 de	MS
Alabaster[z]	3.00 de	MR
Florida 683	4.60 a	HS
Large Smooth Prague[z]	3.33 de	MS
Golden Self-blanching	4.80 a	HS
Burpee's Fordhook	4.40 a	HS
PI 169001[z]	2.87 e	MR
PI 171500[z]	2.93 de	MR

[v] Soil previously planted once to celery in the greenhouse.

[w] Average of three replicates. Disease index for each replicate was calculated for six plants, using a disease rating scale of 0–5, where 0 = healthy plant and 5 = dead plant.

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

[y] Based on mean disease index: 0–2.00 = highly resistant (HR), 2.01–3.00 = moderately resistant (MR), 3.01–4.00 = moderately susceptible (MS), and 4.01–5.00 = highly susceptible (HS). This is an arbitrary grouping scheme and may not be in agreement with the statistical groupings.

[z] Celeriac.

Table 4. Reactions of 18 celery and celeriac cultivars and plant introductions (PIs) to infection by *Fusarium oxysporum* f. sp. *apii* race 2 in the field

Cultivar or PI	Mean disease index[w,x]	Cultivar (PI) reaction[y]
Burpee's Fordhook	3.78 a	MS
352 Marble Ball[z]	1.91 b	HR
Golden Self-blanching	4.72 a	HS
Utah 52-70	4.74 a	HS
Tendercrisp	3.98 a	MS
Florida 683	4.55 a	HS
Giant Pascal	4.46 a	HS
Transgreen	4.73 a	HS
Balder LD[z]	1.93 b	HR
Giant Prague[z]	1.74 b	HR
Surepak	4.56 a	HS
Prague[z]	2.00 b	HR
Alabaster[z]	2.25 b	MR
Large Smooth Prague[z]	2.00 b	HR
PI 204557[z]	1.64 b	HR
PI 171500[z]	1.76 b	HR
PI 169004[z]	1.80 b	HR
PI 169001[z]	1.82 b	HR

[w] Average of three experiments, each consisting of four replicates. Disease index for each replicate in an experiment was calculated for 10 plants, using a disease rating scale of 0–5, where 0 = healthy plant and 5 = dead plant.

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

[y] Based on mean disease index: 0–2.00 = highly resistant (HR), 2.01–3.00 = moderately resistant (MR), 3.01–4.00 = moderately susceptible (MS), and 4.01–5.00 = highly susceptible (HS). This is an arbitrary grouping scheme and may not be in agreement with the statistical groupings.

[z] Celeriac.

success for immediate application. A number of commercially grown celery and celeriac cultivars and plant introductions were evaluated in the greenhouse and in the field. None of the celery cultivars tested were found suitable either for direct use or as tolerant parents in a breeding program. Several celeriac plant introductions (169001, 169004, and 171500), however, were highly resistant. Similar results were reported by Opgenorth and Endo (9).

In a continued search for celery cultivars resistant to Fusarium yellows, Deacon, Tall Utah 52-70 HK, and Bishop as well as two Michigan State University lines (MSU 6837 and MSU 83604) were tested recently. Deacon and Bishop were tolerant of the disease in the field but were not horticulturally acceptable. MSU 83604 and Tall Utah 52-70 HK were susceptible and MSU 6837 was tolerant in both greenhouse and field studies but lacked desirable horticultural characteristics.

Chemical control of the disease appears unlikely, because attempts to control the disease in the field with fungicidal drenches (applied at the time of transplanting and also 4 wk later) were unsuccessful. In a separate experiment, preplant dips with imazalil (Fungaflor 75SP) at 500, 750, and 1,000 ppm a.i. also did not control the disease.

In the absence of disease-tolerant and horticulturally acceptable cultivars, growers are being advised to adopt sanitary culture procedures to limit the spread of the pathogen. Seedbeds are routinely fumigated with methyl bromide (0.68 kg/9.3 m^2; 1.5 lb/100 ft^2) to eradicate the pathogen. This fumigant destroys *F. o.* f. sp. *apii* race 2 in natural organic soil and in celery root pieces present in the soil (2). Although this practice allows production of healthy transplants, complete prevention of the spread of the pathogen, however, appears difficult because it would involve frequent cleaning of field equipment and control of windblown organic soil.

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