Studies on the Inheritance of Resistance to *Fusarium oxysporum* f. sp. *apii* in Celery

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ABSTRACT

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Fusarium yellows, incited by *Fusarium oxysporum* f. sp. *apii*, is currently the most important disease of celery (*Apium graveolens* L.) in North America. No effective control methods have been developed for this pathogen, and at present, breeding for resistance is the approach most widely used. Experiments were conducted to elucidate the inheritance of resistance to race 2 of the pathogen, which was observed previously in the celeriac line PI 169001. F_2 populations derived from a cross of this line with a putatively tolerant selection from the susceptible celery cultivar Tall Utah 52-70R showed segregation of resistance and susceptibility. With one exception, F_2 segregation ratios were between 3:1 and 15:1 resistant/susceptible. Results from progeny tests of 20 resistant F_2 plants were consistent with the conclusion that resistance in PI 169001 was conditioned by a dominant allele at a single locus and that segregation distortion was caused by segregating genes with quantitative effects on resistance contributed by the selected Tall Utah 52-70R parent or both parents. The F_2 segregation from a separate cross was also consistent with the hypothesis of a single dominant resistance gene and a quantitative resistance gene(s) in PI 169001.

Fusarium oxysporum f. sp. *apii* (Snyd. & Hans.) is a soilborne pathogen of celery (*Apium graveolens* L.) and is widely distributed in celery and celeriac-growing areas of North America and Europe (3,11). There are two primary races of the pathogen (12,14). Race 2 is very important in North America because of the lack of resistance in green celery cultivars, the lack of effective control methods, and the rapid spread of the pathogen among and within growing areas (3,11).

Knowledge of the inheritance of resistance in the host and of virulence in the pathogen is very important to the breeder in formulating strategies to breed varieties with effective and durable resistance. Although variable host resistance or tolerance to pathogens often has a simple genetic basis, cases of complex quantitative inheritance have been reported (5). Genetic interpretations are often confounded by the effects of environment on the expression of disease. This study was undertaken to determine the inheritance of host resistance to *F. oxysporum* f. sp. *apii* race 2 observed among a defined set of celery cultivars concomitant with the breeding of horticulturally acceptable race 2-resistant cultivars.

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MATERIALS AND METHODS

Description of host and pathogen material. Host lines used were *A. graveolens* PI 257228 (highly susceptible to race 2, USDA Plant Introduction Station, Geneva, NY), Tall Utah 52-70R (highly susceptible to race 2, Keystone Seed Company, Hollister, CA), and PI 169001 (highly resistant to race 2, USDA Plant Introduction Station, Geneva, NY). OXN40, a selection that showed no disease symptoms, was taken from a severely infested field of Tall Utah 52-70R in Oxnard, CA, in May 1979. The OXN40 S_2 population referred to in the text consisted of bulked, second-cycle, selfed seed descending from the original single plant selection. Twenty S_1 plants were used to produce the S_2 seeds. Similarly, the PI 169001 S_2 population descended from the single plant used as the resistant parent in the original cross. The original reciprocal PI 169001 × OXN40 and OXN40 × PI 169001 hybrids were made, using the same plants for both the male and female parents, by the method of Honma (4). F_2 populations consisted of bulked progeny from five OXN40 × PI 169001 F_1 plants and 12 PI 169001 ×OXN40 F_1 plants.

Two inoculation protocols were used (Table 1). The first was slightly modified from that reported by Schneider (15). A potato-dextrose agar (PDA) slant of a *F. oxysporum* f. sp. *apii* race 2 isolate was collected from an infested celery field near Oceano, CA, in January 1979 and maintained by periodic single-spore transfers to PDA. A small piece of mycelium was obtained from R. W. Schneider, aseptically transferred into 250 ml of sterile potato-dextrose broth (PDB), and cultured on a rotary shaker (125 rpm) for about 3 days at 25 C and 12 hr/day under fluorescent light. The entire culture was then diluted into 2 L of a sterile 0.01 M L-asparagine solution (Sigma, St. Louis). About 30 ml of the resulting microconidial suspension was added to 50 g of sterile, milled barley straw in 1-L dilution bottles (enough to thoroughly moisten the straw). The infested straw was shaken vigorously to thoroughly mix the inoculum and incubated for 14 days at 25 C under fluorescent light. The straw was then air-dried at 25 C and stored at 4 C until used. With this method, virulence remained stable after more than 4 yr of storage.

Steam-sterilized potting mix (1:1:1, v/v/v, sand/peat moss/Yolo loam) was mixed with the barley straw inoculum at a rate of 1 kg moist soil mix to 1 g dry barley straw inoculum. This yielded about 10^5 colony-forming units per gram of soil, as estimated on the basis of dilution plating of the dried barley straw inoculum. Infested soil was mixed for 20–30 min in a concrete mixer and dispensed into 12-cm^2 plastic pots. Eight- to 10-wk-old celery seedlings were transplanted in infested soil, one plant per pot. Plants were grown in a greenhouse in a completely randomized design at 22–25 C during the day with ambient light and at 18–20 C during the night. Pots were watered daily and fertilized once a week to saturation with full-strength Hoagland nutrient solution.

The second inoculation protocol was as described by Opgenorth and Endo (8), except 10-wk-old celery seedlings were transplanted in infested soil in 18-cm round plastic pots, four plants per pot (experiment 2). The inoculum isolate was derived from H171, a race 2 type obtained from R. M. Endo, Department of Plant Pathology, University of California, Riverside. Plants were grown in a greenhouse under ambient light at 22–25 C during the day and 18–20 C during the night.

Disease severity was determined visually by splitting plants longitudinally through the crown at the base of the rosette and estimating the severity of symptoms in the crown and foliar vascular and cortical tissues by a disease rating (DR) scale of 0–7 (Fig. 1), where 0 = no symptoms; 1 = vascular discoloration, primary roots; 2 = mild vascular discoloration in lower half of crown; 3 = severe vascular discoloration in entire crown; 4 = mild rot in crown; 5 = severe rot in crown, slight foliar chlorosis; 6 = severe rot in crown, severe foliar chlorosis, and wilt; and 7 = complete

Table 1. Summary of planting and scoring dates, inoculation protocols, and population sample sizes for experiments 1–5

Experiment	Inoculation protocol[a]	Date transplanted in infested soil	Date scored	Population	Sample size
1	1	12 Feb. 1981	2 Apr. 1981	Tall Utah 52-70R	16
				PI 169001 S_2	72
				OXN40 S_2	71
				PI 169001 × Tall Utah 52-70R F_1	32
				PI 169001 × OXN40 F_2	96
				OXN40 × PI 169001 F_2	49
2	2	12 Feb. 1981	5 May 1981	PI 169001 S_2	16
				Tall Utah 52-70R	16
				PI 169001 × OXN40 F_2	60
3	1	18 June 1981	17 Aug. 1981	Tall Utah 52-70R	6
				PI 169001	6
				OXN40 S_2	71
				PI 169001 × OXN40 F_2	195
				OXN40 × PI 169001 F_2	197
4	1	6 Apr. 1982	19 May 1982	See Table 3	
5	1	24 Feb. 1983	24 May 1983	Tall Utah 52-70R	20
				PI 169001 × PI 257228 F_2	163

[a] See Materials and Methods.

necrosis.

Three separate evaluations were made of each plant by different individuals, the mean of which served as the final DR. At least one of these evaluators participated in all five experiments. A summary of inoculation protocol used, planting and scoring dates, and population sample sizes is provided in Table 1.

RESULTS

Experiment 1: F_2 segregation, inoculation protocol 1. Unselected Tall Utah 52-70R plants showed the most severe disease symptoms of all lines tested, with 81% of plants killed or severely necrotic (DR 7, Fig. 2A). The remaining 19% of the plants showed moderate to severe disease (average DR 3–6). The cultivar Tall Utah 52-70 HK showed intermediate levels of disease severity between PI 169001 and Tall Utah 52-70R. This agrees with previous observations (7,8). The OXN40 S_2 population showed a range of DR from 0 (no disease) to 7 (dead) (Fig. 2). The PI 169001 S_2 population was the most resistant (96% DR 0–1), with the remaining 4% showing DR of 3–5 (Fig. 2C).

Ten F_1 plants from the crosses tested for resistance to race 2 during summer 1980 were resistant. A PI 169001 × Tall Utah 52-70R F_1 population was produced during summer 1980 and was tested for disease resistance in experiment 1. About 97% of these F_1 plants showed only minor or no disease symptoms (DR 0–2, Fig. 2D) and the remaining 3% displayed moderate disease symptoms (DR 4–5).

Individuals with DR of 0–1.99 were classified as resistant (on the basis of the standard response of the PI 169001-resistant parent) and those with DR of 2–7 as susceptible. The distribution of the OXN40 × PI 169001 F_2 population with respect to DR was bimodal (Fig. 2E). The observed ratio was not significantly different from 1:1 but was significantly different from all other ratios tested (chi-square tests, Table 2). Distribution of the PI 169001 × OXN40 F_2 was also bimodal and not significantly different from 15:1 resistant/susceptible (Fig. 2F, Table 2) but was significantly different from 1:1 and 3:1.

Table 2. Chi-square contingency values for observed and expected ratios of resistance to susceptible plants in F_2 populations

Population	R/S[a]	Chi-square values 1:1	3:1	15:1
Experiment 1				
PI 169001 × OXN40 F_2	86:10	174.48*[b]	10.89*	2.84 NS
OXN40 × PI 169001 F_2	26:23	0.18 NS[c]	11.72*	138.59*
Experiment 2				
PI 169001 × OXN40 F_2	50:10	26.67*	2.29 NS	11.11*
Experiment 3				
PI 169001 × OXN40 F_2	169:26	104.86*	14.15*	16.69*
OXN40 × PI 169001 F_2	169:28	100.91*	12.22*	21.33*
Experiment 5				
PI 169001 × PI 257228 F_2	127:41	44.02*	0.03 NS	94.50*

[a] Observed ratio of resistant (DR 0–1.99) to susceptible (DR 2–7) plants.
[b] * = Highly significant ($P \leq 0.001$).
[c] NS = Not significant.

Experiment 2: F_2 segregation, inoculation protocol 2. Disease developed much more slowly with the second inoculation procedure. On the same host lines, four to five additional weeks of growth were required to produce symptoms of equivalent severity. OXN40 S_2 plants were not tested in this experiment, but those of Tall Utah 52-70R showed moderate to severe disease (DR 4–7, Fig. 3A) and PI 169001 S_2 plants had little or no disease (DR 0–1.99, Fig. 3B). The distribution of DR among PI 169001 × OXN40 F_2 plants was not clearly modal (Fig. 3C). Using the same criteria as those in experiment 1 for resistance and susceptibility, the ratio of resistant/susceptible PI 169001 × OXN40 S_2 plants was not significantly different from 3:1 but differed significantly ($P = 0.01$, chi-square test) from 15:1 (Table 2).

Experiment 3: F_2 segregation, inoculation protocol 1. As in experiment 1, the OXN40 S_2 population in experiment 3 showed a much broader range of disease ratings than Tall Utah 52-70R, from which it was selected. Moreover, Tall Utah 52-70R plants were all severely diseased (Fig. 4A), whereas most OXN40 S_2 plants were only moderately diseased (DR 3–6, Fig. 4B), and 4% were resistant. Plants of the PI 169001 S_2 population were 100% resistant (Fig. 4C).

The distribution of the relative frequency of resistant and susceptible plants in the OXN40 × PI 169001 F_2 population was continuous (Fig. 4D). The ratio of resistant/susceptible plants in this population was significantly different from 1:1, 3:1, and 15:1 (Table 2). The PI 169001 × OXN40 F_2 population showed a distribution of DR and ratio of resistant/susceptible plants nearly identical to the OXN40 × PI 169001 F_2 reciprocal (Fig. 4E, Table 2), unlike comparable results in experiment 1.

Experiment 4: F_3 and backcross segregations. Selfed and backcross progeny of 20 resistant F_2 selections were tested using inoculation protocol 1 to further determine character segregation. F_2 selections of PI 169001 and OXN40 cytoplasms were bulked, and in all cases but two, sample sizes equaled or exceeded 25 per family. All but one of the 33 PI 169001 plants tested were resistant,

whereas all Tall Utah 52-70R plants were susceptible (Table 3). As observed in experiments 1 and 3, the OXN40 S_2 population contained certain plants that were clearly less diseased than Tall Utah 52-70R. However, no plants observed in this experiment were considered resistant.

The F_2 progeny families could be classified into one of three groups on the basis of resistant/susceptible segregation ratios. Four families were completely resistant, whereas the remaining 16 families had at least two susceptible plants. The ratio of F_2 progeny families that did and did not segregate (16:4) was significantly different from 2:1 (chi-square test), the expected ratio if the original PI 169001 parent had been homozygous for a single dominant allele conferring resistance and if the OXN40 parent had been homozygous for a corresponding recessive allele for susceptibility. Of the segregating class, most behaved as if resistance was conditioned by a single dominant allele and the F_2 parent was heterozygous (ie, not significantly different for 3:1 if F_3 and 1:1 if backcross to Tall Utah 52-70R). However, at least three of these 16 families showed significant departures from these predictions. The direction of the distortion was consistently toward overrepresentation of the resistant class. The susceptible class of all F_2 progeny families contained plants that showed intermediate DR (2–5), unlike the susceptible control, which consisted only of plants showing DR of 5–7.

Experiment 5: PI 169001 × PI 257228 F_2 segregation. The segregating populations employed in experiments 1–4 were derived from a cross of a resistant plant (PI 169001), with one carrying at least some quantitative resistance genes (OXN40). The simultaneous segregation of different resistance genes could have caused the high representation of inoculated test plants in the intermediate disease response range (DR 2–5), thus making identification of individual genes difficult. For this reason, an F_2 population of PI 169001 × PI 257228 (a true-breeding, very highly susceptible line) was tested for segregation of resistance. The F_1 was completely resistant (n = 97). Using the criteria established to distinguish resistance and susceptibility (DR 0–1.99 = resistant and DR 2–7 = susceptible), a ratio of 122 resistant/41 susceptible plants was observed among F_2 progeny (Table 4), which was not significantly different from 3:1 (chi-square test). However, the susceptible class again had a high representation of intermediate DR (2–5). Lack of sufficient seed forced us to use Tall Utah 52-70R as the susceptible check in this experiment. Previous experiments

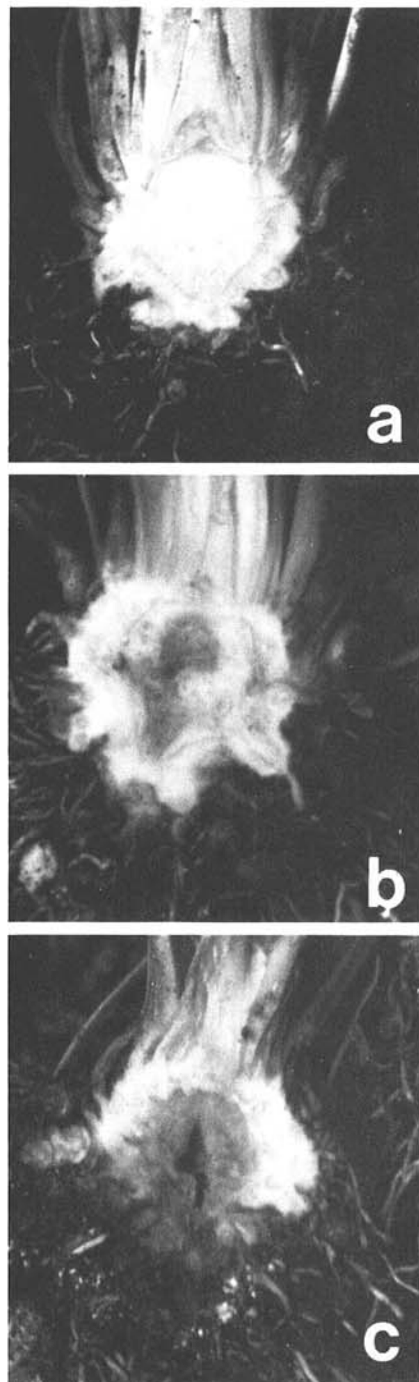

Fig. 1. Vascular discoloration in crown tissue of celery as a criterion for rating disease severity after infection with *Fusarium oxysporum* f. sp. *apii* race 2. Disease rating (DR) scale: 0 = no symptoms; 1 = vascular discoloration, primary roots; 2 = mild vascular discoloration in lower half of crown; 3 = severe vascular discoloration in entire crown; 4 = mild rot in crown; 5 = severe rot in crown, slight foliar chlorosis; 6 = severe rot in crown, severe foliar chlorosis, and wilt; and 7 = complete necrosis. **(A)** DR = 0, **(B)** DR = 3, and **(C)** DR = 5.

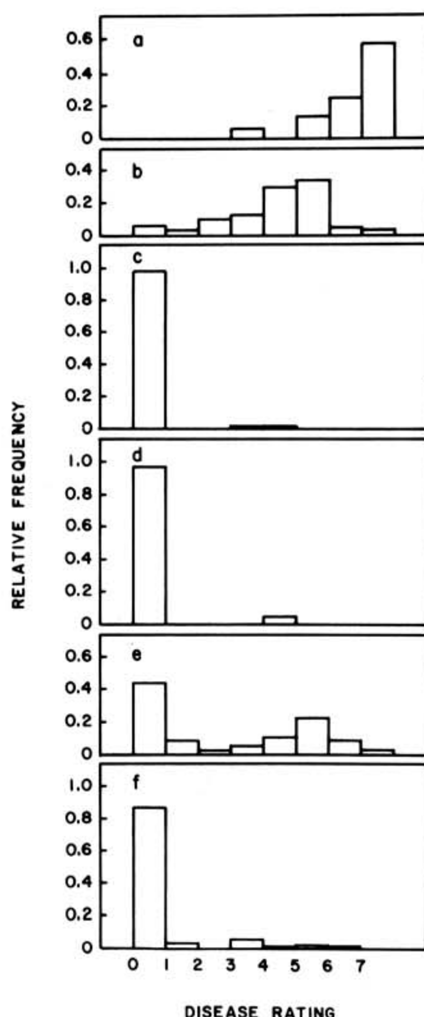

Fig. 2. Results of experiment 1 (F_2 segregation, inoculation protocol 1): (a) cultivar Tall Utah 52-70R, (b) OXN40 S_2, (c) PI 169001 S_2, (d) PI 169001 × Tall Utah 42-70R F_1, (e) OXN40 × PI 169001 F_2 and (f) PI 169001 × OXN40 F_2. Disease rating scale: 0 = no symptoms; 1 = vascular discoloration, primary roots; 2 = mild vascular discoloration in lower half of crown; 3 = severe vascular discoloration in entire crown; 4 = mild rot in crown; 5 = severe rot in crown, slight foliar chlorosis; 6 = severe rot in crown, severe foliar chlorosis, and wilt; and 7 = complete necrosis.

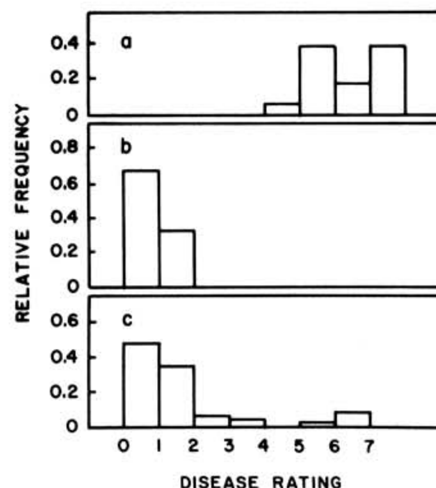

Fig. 3. Results of experiment 2 (F_2 segregation, inoculation protocol 2): (a) cultivar Tall Utah 52-70R, (b) PI 169001 S_2, and (c) PI 169001 × OXN40 F_2. Disease rating scale: 0 = no symptoms; 1 = vascular discoloration, primary roots; 2 = mild vascular discoloration in lower half of crown; 3 = severe vascular discoloration in entire crown; 4 = mild rot in crown; 5 = severe rot in crown, slight foliar chlorosis; 6 = severe rot in crown, severe foliar chlorosis, and wilt; and 7 = complete necrosis.

had shown that PI 257228 was more susceptible to race 2 than Tall Utah 52-70R (9). In our experiment, 18 of 20 susceptible check plants were dead and the remaining two had a DR of 6.

Other experiments. Numerous accessions in addition to PI 169001 were scored as resistant by Opgenorth and Endo (8) and Orton (9) in tests with race 2 in *A. graveolens* germ plasm. Extensive genetic studies on alternative sources of race 2 resistance have not been pursued. However, a few crosses were performed to explore the possibility that other sources of resistance were nonallelic to resistance in PI 169001. The approach was to synthesize F_1 hybrids of resistant lines and then testcross the hybrids to Tall Utah 52-70R (highly susceptible). Assuming the original resistant lines were homozygous (a reasonable assumption because of the apparent absence of susceptible plants in these lines), all testcross progeny should have been resistant if the genetic source of resistance was allelic. However, if the resistance was encoded by separate unlinked genes and recessive susceptible alleles were borne by plants at the corresponding resistance locus of the other parent in the original cross, one-fourth of the testcross progeny should have been susceptible. Among three such allelism tests performed, two showed some segregation among testcross progeny (Table 5); however, the segregation ratios observed in these families were clearly different from the expected 3:1 predicted earlier.

DISCUSSION

Intensive efforts have focused on developing effective artificial methods to distinguish resistance from susceptibility in celery to *F. oxysporum* f. sp. *apii*. Transplanting established seedlings into infested soil and maintaining them under ideal conditions for 6–8 wk has yielded the most consistent results (9,10,15). This technique produces results similar to those obtained from experiments planted in naturally infested fields, except they are more severe and definitive (T. J. Orton, *unpublished*). Puhalla (13) examined the genetic relationships of a wide spectrum of race 2 isolates and concluded that they are all nearly identical. Hence, the screening procedures described are a reasonable and desirable substitute for direct field screening.

Tall Utah 52-70R was highly susceptible in all five experiments. PI 169001 was highly resistant, with rare exceptional susceptible responses. S_2 populations of OXN40 consistently showed a range of disease responses, some exceeding the resistance of Tall Utah 52-70R, the line from which it was selected (Figs. 1 and 3, Table 3). S_2 populations of the original PI 169001 parent were 100% resistant in experiments 2 and 3 but only 97% resistant in experiment 1 (Fig. 3C). Moreover, PI 169001 × Tall Utah 52-70R F_1 hybrids were also 97% resistant and 3% susceptible in this experiment (Fig. 3D). Possible sources of the susceptible plants in the populations are 1) mutation to susceptibility at the presumed resistance locus, 2) contaminated seed lots, 3) instability of resistance phenotype (incomplete penetrance, variable expressivity), and 4) rare genetic recombinants for genotypes giving rise to susceptibility by epistasis. Because susceptible plants in presumably resistant genetic backgrounds

Table 3. Segregation ratios among 20 PI 169001 × OXN40 (and reciprocal) F_2 progeny families (experiment 4)

F_2 parent	Population type[a]	Number of plants		χ^2 (3:1 or 1:1)[b]
		Resistant	Susceptible	
2	F_3	31	18	NS[c]
5	F_3	22	6	NS
8	BC	7	11	NS
9	F_3	29	4	NS
11	F_3	28	4	NS
	BC	12	10	NS
14	BC	18	18	NS
15	F_3	32	2	*
16	F_3	24	4	NS
17	F_3	38	0	**
19	F_3	12	0	**
	BC	16	0	**
21	F_3	29	3	*
	BC	9	6	NS
22	F_3	27	2	*
24	F_3	24	4	NS
25	F_3	21	8	NS
	BC	14	9	NS
27	F_3	28	2	*
30	F_3	32	10	NS
32	F_3	33	0	**
35	F_3	31	0	**
38	F_3	16	5	NS
50	F_3	26	4	NS
Resistant control (PI 169001)		32	1	
Susceptible control (Tall Utah 52-70R)		0	39	

[a] F_3 = Bulked self-pollinated progeny and BC = bulked progeny of backcrosses to cultivar Tall Utah 52-70R.
[b] 3:1 If F_3, 1:1 if BC.
[c] * = $P \leq 0.05$, ** = $P \leq 0.01$, and NS = not significant.

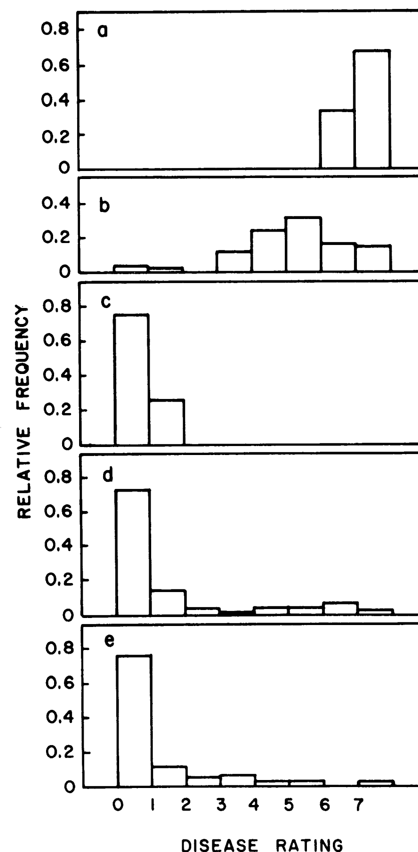

Fig. 4. Results of experiment 3 (F_2 segregation, inoculation protocol 1): (a) cultivar Tall Utah 52-70R, (b) OXN40 S_2, (c) PI 169001, (d) OXN40 × PI 169001 F_2, and (e) PI 169001 × OXN40 F_2. Disease rating scale: 0 = no symptoms; 1 = vascular discoloration, primary roots; 2 = mild vascular discoloration in lower half of crown; 3 = severe vascular discoloration in entire crown; 4 = mild rot in crown; 5 = severe rot in crown, slight foliar chlorosis; 6 = severe rot in crown, severe foliar chlorosis, and wilt; and 7 = complete necrosis.

Table 4. Segregation of resistance to *Fusarium oxysporum* f. sp. *apii* race 2 among F_2 progeny of PI 169001 × PI 257228

Disease rating[a]	No. of plants
Resistant	
0	107
1	15
Total	122
Susceptible	
2	14
3	6
4	5
5	6
6	7
7	3
Total	41

[a] Resistant = $0 \leq DR < 2$, susceptible = $2 \leq DR \leq 7$.

Table 5. Race 2 resistance allelism test among *Apium graveolens* germ plasm lines

Pedigree	Number of plants	
	Resistant[a]	Susceptible
(PI 257231 × PI 320912) × Tall Utah 52-70R	37	2
(PI 320912 × PI 169008) × Tall Utah 52-70R	27	0
(PI 176418 × PI 169005) × Tall Utah 52-70R	4	4

[a] Resistant = $0 \leqslant DR < 2$, susceptible = $2 \leqslant DR \leqslant 7$.

occurred at such low frequencies, effects on segregation ratios should have been inconsequential.

The F_1 of PI 169001 × Tall Utah 52-70R behaved almost identically to PI 169001 with respect to resistance, prompting the conclusion that the resistance trait is dominant. However, segregation patterns observed among F_2 and F_3 populations of OXN40 × PI 169001 (and reciprocal) and PI 169001 × PI 257228, though supporting the conclusion of dominant resistance, were not consistent with simple inheritance. First, four of five independent tests of OXN40 × PI 169001 and reciprocal F_2 populations yielded segregations somewhere between 3:1 and 15:1 resistant/susceptible. The remaining F_2 population differed strikingly from the rest in being significantly different from 3:1 but not significantly different from 1:1 (Fig. 2E). The reasons for this are unknown. Moreover, OXN40 × PI 169001 (and reciprocal) F_2 plants could be divided into three classes on the basis of segregation patterns observed among F_3 and backcross progeny: 13, 3, and 4 populations, respectively, showed ratios expected if the F_2 were 1) heterozygous for a dominant resistant and recessive susceptible allele, 2) giving a resistant/susceptible ratio significantly greater than that expected in case 1, and 3) 100% resistant (Table 3). Only two classes, ie, segregating 3:1 (67%) and 100% resistant (33%), should have been observed if resistance was conditioned only by a single dominant gene.

A second set of observations was also inconsistent with the hypothesis of simple inheritance. Resistant and susceptible checks in all experiments had distinctly different DR. However, a significant representation of plants in all experiments scored as susceptible showed DR intermediate to those of the check lines. In the OXN40 × PI 169001 and reciprocal derivatives, this was probably at least partly due to alleles contributing quantitatively to resistance from the OXN40 parent. Such alleles could also have contributed to the observed resistant/susceptible segregation ratios described before. The high incidence of intermediate responses observed among F_2 progeny of PI 169001 × PI 257228 can only be explained by the contribution of a single dominant resistance gene and other genes of relatively quantitative effect from PI 169001. One consequence of such nonmodal distributions was that the distinction of resistance from susceptibility became more arbitrary. It is possible in these cases that slight variances in scoring perceptions or environment contributed to unusual segregation ratios.

Previous studies of wilts caused by *F. oxysporum* pathogens in *Lycopersicon* sp., *Cucumis sativus, C. melo,* and *Citrullis vulgaris* (by race 1) have demonstrated the existence of dominant resistance alleles at single loci (1,6,7,14,16). However, resistance to *F. oxysporum* in sweet potato and radish has been reported to be inherited quantitatively (2,12).

These data support the following conclusions: 1) resistance to *F. oxysporum* f. sp. *apii* race 2 from PI 169001 is dominant over susceptibility from Tall Utah 52-70R, 2) a single major dominant gene and independent quantitative genes contributing to resistance are present in PI 169001, 3) quantitative genes conditioning enhanced resistance can be selected from normally susceptible populations such as Tall Utah 52-70R, and 4) preliminary evidence indicates that at least some race 2-resistant lines carry different resistance genes.

Efforts are being made to transfer the genes for resistance from PI 169001 into North American commercial celery genetic background (9,10). Individuals segregating for resistance and desirable horticultural characters have been observed in BC_1 and BC_2 populations. Trials in severely infested fields have shown the gene or genes to be effective in reducing disease losses to well below the economic threshold. Evidence further indicates the existence of resistance genes at loci independent of the putative major resistance locus in PI 169001. Perhaps, different resistance alleles could be accumulated in an acceptable variety to enhance the stability of resistance.

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LITERATURE CITED

1. Bohn, G. W., and Tucker, C. M. 1939. Immunity to Fusarium wilt in the tomato. Science 89:603-604.
2. Collins, W. W. 1977. Diallel analysis of sweet potatoes for resistance to Fusarium wilt. J. Am. Soc. Hortic. Sci. 102:109-111.
3. Hart, L. P., and Endo, R. M. 1978. The reappearance of Fusarium yellows of celery in California. Plant Dis. Rep. 62:138-142.
4. Honma, S. 1959. A method for celery hybridization. Proc. Am. Soc. Hortic. Sci. 73:345-348.
5. Nelson, R. R. 1973. Genetics of horizontal resistance to plant diseases. Annu. Rev. Phytopathol. 16:359-378.
6. Netzer, D., Niego, S., and Galun, E. 1977. A dominant gene conferring resistance to Fusarium wilt in cucumber. Phytopathology 67:525-527.
7. Netzer, D., and Weintall, C. 1980. Inheritance of resistance in watermelon to race 1 of *Fusarium oxysporum* f. sp. *niveum*. Plant Dis. 64:853-854.
8. Opgenorth, D. C., and Endo, R. M. 1979. Sources of resistance to Fusarium yellows of celery in California. Plant Dis. Rep. 63:165-169.
9. Orton, T. J. 1980. Breeding celery for disease resistance and improved quality. Pages 45-56 in: California Celery Research Program, 1978–1979 Annual Report. F. Pusateri, ed. Calif. Celery Res. Adv. Board Publ.
10. Orton, T. J. 1982. Breeding celery for disease resistance and improved quality. Pages 41-62 in: California Celery Research Program, 1980–1981 Annual Report. F. Pusateri, ed. Calif. Celery Res. Adv. Board Publ.
11. Orton, T. J., and Puhalla, J. E. 1980. Researchers take aim on *Fusarium* in celery. Western Grower and Shipper 51:22-28.
12. Peterson, J. L., and Pound, G. S. 1960. Studies on resistance in radish to *Fusarium oxysporum* f. sp. *conglutinans*. Phytopathology 50:807-816.
13. Puhalla, J. E. 1982. Relationships among races of the Fusarium yellows pathogen on celery in California. Pages 65-72 in: California Celery Research Program, 1980–1981 Annual Report. F. Pusateri, ed. Calif. Celery Res. Adv. Board Publ.
14. Risser, G., Banihasheni, Z., and Davis, D. W. 1976. A proposed nomenclature of *Fusarium oxysporum* f. sp. *melonis* races and resistance genes in *Cucumis melo*. Phytopathology 66:1105-1106.
15. Schneider, R. W. 1979. Etiology and control of Fusarium yellows of celery. Pages 15-26 in: California Celery Research Program, 1977–1978 Annual Report. F. Pusateri, ed. Calif. Celery Res. Adv. Board Publ.
16. Stall, R. E., and Walter, J. M. 1965. Selection and inheritance of resistance in tomato isolates of races 1 and 2 of the Fusarium wilt organism. Phytopathology 55:1213-1215.