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Proc. Fla. State Hort. Soc. 102:358-360. 1989.

FUSARIUM WILT OF TOMATO IN FLORIDA BEFORE AND AFTER AN OVERSEASONING PERIOD

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Additional Index Words. Lycopersicon esculentum, Fusarium oxysporum f. sp. *lycopersici.*

Abstract. **During August 1987, a field of fine sand was artificially infested with races 1, 2, and 3 of the tomato *Lycopersicon esculentum* Mill.) wilt Fusarium (*Fusarium oxysporum* (Schlecht.) f. sp. *lycopersici* (Sacc.) Snyder and Hansen. The field then was planted to 2 consecutive tomato crops, a fall crop followed by a spring crop. The 3-month midwinter overseasoning period between crops greatly reduced the incidence of Fusarium wilt of race 1-resistance and tolerant 'Improved Rutgers' (caused by race 2 or 3), race 1-resistant 'Manapal' (caused by race 2 or 3), race 1 and 2-resistant 'Walter' (caused by race 3). Disease incidence caused by race 1 (but not race 2 or 3) was slightly decreased on susceptible 'Bonny Best.' Yield losses were sharply reduced by the overseasoning period on 'Rutgers' (caused by race 2 or 3), 'Manapal' (caused by race 2 or 3), 'Walter' (caused by race 3), and 'Bonny Best' (caused by race 1 or 2, but not race 3). Based on yields and disease incidence, race 3 survived better than race 2, and race 2 survived better than race 1. 'Manapal' was less susceptible to race 2 or 3 than 'Bonny Best' and no more susceptible than 'Improved Rutgers' with genes for tolerance to race 1 in addition to the I gene for resistance to race 1. 'Walter' was less susceptible to race 3 than 'Bonny Best' and no more susceptible than 'Improved Rutgers' or 'Manapal.'**

Florida Agricultural Experiment Station Journal Series No. N-00088.

Fusarium wilt, caused by *Fusarium oxysporum* (Schlecht.) f. sp. *lycopersici* (Sacc.) Snyder and Hansen, remains one of the most destructive diseases of tomato. The most practical means of control is the use of resistant cultivars. With the appearance of race 3, however, there are no resistant cultivars available for the growers of fresh market tomatoes in the United States. Moreover, some question the wisdom of the use of monogenic resistance claiming that cultivars with monogenic resistance will be highly susceptible should a pathogenic race appear that is able to incite wilt of these heretofore resistant cultivars (1,4).

Rotation with other crops is another means of reducing the severity of Fusarium wilt. However, the literature indicates that years are necessary to rid the soil of race 1 (2,3). No information or estimates are available on the length of time needed for populations of races 2 and 3 to decline enough to permit a second crop to be grown successfully. It was stated that race 1 should survive better than race 2, and that race 2 should survive better than race 3 (4). However, no experimental evidence was presented to support this view.

A field experiment, therefore, was carried out to obtain information on the relative overseasoning ability of races 1, 2, and 3 of the tomato wilt Fusarium and to determine the effect of the 3 races on the incidence and severity of disease on 4 tomato cultivars with different wilt-resistant genotypes.

Materials and Methods

Raised beds of EuaGallie fine sand were formed, fertilized (using accepted commercial practices), and fumigated 11 August 1987. A 67% methyl bromide: 33% chloropicrin broad-spectrum fumigant was used at a 350 lb./acre. Beds were covered immediately after fumigation with 1.25 mil white-on-black polyethylene mulch. Two weeks later 2.25 inch diameter holes (40 holes per 50 foot long whole plot) were cut through the mulch in the drill row. The soil in 10 of these holes were infested with race 1, 10 with race 2, 10 with race 3, and 10 remained noninfested. Approximately 92 million microspores, which had been grown 2 weeks at 82F on vermiculite saturated with a liquid medium high in ammonia-nitrogen and micronutrients, were used to infest the soil in each hole. The next day, container-grown plants were set into the holes. Four cultivars were used: 1) 'Bonny Best' (no tolerance or resistance genes), 2) 'Manapal' (I gene for resistance to race 1), 3) 'Walter' (I and I2 genes for resistance to races 1 and 2, respectively), and 4) 'Improved Rutgers' (I gene for resistance to race 1 and polygenes for tolerance to race 1). Whole plots were cultivars (40 plants of one cultivar/whole plot) and were replicated 4 times. Subplots consisted of 3 Fusarium races (10 plants/race plus 10 control plants in noninfested soil).

All plants were evaluated for Fusarium wilt symptoms weekly throughout the season, but only the 11 Nov. data are presented. Fruit were harvested 3 times at weekly intervals from 17 Nov. to 2 Dec. Then all plants were severed at ground level and the vines removed from the field. Plots were left intact and were reset with container-grown plants of the some 4 cultivars on 2 Mar. 1988. Each cultivar was set into its respective plots from the preceding season. These plants in turn were evaluated weekly for symptoms of Fusarium wilt; however, only data from the 23 May evaluations are presented. Fruit were harvested 3 times from 24 May to 16 June.

Fertilizer was supplied to the second crop of the same rate as the first crop by cutting holes through the mulch on the bed shoulder and plugging in the fertilizer. Commercial insecticides and fungicides were applied to both crops as needed.

Results and Discussion

In the first crop, 6% of the 'Bonny Best' plants in noninfested plots were exhibiting Fusarium wilt symptoms by 17 Nov., indicating some Fusarium contamination or regrowth (Table 1). At that time, 96, 94, and 96% of the 'Bonny Best' plants were showing symptoms caused by races 1, 2 and 3, respectively. None of the 'Manapal' plants in noninfested soil were diseased and only 4% of the plants in race 1-infested soil were diseased. Only 38 and 60% of the 'Manapal' plants in race 2 and race 3-infested plots, respectively, showed wilt symptoms, indicting that 'Manapal' with the I gene for resistance to race 1 was not ultra-susceptible to race 2 or race 3. Certainly it was far more tolerant to these 2 races than 'Bonny Best.' None of the 'Walter' plants (resistant to races 1 and 2) in noninfested, race 1, or race 2-infested plots developed *wilt symptoms* and only 62% of the plants were affected by race 3. Obviously, 'Walter' was not ultra-susceptible to race 3, in contrast to some predictions. None of the 'Improved Rutgers' plants in noninfested soil were diseased and only 8% were dilseased in race 1-infested soil. However, race 2 and race 3 caused 68 and 54%, respectively, of the plants to be diseased. Nonetheless, the incorporation of the I gene for resistance to race 1 into the tolerant 'Rutgers' genotype had not created a highly race 2 and 3-susceptible cultivar compared to 'Bonny Best.' Additionally the incidences of disease caused by races 2 and 3 on 'Manapal' were no greater than those caused by these races on the 'Improved Rutgers' cultivar, and the incidence of disease caused by race 3 on 'Walter' was no greater than that caused by race 3 on 'Improved Rutgers.' So the developers of 'Improved Rutgers,' 'Manapal,' and 'Walter' by incorporating single dominant genes for resistance had not developed cultivars that would drastically succumb to new pathogenic races if the Fusarium wilt pathogen.

Table 1. Per cent incidence of Fusarium wilt of tomato cultivars of different Fusarium wilt resistance genotypes planted in field plots infested with races 1, 2, or 3.

	Fusarium wilt incidence (%)[z]							
	Fall crop (16 Nov.)				Spring crop (23 May)			
Cultivar	NI	R1	R2	R3	NI	R1	R2	R3
Bonny Best[y]	6	96	94	96	30	70	94	100
Manapal	0	4	38	60	2	0	4	36
Walter	0	0	0	62	0	0	0	30
Improved Rutgers	0	8	68	54	0	0	2	22
LSD (5%)		22.0				23.7		

[z]NI = non-infested; R1, R2, R3 = race 1, race 2, and race 3, respectively.
[y]'Bonny Best' = no genes for tolerance or resistance, 'Manapal' = I gene for resistance to race 1, 'Walter' = I and I2 genes for resistance to races 1 and 2, 'Improved Rutgers' = I gene for resistance to race 1 and polygenes for tolerance to race 1.

During the second season, 30% of the 'Bonny Best' plants in noninfested soil by first harvest were diseased (Table 1). Race 1, 2, and 3 caused 70, 94, and 100% wilt, respectively. Only the incidence of disease caused by race 1 was less than that caused by race 1 at harvest time in the first crop, indicating that race 1 had not survived the 3 month midwinter overseasoning period as well as races 2 and 3. During the second crop, 2% of the 'Manapal' plants in noninfested soil developed symptoms. There was no disease in race 1-infested plots and only 4% of the 'Manapal' plants were affected by race 2, a reduction in incidence from the first crop of 89%. The incidene of disease caused by race 3 decreased from 60 to 36%, a reduction of 40%. Consequently, race 3 seemed to survive better than rae 2. During the first crop 62% of the 'Walter' plants succumbed to race 3, whereas only 30% wilted the second crop, a reduction of 52%. The incidence of race 1-incited disease of 'Improved Rutgers' decreased from 8 to 0%, that of race 2-incited disease from 68 to 2% (97% reduction), and that of race 3-incited disease from 54 to 22% (59% reduction). Using disease incidence as an indication of survival ability of each race, it would seem that race 1 did not survive as well as race 2 and that race 2 did not survive as well as race 3.

During the first season, total 'Bonny Best' yields in race 1, 2, and 3-infested soil were 20, 20, and 6%, respectively, of the yields of 'Bonny Best' in noninfested soil (Table 2). The second season 'Bonny Best' yield loss, compared to noninfested plots, was greatly alleviated. However, 'Bonny Best' yields in race 1-infested plots were greater than race 2-infested plot yields, which in turn were greater than yields in the race 3-infested plots. This also indicated that race 3 survived better than race 2 and that race 2 survived better than race 1. Yields of the other cultivars, compared to the yields obtained from noninfested plots, greatly increased the second season.

A 3 month midwinter overseasoning period between tomato crops seemingly resulted in greatly reducing the incidence and severity of Fusarium wilt caused by race 1, 2, or 3. Perhaps a 5 to 7 year crop rotation, as Jones, et al. (2) suggested, is not necessary to greatly reduce the severity

Table 2. Fruit yields for tomato cultivars of different Fusarium wilt resistance planted into field plots infested with race 1, 2, or 3.

	Fruit yield (% of noninfested plots)[z]					
	Fall crop			Spring crop		
Cultivar	R1	R2	R3	R1	R2	R3
Bonny Best	20	20	6	70	42	13
Manapal	67	66	68	93	88	90
Walter	102	81	62	99	91	90
Improved Rutgers	85	71	69	96	114	89
LSD (5%)		13			22	

[z]R1, R2, and R3 = race 1, race 2, and race 3 infested plots.

of Fusarium wilt of tomato and a successful second crop could be raised much sooner.

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Proc. Fla. State Hort. Soc. 102:360-362. 1989.

EVALUATION OF FUNGICIDES FOR CONTROL OF EARLY BLIGHT IN FLORIDA CELERY PRODUCTION

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Additional Index Words. *Cercospora apii, Apium graveolens,* foliar diseases.

Abstract. **Studies were conducted to evaluate the efficacy of several fungicides at various rates and combinations for controlling early blight of celery (*Apium graveolens* L. var. dulce) caused by *Cercospora apii*Fres. during fall 1987 and spring 1988. All fungicide treatments tested provided for significant reductions in early blight. However, marketable yield was not significantly increased over that present in untreated controls in all treatments. Several fungicide treatments, particularly a chlorothalonil/maneb/copper oxychloride premix and propiconazole alone and in combination with other fungicides provided excellent early blight control. Thiophanate-methyl, which breaks down to the same active ingredient as that in benomyl, was relatively ineffective. This performance may reflect the presence of a resistant strain of *C. apii*, since benomyl resistance has been recorded previously in Florida.**

Florida is the nation's second largest producer of celery (*Apium graveolens* L. var. dulce). Approximately 9,000 acres were planted during the 1987-88 growing season, producing a crop valued at over $48 million (4). Nearly 80 percent of this production is located on the organic soils of the Everglades Agricultural Area in south Florida. Early blight, caused by the fungus *Cercospora apii* fres., is the most important disease of celery in Florida (2) and yield losses of up to 100% in individual fields have been reported (7). Florida's warm temperatures, high relative humidity, and long dew periods combine to provide optimum environmental conditions for early blight development (1). Although resistance to this disease has been incorporated into several commercially available varieties (11,12), fungicides must still be applied on a regular basis to keep early blight under control. Many fungicides have been evaluated over the last several decades (5, 6, 9, 10), with mancozeb and chlorothalonil demonstrating the most consistent, efficacious early blight control. Although benzimidazole fungicides appeared promising initially, resistance to this class of fungicides by *C. apii* was soon reported (3).

A relatively new class of fungicides known as the ergosterol biosynthesis inhibitors has demonstrated activity against a broad spectrum of fungal pathogens, including *Cercospora* spp. (8). In addition, existing fungicides are continually undergoing modifications in chemistry and formulation. The objective of these experiments was to compare newly developed fungicides, currently registered fungicides, and fungicides at various rates and in combination for early blight control.

Materials and Methods

Two field experiments for evaluation of fungicides for early blight control were conducted at the Everglades Research and Education Center, Belle Glade. In experiment 1, the blight susceptible cultivar Florida 2-14 was transplanted on 20 Oct. 1987 and harvested on 10 Feb. 1988. Florida Slobolt, a moderately susceptible cultivar, was transplanted on 11 Mar. 1988 and harvested on 10 June in the second experiment. Both experiments were conducted on Pahokee muck with a soil pH of 6.1 and were fertilized according to soil test recommendations. Plants were spaced at 8 inch intervals with 2 ft row spacing. Prometryn (1.6 lb. ai/acre) and fluazifop (0.188 lb. ai/acre) were postplant applied for weed control and cyromazine (0.12 lb. ai/acre) was applied for leafminer control. Randomized complete block designs with 5 and 4 replications were used for experiments 1 and 2, respectively. Each treatment plot consisted of three 25 ft sprayed rows bordered by nonsprayed guard rows. Fungicide sprays were applied in

Florida Agricultural Experiment Station Journal Series No. N-00156.
The author wishes to thank A. Duda and Sons, Inc. and South Bay Growers, Inc. for their assistance in conducting these studies.