Induced Resistance to Fusarium Wilt of Watermelon Under Simulated Field Conditions

R. D. MARTYN, Associate Professor, C. L. BILES, Graduate Research Assistant, and E. A. DILLARD III, Technician II, Department of Plant Pathology and Microbiology, Texas A&M University, College Station 77843

ABSTRACT

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Race 2 of *Fusarium oxysporum* f. sp. *niveum* overcomes all current wilt-resistant watermelon cultivars. In previous studies, resistance to race 2 was induced in the greenhouse by prior inoculation with avirulent races (races 0 or 1) or with the cucumber wilt pathogen, *F. o.* f. sp. *cucumerinum*. In the present study, microplots were used to evaluate induced resistance under simulated field conditions. When *F. o. niveum* race 2 was at or near normal field levels (750 cfu/g of soil), *F. o. niveum* race 1 provided good protection of Calhoun Gray watermelons throughout the season. On plants induced by race 1, wilt symptoms were delayed by as much as 2 wk, 35% fewer plants died, and induced plants were healthier overall. When *F. o. cucumerinum* was used as an inducing agent, wilt symptoms also were delayed 2 wk, but, by the end of the season, disease severity was similar to the noninduced plants. When field populations of *F. o. niveum* race 2 were increased fivefold to 4,000 cfu/g of soil, *F. o. niveum* race 1 caused a delay in symptom expression; however, by the end of the season, there was no difference in the number of dead plants or disease severity between the induced and noninduced plants. Thus, it appears induced resistance can be overcome by a high level of challenge inoculum. Race 2 was recovered from the roots and lower stem of plants induced by race 1; therefore, induced resistance did not affect initial penetration of race 2 but did reduce the level of colonization and spread inside the vascular system.

Additional keywords: *Citrullus lanatus*, cross-protection

Watermelon (*Citrullus lanatus* (Thunb.) Matsum. & Nakai) is one of the most extensively planted summer fruit crops in the United States. Approximately 91,000 ha are planted commercially in the United States each year (1). Texas and Florida account for almost 50% of the total production area, each growing approximately 24,000 ha. Fusarium wilt, caused by the soilborne fungus *Fusarium oxysporum* Schlechtend.:Fr. f. sp. *niveum* (E. F. Sm.) W. C. Snyder & H. N. Hans., is one of the most serious diseases of watermelon and in many areas is a factor that limits production. The use of wilt-resistant cultivars has been the most effective means of controlling Fusarium wilt (9,11); however, a new race of the pathogen (*F. o. niveum* race 2) recently appeared that overcomes resistance in all currently grown cultivars (19,20,26).

In many cases, resistance can be induced in an otherwise susceptible plant by prior inoculation with an avirulent isolate, race, or nonspecific pathogen. Numerous reports document various degrees of protection with fungi, bacteria, and viruses (12,16,17,27). In most studies, induced resistance depends on the concentration of inducing inoculum, the age of the plant at the time of induction, temperature, and the length of time between induction and challenge. Several researchers have investigated the phenomenon of induced resistance (cross-protection) to the Fusarium vascular wilt pathogens (3,5,6,14,28,33). Fusarium wilt diseases appear to be ideal systems to study induced resistance because of the pathogen's ability to initially penetrate a wide range of plant species and still display a high degree of host specificity in causing disease.

Among the cucurbits, induced resistance has been demonstrated for Fusarium wilt of watermelon (*F. o. niveum*) (4,5,18, 21,30), muskmelon (*F. o.* f. sp. *melonis* W. C. Snyder & H. N. Hans.) (25), and cucumber (*F. o.* f. sp. *cucumerinum* J. H. Owen) (10,13). Most reports of induced resistance to the Fusarium vascular wilts are limited to laboratory and greenhouse experiments. Shimotsuma et al (30) reported that watermelons were protected from *F. o. niveum* in the field when induced with *Helminthosporium carbonum* Ullstrup, a corn pathogen; however, it is difficult to ascertain the degree of control from the data.

Previous work in our laboratory has shown that watermelons can be protected from a virulent race of *F. o. niveum* by prior inoculation with an avirulent race or with the cucumber wilt pathogen, *F. o. cucumerinum* (4,18). Additionally, we have shown that Fusarium wilt resistance in commercial cultivars can be overcome by high inoculum densities of the pathogen (24,32). The present study was initiated to evaluate induced resistance to race 2 under simulated field conditions. A preliminary report of this research has been published (21).

MATERIALS AND METHODS

Field plots. To simulate field conditions, 30 microplots (0.6 × 1.0 × 0.6 m) constructed of Wolmenized treated lumber containing a sandy loam soil (91% sand, 2% silt, 7% clay, and < 1% organic matter) to a depth of 60 cm were used. A graded French drain and gravel bed was incorporated underneath the entire microplot system at the time of construction in 1985. All microplots were arranged in rows and spaced 1 m apart on all sides. A 3- to 5-cm lip of each microplot remained above the soil level to help prevent cross-contamination by runoff and flooding. Microplots were fumigated at the beginning of each year with methyl bromide (98%) plus chloropicrin (2%) at 1 kg/10 m^2, under plastic (6 mil). The plastic was removed after 48 hr and the plots were aerated by turning the soil with a shovel weekly for 6 wk before infesting with the challenge inoculum. By the time the microplots were infested with the challenge inoculum, much of the normal soil microflora had reestablished.

Induction of plants. Watermelon cultivar Calhoun Gray, resistant to *F. o. niveum* races 0 and 1 but susceptible to race 2, was used throughout the studies. The same commercial seed lot was used both years. Transplants were grown in polystyrene foam trays (5-cm^2 cells) as previously described (4). The induction process was essentially the same as previously described (4). Plants were grown in a growth room for 18 days, and the emerging roots were gently washed and uniformly trimmed to 5 cm

Present address of second author: Department of Entomology, Plant Pathology, and Weed Science, New Mexico State University, Las Cruces 88003.

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in length without removing the plants from the cells. The polystyrene foam trays containing the plants were then placed in plastic trays (37 × 15 × 5 cm), each containing 250 ml of a microconidial suspension (10^6 per milliliter) of inducing inoculum or distilled water. Fusarium isolates used for induction included *F. o. niveum* race 1 (ATCC 18467) or *F. o. cucumerinum* (ATCC 16416). After 10 min, the plants were returned to their respective growing trays in the growth room for 3 days, after which they were transplanted into the microplots.

Challenge inoculation. Challenge inoculum consisted of an isolate of *F. o. niveum* race 2 (ATCC 62939) from Texas. Inoculum was grown in sand/cornmeal as previously described (22,31) and used to infest the microplots 2 wk before transplanting. Composite soil samples (eight 2.5-cm-diameter × 25-cm-deep cores per plot) were collected from each microplot 2 wk after infestation and assayed for the established densities of *F. oxysporum* on duplicate soil-dilution plates as previously described (22,31).

In 1988, 200 g of race 2-cornmeal inoculum was added to each microplot, resulting in a mean established population of 750 (±450) cfu/g of soil. This was within the range reported for naturally infested watermelon fields (26). In the 1988 test, two different inducing agents were used: *F. o. niveum* race 1, avirulent on Calhoun Gray, and *F. o. cucumerinum*, a nonpathogen of watermelon. Previous work (4) indicated that each organism induced resistance to race 2 under greenhouse conditions, but *F. o. niveum* race 1 offered a higher level of protection. Distilled water served as a noninduced control.

Three days after induction, four watermelon plants were transplanted to each *F. o. niveum* microplot infested with race 2 and to control microplots infested with *F. o. niveum* race 1, *F. o. cucumerinum*, or noninfested. There were five replicate microplots for each treatment. Plants were maintained insect-free and foliar pathogen-free by timely application of registered pesticides. All plots were fertilized with approximately 100 g of 14-14-14 (NPK) encapsulated Osmocote at transplanting and irrigated as needed until maturity (approximately 70 days).

The same microplots were used in the 1989 test; however, in 1989, only *F. o. niveum* race 1 was used as an inducer. All microplots were fumigated as in 1988 and reinfested with the race 2 challenge inoculum. Because Fusarium wilt resistance in watermelon is known to be affected by inoculum concentration (24, 32), the concentration of the race 2 challenge inoculum was increased fivefold to an average of 4,000 (±1,500) cfu/g of soil. All other parameters were the same as the 1988 test.

Disease assessment. Criteria used to assess disease and evaluate protection included 1) time of disease onset (earliness), 2) disease incidence, 3) disease severity, and 4) percent dead plants. Disease severity was based on the number of wilted or dead runners on a single plant and rated on 0–4 scale, where 0 = no runners showing symptoms, 1 = one runner wilted or dead, 2 = two runners wilted or dead, 3 = three runners wilted or dead, and 4 = any combination of four or more runners wilted or dead. Typically, watermelons have an average of four runners per plant. Initial disease assessments were made at appearance of the first symptoms and every 2 days thereafter for the remainder of the season.

Isolation of *F. oxysporum*. At the end of the 1988 field test, all plants from each treatment were collected and separated into root tissue and lower stem tissue. Ten sections from both root and lower stem tissue of each plant were surface-disinfested in 0.525% NaOCl for 5 min and transferred to two plates of Komada's *Fusarium*-selective medium (five sections per plate) (15) and incubated at 22 C for 72–120 hr. A few plants from the noninduced treatment and an occasional plant from the induced treatment were badly decomposed and no isolations were made.

The number of sections from which *F. oxysporum* grew on each plate was counted and the two plates were averaged for each tissue type and treatment. A treatment mean was then calculated for each treatment and tissue type. A subsample of these isolates (two or three from each treatment) was chosen and used to root-dip inoculate cucumbers (*Cucumis sativus* L. 'Comet A') and the three watermelon cultivars Black Diamond, Charleston Gray, and Calhoun Gray. Inoculation procedures were the same as those used previously (4). The purpose of these isolations and inoculations was to identify the specific isolates recovered and to determine if the protection induced by *F. o. niveum* race 1 was attributable to prevention of initial infection by race 2 or to limited vascular colonization by race 2 after infection.

RESULTS

1988 field test. In 1988, inoculation with *F. o. niveum* race 1 induced a delay

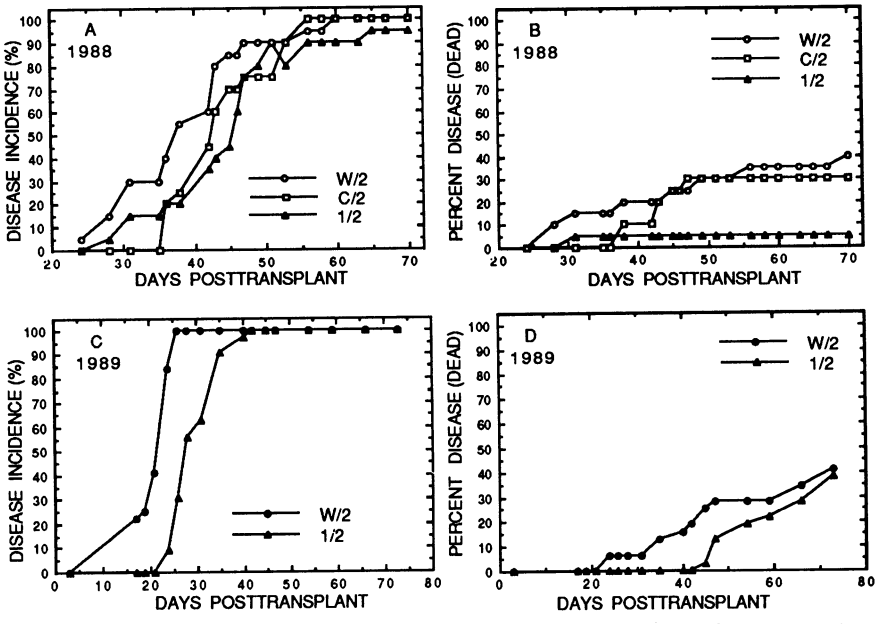

Fig. 1. Disease progression in microplots of induced and noninduced Calhoun Gray watermelons challenged with *Fusarium oxysporum* f. sp. *niveum* race 2 in 1988 and 1989. **(A)** Disease incidence from 1988 field test in which challenge inoculum averaged 750 cfu/g of soil, **(B)** plant death from 1988 test, **(C)** disease incidence in 1989 field test in which challenge inoculum averaged 4,000 cfu/g of soil, and **(D)** plant death from 1989 test.

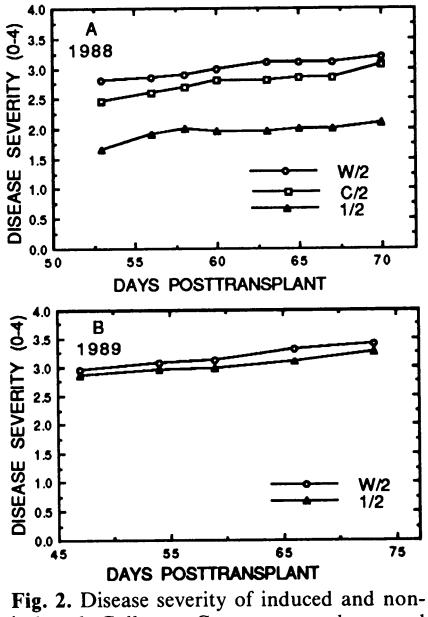

Fig. 2. Disease severity of induced and noninduced Calhoun Gray watermelons and challenged with *Fusarium oxysporum* f. sp. *niveum* race 2 in **(A)** 1988 (average concentration of race 2 in microplots was 750 cfu/g of soil) and **(B)** 1989 (average concentration of race 2 was 4,000 cfu/g of soil).

in symptom expression (Fig. 1A), a reduction in plant death (Fig. 1B), and a reduction in disease severity (Fig. 2A) when compared with the noninduced treatment. The first plants began to die 26 days after transplant in the noninduced treatment and plant death increased linearly throughout the season. By the end of the test, 40% of the noninduced Calhoun Gray watermelons were dead (Fig. 1B). In contrast, a few plants (5%) died 32 days after transplant in the treatment induced by race 1, but no more death occurred for the remainder of the season.

Disease severity in the noninduced plants was high, ranging from 2.5 by 52 days to 3.2 at the end of the season (Fig. 2A). No marketable fruits were produced on these vines. Disease severity in the plants induced by race 1 was lower, averaging 1.5 by day 52 and 2.0 at the end of the season (Fig. 2A). When *F. o. cucumerinum* was the inducer, a delay in symptom expression similar to that caused by *F. o. niveum* race 1 occurred, but, by 42 days, disease severity and plant death were similar to the noninduced treatment.

When disease incidence was used as the protection criterion, i.e., all plants showing symptoms regardless of the extent, the induced treatments resulted in a delay only in symptom expression, and by the end of the season there were no differences in disease incidence between treatments (Fig. 1A). There were, however, visual differences in the overall health of the plants. Plants in the microplots induced by race 1 had much more extensive and vigorous vine growth and fruit set. Typically, watermelons are planted in hills of one to two plants on 3-m centers, which allows for ample growing space. However, in the microplots, there were four plants per plot on 1-m centers and yield was reduced significantly in terms of number and size of the fruit because of excessive crowding. Therefore, yield data were not collected in this experiment. However, some market-quality fruit were produced in the plants induced by race 1, whereas no marketable fruit were produced on the noninduced plants.

1989 field test. Because *F. o. cucumerinum* did not induce a significant level of resistance in the 1988 field test, it was not included in the 1989 test. With the higher population level of race 2 used in 1987, wilt appeared at 15 days in the noninduced plants (Fig. 1C) and plants began to die at 20 days (Fig. 2D). All plants in the noninduced treatment were wilted by 25 days after transplant (Fig. 1C) and by 75 days, 42% of the plants were dead (Fig. 2D). In contrast, wilt symptoms did not appear in the plants induced by race 1 until 25 days, and plants did not begin to die until 45 days. However, by the end of the season, there were no differences in disease incidence (Fig. 1C), percent dead plants (Fig. 2D), or overall disease severity (Fig. 2B) between the induced and noninduced treatments. Visual differences in vine appearance and fruit set between treatments were evident, but these were not as dramatic as in the 1988 test; only a few marketable fruit were produced on induced plants.

Isolation of *F. oxysporum* from plants. *F. oxysporum* was recovered from each of the control treatments at low frequencies (Table 1). Based on pathogenicity results of the subsamples, all of these isolates were nonpathogenic except for approximately half of those recovered from the *F. o. niveum* race 1/water treatment. In this case, they were determined to be race 1. *F. o. cucumerinum* was not recovered. *F. oxysporum* was recovered at high frequencies from all root and stem tissues from each induction treatment (Table 1). Based on pathogenicity of the subsample from these isolations, *F. o. cucumerinum* was not recovered from either roots or stems, as none of the isolates were pathogenic on cucumber. The majority (approximately 80%) of the isolates of *F. o. niveum* recovered from the race 1 and race 2 treatments were pathogenic on all three watermelon differential cultivars and, therefore, were considered to be race 2. Race 1 was only occasionally recovered. Only isolates of race 2 were recovered from the noninduced water/race 2 treatment.

Table 1. Isolation of *Fusarium oxysporum* from Calhoun Gray watermelon root and stem tissue after induction inoculation and 11 wk after challenge inoculum

Induction/challenge treatment[y]	Tissue[z]	
	Root	Stem
Water/Water	1.1 a	0.1 a
FOC/Water	1.4 a	0.9 b
FON-1/Water	1.6 a	1.5 b
Water/FON-2	3.6 b	4.5 c
FOC/FON-2	3.3 b	3.6 c
FON-1/FON-2	2.9 b	4.9 c

[y] FOC = *Fusarium oxysporum* f. sp. *cucumerinum*; FON-1 = *F. o.* f. sp. *niveum* race 1; FON-2 = *F. o. niveum* race 2.

[z] Treatment mean of the number of sections out of five from which *F. oxysporum* was isolated. Data are from approximately 200 sections for each tissue and treatment. Numbers in a column followed by the same letter are not significantly different at the $P = 0.5$ level according to Duncan's multiple range test.

DISCUSSION

Although there are occasional contrary reports in the literature (30), results of most studies have shown that pathogens closely related to the challenge isolate (i.e., avirulent races, formae speciales, and different strains of a virus) are better inducers of resistance than those that are nonpathogens or pathogens of unrelated hosts.

Davis (5) reported that several different formae speciales of *F. oxysporum* were more effective in inducing resistance to a given host's pathogenic forma specialis than were other root pathogens (*Verticillium albo-atrum* Reinke & Bethier and *Rhizoctonia solani* Kühn) or nonpathogens (*Penicillium notatum* Westling and *Neurospora crassa* Shear et Dodge). In another study, Davis (6) found no differential ability among four formae speciales of *F. oxysporum* to induce resistance in tomato to *F. o.* f. sp. *lycopersici* (Sacc.) W. C. Snyder & H. N. Hans.; all offered partial but equal protection. However, Martyn (18), working with Fusarium wilt of watermelon, reported that two closely related formae speciales, *F. o. melonis* (muskmelon wilt) and *F. o. cucumerinum* (cucumber wilt), differentially protected watermelon from *F. o. niveum*. In his study, *F. o. cucumerinum* offered 100% protection against *F. o. niveum*, whereas *F. o. melonis* provided only slight protection.

Biles and Martyn (4) found that avirulent races of *F. o. niveum* were better inducers of resistance than related formae speciales and that there were additional differences between the avirulent races. Similar results were reported by Mas et al (25) with Fusarium wilt of muskmelon. In their study, avirulent races of *F. o. melonis* (race 0 or 1) were effective inducers of resistance to a virulent race (race 1 or 2). Results from the present field study support these general observations. An avirulent race of *F. o. niveum* induced a high level of resistance in watermelon to race 2, whereas a different cucurbit forma specialis (*F. o. cucumerinum*) did not.

Gessler and Kuć (10) reported that several formae speciales of *F. oxysporum* induced resistance in cucumber to *F. o. cucumerinum* in flask culture but not in a synthetic soil mix. In addition, inoculation of cucumber cotyledons with either *Colletotrichum lagenarium* (Passerim) Ellis et Halsted or tobacco necrosis virus also induced resistance to subsequent challenges by *F. o. cucumerinum*. Ishiba et al (13) and Biles and Martyn (4) demonstrated the opposite effect, i.e., induced resistance to a root pathogen (*F. o. cucumerinum* and *F. o. niveum*, respectively) also offered protection of the leaves from *C. lagenarium*.

Most reports of induced resistance to the Fusarium vascular wilts have been laboratory or greenhouse studies. In our study, induced resistance to race 2 of the watermelon wilt *Fusarium* was achieved under field conditions. *F. o. niveum* race 1 (avirulent on Calhoun Gray) provided protection against the virulent race 2 when the population of race 2 averaged 750 cfu/g of soil. *F. o. cucumerinum* was not an effective inducer of resistance in

the field in the 1 yr tested, even though it did induce resistance under greenhouse conditions (4). *F. o. cucumerinum* did, however, delay symptom development, and under some conditions, delayed symptom expression could result in reduced disease and increased yield.

An important result of the protection induced by race 1 was the reduction in overall disease severity. It is common in vine crops such as watermelon and muskmelon for one or two runners to wilt while the remaining two or three runners appear healthy and bear fruit. Therefore, a further reduction in the number of wilted runners attributable to induced resistance could result in better yields, even in the presence of some disease. This is demonstrated by comparing the disease incidence data in which all plants with symptoms were included with the disease severity data. There was no difference between the induced treatments and the noninduced control based on disease incidence, whereas there were differences in disease severity.

When the concentration of the challenge inoculum (race 2) was increased fivefold in 1989, induced resistance was not observed except for a delay in symptom expression. These results are similar to those reported by Martyn and McLaughlin (24) and Sumner (32) in which resistance to Fusarium wilt in commercial watermelon cultivars could be overcome by logarithmic increases in inoculum concentration. Douglas (8) reported similar results for Fusarium wilt of muskmelon.

Fusarium wilt diseases are affected by environmental parameters such as soil temperature and moisture; however, it is unlikely that these were responsible for the differences in the effectiveness of induced resistance from 1988 and 1989. Maximum and minimum air temperatures for May–July 1988 and 1989 were 32.3/19.2 C and 31.7/21.0 C, respectively. Similarly, there was no major difference in maximum and minimum soil temperature between the 2 yr (28.1/23.8 C and 25.9/24.8 C, respectively). There was a difference in rainfall over the 3 mo between years. In 1988, 20.3 cm of rain was received compared with 31.1 cm during the same 3 mo of 1989. It is doubtful, however, that this difference had any significant biological effect on disease development. Twenty centimeters of rain is generally adequate to grow watermelons, and in the cases when irrigation was needed, all microplots were equipped with an automatic drip irrigation system. Also, 31 cm of rain is not excessive for watermelon growth, and there was no flooding or standing water on any of the microplots because of the drainage system designed into the plots.

Race 2 was present within the vascular system of plants induced by race 1. This suggests that the induced resistance observed did not prevent initial infection by race 2 but may have limited its colonization. This is consistent with previous observations by Martyn (18) in which *F. o. niveum* could not be recovered from upper stem tissue when watermelons first were inoculated (induced) with *F. o. cucumerinum*. Similar results in reduced colonization of resistant hosts by formae speciales of *F. oxysporum* have been reported (2,7,23,29).

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