Local and Systemic Resistance Induced in Watermelons by Formae Speciales of *Fusarium oxysporum*

C. L. Biles and R. D. Martyn

Graduate research assistant and associate professor, Department of Plant Pathology and Microbiology, Texas A&M University, College Station 77843.
Present address of senior author: USDA, ARS, Appalachian Fruit Research Laboratory, Kearneysville, WV 25430.
Acknowledgment is given to James Eblin and E. H. Gehring for technical assistance throughout this project.
Accepted for publication 1 April 1989 (submitted for electronic processing).

ABSTRACT

Biles, C. L., and Martyn, R. D. 1989. Local and systemic resistance induced in watermelons by formae speciales of *Fusarium oxysporum*. Phytopathology 79:856-860.

Watermelon cultivars differentially resistant to Fusarium wilt were preinoculated (induced) with *Fusarium oxysporum* f. sp. *cucumerinum* or avirulent races of *F. o. niveum* 24 or 72 hr prior to challenge with a virulent race of *F. o. niveum*. All of the inducer treatments significantly reduced wilt symptoms ($P \leq 0.05$). Avirulent races of *F. o. niveum* induced a higher level of resistance than did *F. o. cucumerinum*. An interval of 24 hr between induction and challenge provided significant protection; a 72-hr interval further enhanced resistance. When roots of the wilt-susceptible watermelon cultivar Black Diamond were induced with *F. o. cucumerinum* and the leaves inoculated with *Colletotrichum lagenarium* 24 or 72 hr later, 50% fewer lesions developed on leaves of induced plants than on noninduced inoculated controls. This suggests that induced resistance to *F. o. niveum* is both local and systemic, as well as nonspecific.

Additional keywords: anthracnose, *Citrullus lanatus*.

There are several serious diseases of watermelon (*Citrullus lanatus* (Thunb.) Matsum. & Nakai) that reduce yield and quality (17). Of these, Fusarium wilt, caused by *Fusarium oxysporum* Schlect. emend. Snyd. & Hans. f. sp. *niveum* E. F. Sm., and anthracnose, caused by *Colletotrichum lagenarium* (Pass.) Ell. & Halst., have been a primary concern of watermelon breeders in regard to disease resistance (17). Because of the persistent nature of the wilt pathogen and the cultural practices employed in watermelon production, resistant varieties are the most economical and efficient method to control Fusarium wilt and anthracnose. However, as resistant cultivars are utilized, pathogen populations may change, and resistance be overcome by new virulent populations (races) of the pathogen. Therefore, breeders continually strive to develop germ plasm resistant to the developing races. Recently, a new race of *F. o. niveum*, designated race 2 (*F. o. niveum*-2) was identified (13,18), and, currently, there are no resistant cultivars available. Race 2 is now well established in Texas (13) and also occurs in Oklahoma (3) and Florida (14). Netzer and Dishon (19) tested 130 domestic cultivars of *C. lanatus* and accessions of *Citrullus* spp., a wild species from Israel (*C. colocynthis*), and two wild species from Africa (*C. rehmi* and *C. ecirrhosus*) for resistance to race 2, but resistance was not found. In an effort to develop an alternative control method for Fusarium wilt, the biological control strategy of induced resistance was tested in our laboratory. Induced resistance in plants has been shown by several workers to be a common response to nonpathogenic bacteria, viruses, and fungi (11). Induced resistance is accomplished by the inoculation of a plant with an avirulent or nonpathogenic isolate prior to or concomitant with a challenge inoculation with a pathogen.

Effective inducers of resistance are often closely related physiologically and taxonomically to the challenge isolate. Working with Fusarium wilts of tomato, cabbage, flax, carnation, and watermelon, Davis (5) observed that formae speciales of *F. oxysporum* were more effective inducers than other fungi. Mas et al (15) investigated the effect of different races of *F. o. melonis* on muskmelon and observed that inoculation with a mixture of compatible and incompatible races resulted in a resistant response in a susceptible cultivar. They concluded that it was the incompatible race that triggered the resistant response. Martyn (12) inoculated watermelon with *F. o. melonis* and *F. o. cucumerinum* and concluded that *F. o. cucumerinum* provided a higher level of protection against *F. o. niveum*, implying that some formae speciales of *F. oxysporum* may be more effective inducers than others.

The protection of cucumber from anthracnose (caused by *C. lagenarium*) by prior inoculation with an avirulent isolate of *F. o. cucumerinum* was demonstrated by Ishiba et al (9). They concluded that the resistant response to anthracnose was systemic. *F. o. cucumerinum* was detected primarily in the root and hypocotyl, but not at the leaf infection site or in the stem. Histological work by Martyn (12) with watermelon supports this conclusion.

The purpose of this research was to investigate the ability of an avirulent race (race 0) of *F. o. niveum* (*F. o. niveum*-0) and an incompatible pathogen (*F. o. cucumerinum*) to induce resistance in watermelon to the virulent *F. o. niveum*-2; to determine the time between induction and challenge that gives the highest level of protection; and to test whether the resistance response is systemic and nonspecific, by using *C. lagenarium* as a challenge inoculum on the leaves of watermelons after their roots had received an induction treatment. Portions of this work have been previously reported (1,2).

MATERIALS AND METHODS

Cultivars. The watermelon cultivars used were Black Diamond, Calhoun Gray, and Dixielee. Black Diamond is susceptible to all three races of *F. o. niveum*. Calhoun Gray and Dixielee both are resistant to race 0 and race 1 (*F. o. niveum*-1) but susceptible to race 2 (13). Preliminary experiments also included Jubilee and Royal Jubilee. Jubilee is moderately resistant to race 0 but susceptible to races 1 and 2. Royal Jubilee is resistant to races 0 and 1 but susceptible to race 2.

Cultures. Isolates of *Fusarium* used throughout the study were *F. o. niveum*-0, isolate 60-3A(11) (originally obtained from J. M. Crall, University of Florida, Leesburg); *F. o. niveum*-1, ATCC 18467; *F. o. niveum*-2, ATCC 62940; and *F. o. cucumerinum*, ATCC 16416. *C. lagenarium* was obtained from Joe Kuć, University of Kentucky, Lexington.

All isolates of *Fusarium* were increased from single-spored cultures and stored in sterile soil tubes (16) at room temperature. Active cultures were obtained by placing a small aliquot of soil culture in a 250-ml flask containing 50 ml of liquid mineral salts broth (7) and incubating the flask at 22 C on a rotary shaker at 110 rpm. After 3–4 days, the fungal slurry was strained through eight layers of sterile cheesecloth, and the predominantly microconidial suspension was calibrated with a hemacytometer.

Plant growth. Seeds of each watermelon cultivar were obtained from commercial sources and individually planted in 5×5 cm^2 cells in polystyrene foam trays (Speedling brand), having 12 cells per tray, with 1×1 cm^2 bottom holes for drainage and root emergence. Sunshine Potting Mix #1 was used as a growth medium. The trays were then placed inside a larger flat containing a mixture of vermiculite and peat (1:1, v/v) to allow for root growth. Plants in greenhouse experiments were grown with available daylight and at ambient temperatures and irrigated with distilled water as needed. Watermelons for the experiments on systemic induced resistance were grown in similar flats and soil as previously described but maintained in a growth room (25–30 C) with fluorescent lights (860 $\mu E \cdot m^{-2} \cdot sec^{-1}$) with a 16-hr photoperiod.

Induction and challenge inoculations. The plants were grown for approximately 25 days, and emerging roots gently washed and trimmed without removing the plants from the cells. The polystyrene foam trays were then placed in plastic trays ($37 \times 15 \times 5$ cm), each containing 250 ml of a microconidial suspension (10^6 conidia per milliliter) of inducing inoculum—either *F. o. cucumerinum, F. o. niveum*-0, or *F. o. niveum*-1—or 250 ml of water. After 10 min, the plants were returned to their respective growing trays. They were challenged either 24 or 72 hr later with *F. o. niveum*-2 by the same root-dip method but without additional trimming or washing of the roots. Five greenhouse experiments were conducted to determine the induction efficiency of a 24-hr interval between induction and challenge. Each experiment was a randomized split-plot design, three-way factorial, with three cultivars (Black Diamond, Calhoun Gray, and Dixielee) and five treatments (Table 1). Disease progression was monitored for 35 days. Nine to 12 observations of the treatments within each experiment were made. One experiment was conducted in which the challenge inoculum was applied 72 hr after induction.

The five greenhouse experiments with a 24-hr interval between induction and challenge were combined, and each experiment was considered a replication. Disease ratings were taken every 2 days, but only data from days 15, 20, 25, 30, and 35 were used in the statistical analysis. Data expressed as percentages were transformed to the arc sines of their square roots. Duncan's multiple comparison test and least significant difference (LSD) test were used to distinguish between treatments and between cultivars (20).

Testing for systemic induction. To determine if resistance to *F. o. niveum* induced by *F. o. cucumerinum* is systemic, Black Diamond watermelon plants were inoculated with *F. o. cucumerinum* as previously described, and the second and third true leaves were challenged with *C. lagenarium*. In the initial experiment, 150 μl of a conidial suspension of *C. lagenarium* (10^5 conidia per milliliter) was applied as a droplet to the leaf surface 3 days after inoculation of the roots with *F. o. cucumerinum* and 2 days after challenge with *F. o. niveum*-2 as described by Dean and Kuć (6). In further experiments, only *F. o. cucumerinum* was used to induce resistance to *C. lagenarium*, and there was no subsequent wilt challenge with *F. o. niveum*-2. In these tests, filter disks (6 mm in diameter) were saturated with a conidial suspension of *C. lagenarium* (5×10^6 conidia per milliliter), and 10 disks were applied to each leaf. The plants were enclosed in humidity chambers at ambient room temperature for 24 hr in the dark, and afterwards the chambers were opened gradually to allow for equilibration. The plants were returned to the growth room, and lesions were counted 5 days later.

Each plant was considered a replication, and each experiment had eight to 10 plants per treatment. Each experiment was a complete randomized block, with one cultivar and three treatments: water followed by *C. lagenarium, F. o. cucumerinum* followed by *C. lagenarium*, and *F. o. niveum*-2 followed by *C. lagenarium*. A 24- or 72-hr interim was allowed between induction and challenge. The experiments with a 72-hr interim were repeated three times, but only one experiment was conducted with a 24-hr interim. The general linear model of the Statistical Analysis System was used to analyze variance within each experiment. Duncan's and Fisher's LSD tests were used to determine differences between treatments.

TABLE 1. Induction and challenge treatments in greenhouse experiments on induced resistance to *Fusarium oxysporum* f. sp. *niveum* race 2

Trt. no.	Inducer treatment	Challenge treatment
1	Water	Water
2	*F. o. cucumerinum*	*F. o. niveum* race 2
3	*F. o. niveum* race 0	*F. o. niveum* race 2
4	*F. o. niveum* race 1	*F. o. niveum* race 2
5	Water	*F. o. niveum* race 2

RESULTS

Experiments conducted in both the greenhouse and the growth chamber indicated that prior inoculation with the avirulent *F. o. niveum*-0 protected several different watermelon cultivars from the highly aggressive *F. o. niveum*-2 (Table 2). Similarly, prior inoculation with *F. o. cucumerinum* protected Black Diamond from *F. o. niveum*-2, compared with the noninducing treatment (29 vs. 71% wilt). Prior inoculation with the avirulent *F. o. niveum*-0 resulted in 83, 45, and 41% less wilt in three experiments with Calhoun Gray, 40 and 67% less wilt in Dixielee, and 53% less wilt in Royal Jubilee. The cultivar Jubilee had 46% less disease when induced with *F. o. niveum*-0 than it had in the treatment with water followed by *F. o. niveum*-2. *F. o. niveum*-0 did not cause wilt in the resistant cultivars Royal Jubilee, Calhoun Gray, and Dixielee. Figure 1 shows the reaction of Calhoun Gray plants in one experiment.

Disease progression was followed for 35 days in six greenhouse experiments. There was a significant difference ($P \leq 0.01$) in final disease incidence in the five experiments in which the time between induction and challenge was 24 hr. The induction/challenge treatment with *F. o. cucumerinum*/*F. o. niveum*-2 resulted in significantly less disease in the cultivar Black Diamond than the treatment with water/*F. o. niveum*-2 (30 and 93%, respectively). The highest level of disease in Black Diamond occurred in the treatment with *F. o. niveum*-1/*F. o. niveum*-2 (Table 3), but it was not significantly higher than that in the treatment with *F. o. niveum*-0/*F. o. niveum*-2. Both of these treatments caused

TABLE 2. Percentage of dead plants in greenhouse experiments on induced resistance to *Fusarium oxysporum* f. sp. *niveum* race 2

Induction/challenge treatment	Cultivar[x,y]				
	BD	J	RJ	CG	DL
Water/water	0	0	0	0	0
F. o. niveum race 0/ *F. o. niveum* race 0	—[z]	30	—	0	0
F. o. niveum race 0/ water	—	50	0	0	0
F. o. cucumerinum/ water	0	—	—	—	—
F. o. cucumerinum/ *F. o. niveum* race 2	29	—	—	—	—
F. o. niveum race 0/ *F. o. niveum* race 2	—	54	25	16	0
Water/ *F. o. niveum* race 2	71	100	78	57	40

[x]Twelve to 20 plants per treatment were tested.
[y]BD = Black Diamond; J = Jubilee; RJ = Royal Jubilee; CG = Calhoun Gray; DL = Dixielee.
[z]No treatment was applied.

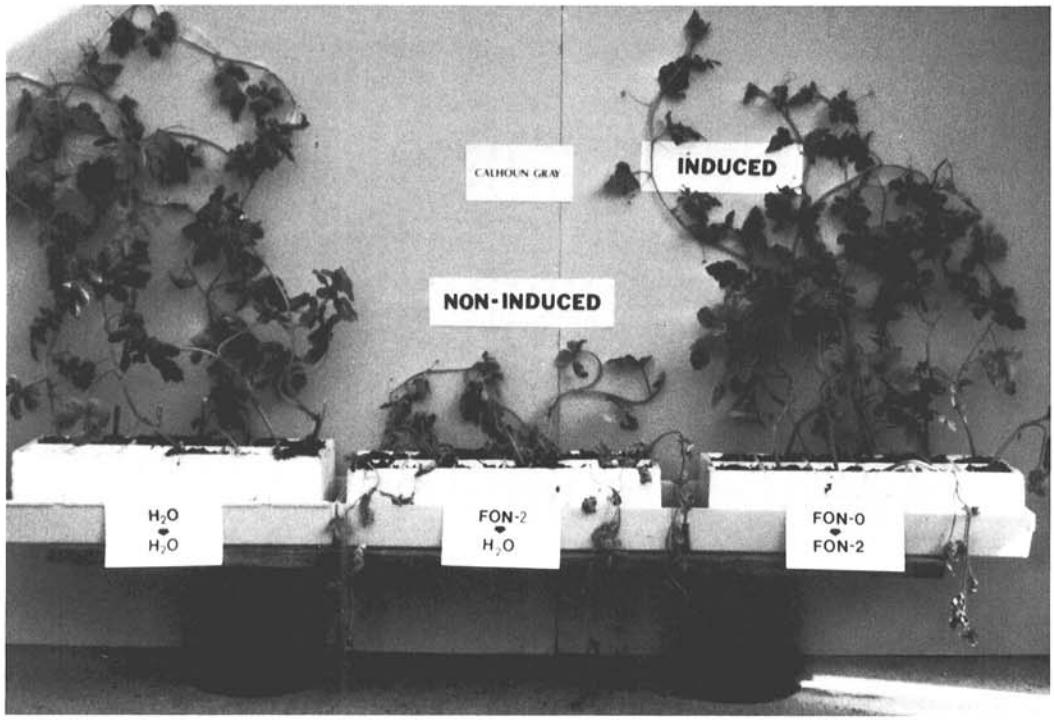

Fig. 1. A plant of the watermelon cultivar Calhoun Gray induced with *Fusarium oxysporum* f. sp. *niveum* race 0 (FON-0) and challenged 24 hr later with *F. o. niveum* race 2 (FON-2) (right), compared with a control plant treated with water (left) and a noninduced control plant challenged with *F. o. niveum* race 2 (center).

TABLE 3. Fusarium wilt incidence in watermelon cultivars after roots were induced with various treatments and challenged 24 hr later with *Fusarium oxysporum* f. sp. *niveum* race 2

	Induction/challenge treatment[y,z]				
Cultivar	W/W	C/2	0/2	1/2	W/2
Black Diamond	0 d	0.30 c	1.28 a	1.45 a	0.93 b
Calhoun Gray	0 b	0.26 b	0.28 b	0.22 b	0.65 a
Dixielee	0.01 b	0.22 b	0.15 b	0.07 b	0.65 a

[y] W = water; C = *F. o. cucumerinum*; 0 = *F. o. niveum* race 0; 1 = *F. o. niveum* race 1; 2 = *F. o. niveum* race 2. The values are arc sines ($\times 10^{-2}$) of the square roots of percentages.

[z] Numbers in the same row followed by the same letter are not significantly different ($P = 0.05$).

significantly higher levels of disease in Black Diamond than did the treatment with water/*F. o. niveum*-2.

Calhoun Gray and Dixielee were protected ($P \leq 0.05$) from *F. o. niveum*-2 by induction by either *F. o. cucumerinum, F. o. niveum*-0, or *F. o. niveum*-1. Disease incidence in Calhoun Gray treated with *F. o. cucumerinum*/*F. o. niveum*-2, *F. o. niveum*-0/*F. o. niveum*-2, *F. o. niveum*-1/*F. o. niveum*-2, and water/*F. o. niveum*-2 was 14, 15, 10, and 45%, respectively. Disease incidence in Dixielee with these treatments was 20, 10, 3, and 50%, respectively (Table 3). There were also differences between treatments and between cultivars with respect to the rate of disease development. In all cases, prior inoculation of a cultivar with one or more avirulent isolates resulted in slower disease progression (Fig. 2). In Black Diamond, 50% wilt was achieved within 15–20 days after treatment with the virulent race but no prior induction. However, when Black Diamond was first induced with *F. o. cucumerinum* and then challenged with *F. o. niveum*-2, 50% wilt did not occur during the duration of the experiment (40 days). Similarly, with induction treatments, 50% wilt was not achieved in Dixielee and Calhoun Gray during the course of the experiment (40 days). The induction of resistance by an avirulent pathogen reduced both the rate of disease progression and the final disease incidence.

Similar trends were observed when there was a 72-hr lag period between induction and challenge (Fig. 3). *F. o. cucumerinum* protected Calhoun Gray and Dixielee at approximately the same level at 72 hr as it did at 24 hr. In contrast, *F. o. niveum*-0 and *F. o. niveum*-1 were more effective inducers when a 72-hr interim was used. No wilt symptoms occurred in Calhoun Gray throughout the course of the experiment when it received a prior inoculation with *F. o. niveum*-0 or *F. o. niveum*-1.

Systemic induction. In the first experiment on systemic induction, a conidial suspension of *C. lagenarium* (10^5 conidia per milliliter) was applied directly to the leaves 72 hr after root treatment with either water, *F. o. cucumerinum*/*F. o. niveum*-2 (with a 24-hr interim between the root inoculations), or water/*F. o. niveum*-2. The plants treated with *F. o. cucumerinum*/*F. o. niveum*-2 had the fewest anthracnose leaf lesions, with a mean of 2.3 lesions per leaf. The plants treated with water had a mean of 5.9 lesions per leaf, and those treated with *F. o. niveum*-2 had a mean of 8.8 per leaf. Wilt symptoms were observed 14 days after inoculation in 10 and 91% of the plants, respectively, in the treatments with *F. o. cucumerinum*/*F. o. niveum*-2 and with water/*F. o. niveum*-2.

In the second and third experiments, filter disks were used to apply *C. lagenarium* to the leaves. In experiment 2, there were significant differences between the induction treatment with *F. o. cucumerinum* and the treatments with water and with *F. o. niveum*-2 when a 72-hr interim was imposed between induction and challenge with *C. lagenarium*. Mean lesion counts for the root treatments with water, *F. o. cucumerinum*, and *F. o. niveum*-2 were 5.2, 2.9, and 5.5 lesions per leaf, respectively. In experiment 3, only 24 hr was allowed between root treatment and leaf challenge. Mean lesion counts for the treatments with water, *F. o. cucumerinum*, and *F. o. niveum*-2 were 4.2, 3.5, and 6.7 lesions per leaf, respectively. Significantly more lesions occurred in the root treatment with *F. o. niveum*-2 than in the other two treatments. Lesion counts in the treatments with water and with *F. o. cucumerinum* were not significantly different, although the mean trend was similar to that in experiments using a 72-hr interim between induction and challenge. Each experiment using the root induction treatments and *C. lagenarium* on the leaves resulted in fewer lesions on plants whose roots were previously inoculated with *F. o. cucumerinum*. Plants whose roots were previously inoculated with *F. o. niveum*-2 had the highest number of lesions

caused by *C. lagenarium*. Although quantitative measures were not taken, differences between the induced and the noninduced plants with respect to lesion size were observed.

DISCUSSION

Induced resistance has been reported for several crops treated with several different pathogens and inoculation methods (4,11).

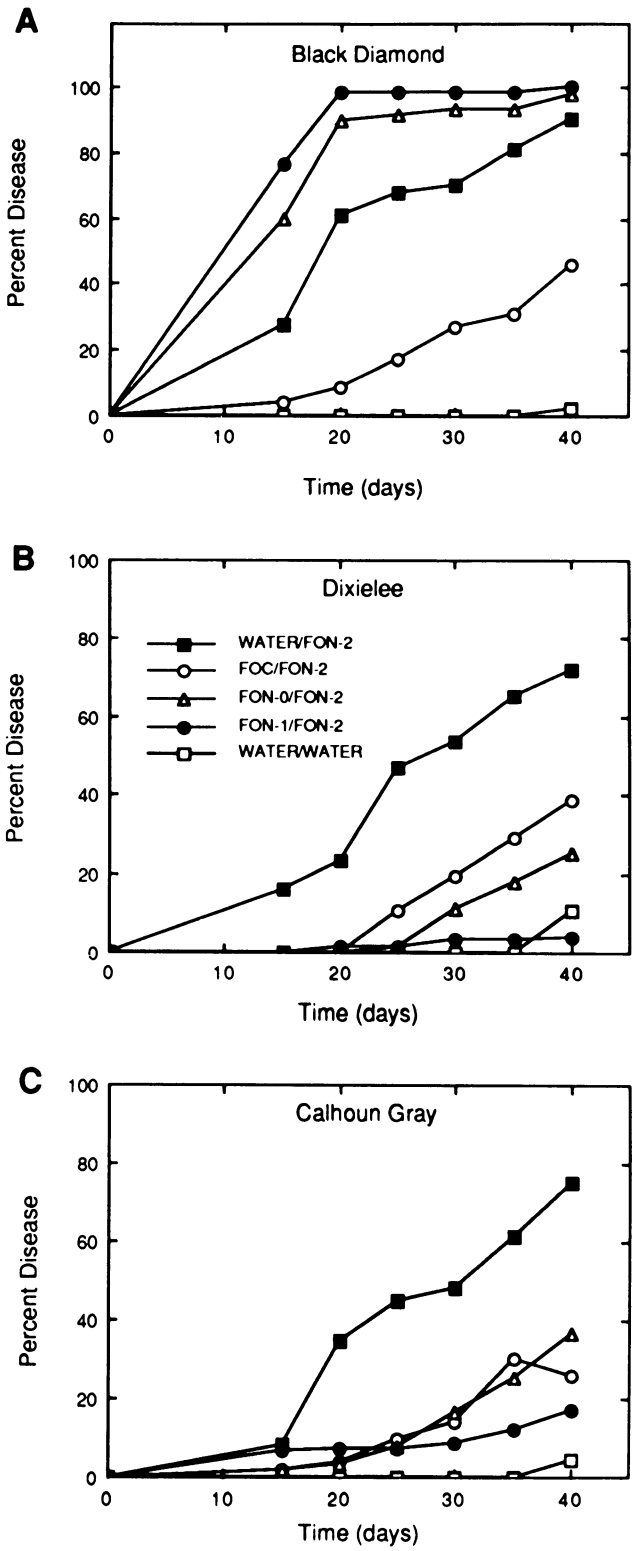

Fig. 2. Fusarium wilt progression in the watermelon cultivars **A,** Black Diamond, **B,** Dixielee, and **C,** Calhoun Gray after induction with either *Fusarium oxysporum* f. sp. *cucumerinum* (FOC), *F. o. niveum* race 0 (FON-0), or *F. o. niveum* race 1 (FON-1) and challenged 24 hr later with *F. o. niveum* race 2 (FON-2).

Inoculation methods and plant growth media affect the inducibility of the plant. Gessler and Kuć (8) observed that the inoculation of cucumber roots to induce resistance to *F. o. cucumerinum* was more effective in an agar medium than in a greenhouse potting mix. They also observed that 72 hr between induction and challenge was necessary for adequate protection from a virulent race of *F. o. cucumerinum* and that resistance was both localized and systemic. The inoculation of roots with microconidia of *F. o. cucumerinum* (10^4 microconidia per milliliter) protected a distant root from challenge 3 days later. The inoculation of leaves with *C. lagenarium, Cladosporium cucumerinum*, or tobacco necrosis virus protected roots from a virulent race of *F. o. cucumerinum* (11). The inoculation of cucumber roots with a virulent race of *F. o. cucumerinum* did not protect leaves from *C. lagenarium* but, instead, resulted in wilt (8). In the present study, 24 hr between induction and challenge provided significant local protection in plants grown in a greenhouse potting mix. With *F. o. cucumerinum*, the level of protection attained was approximately the same when either a 24- or a 72-hr interim was used, whereas *F. o. niveum*-0 and *F. o. niveum*-1 protected best when a 72-hr interim was used. Davis (5) induced roots of several vegetable crops and found that the time interval between induction and challenge did not affect the induction process. Wymore and Baker (21) observed that cross-protection of tomato with *F. o. dianthi* was only

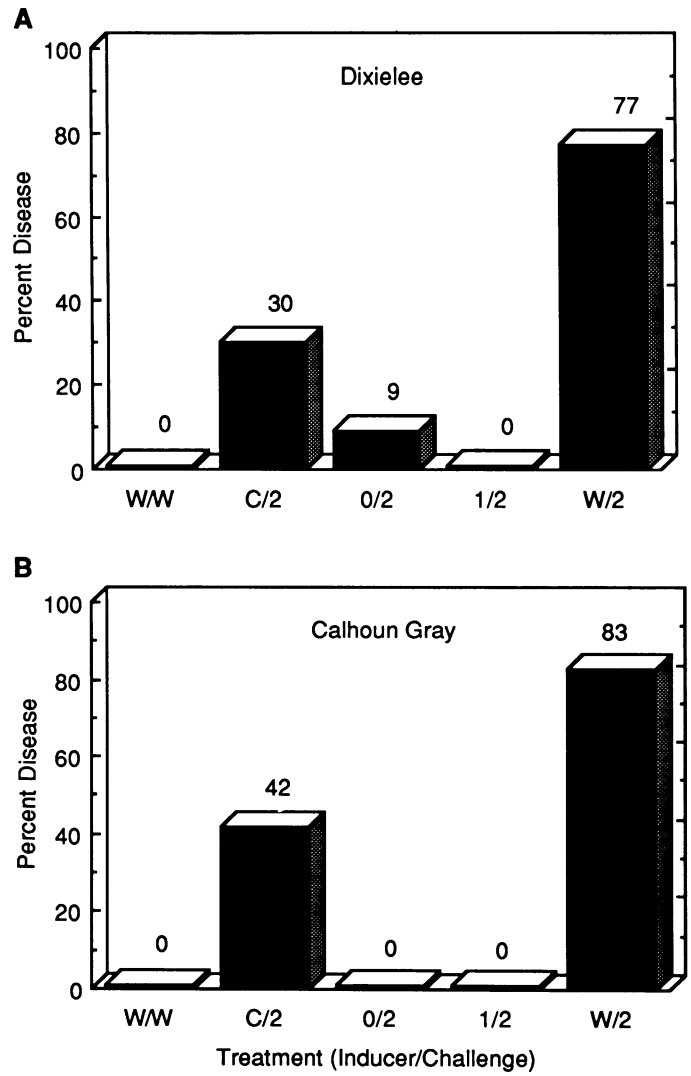

Fig. 3. Fusarium wilt incidence in the cultivars **A,** Dixielee and **B,** Calhoun Gray induced and challenged 72 hr later. The inducing treatments were water (W), *Fusarium oxysporum* f. sp. *cucumerinum* (C), *F. o. niveum* race 0 (0), and *F. o. niveum* race 1 (1). The challenge inoculum was *F. o. niveum* race 2 (2). The data reflect the percent disease incidence 30 days after induction.

effective a few days after induction, but that this protection was ephemeral. They concluded that induction is dependent on the isolate and the concentration of inoculum. Inoculum concentration had to be equal to or greater than that of the challenge inoculum. Our results suggest that avirulent races of *F. o. niveum* provide a higher level of protection of watermelon against the virulent race 2 of *F. o. niveum* than the nonpathogenic *F. o. cucumerinum* does. A response may be triggered by the closely related avirulent races of *F. o. niveum* that is not induced by *F. o. cucumerinum*. A lag period may allow the induction signal to move in greater quantity from the induction site to the rest of the root.

Systemic induction of resistance has been achieved by inoculating cucumber roots with an avirulent *F. o. cucumerinum* to protect the leaves from a later challenge with *C. lagenarium* (9). We observed a similar phenomenon in watermelon, even though the techniques used were different. Ishiba et al (9) cut the roots off at the hypocotyl and then soaked the hypocotyl in a conidial suspension of *F. o. cucumerinum* for 2 hr. They concluded that the induction of systemic resistance occurred from hypocotyl-root to leaf. The present study, conducted with watermelon, confirms these earlier results with cucumber and implies that similar mechanisms of induction occur within the cucurbits. Systemic induction in watermelon was most evident when the plants were challenged 72 hr after induction, implying that resistance mechanisms may be stimulated from a translocated elictor of root origin. Dean and Kuć (6) girdled leaf petioles induced with an avirulent *C. lagenarium* to observe the effect on leaves later challenged with the pathogen. They found that the longer the induced leaf remained ungirdled, the higher the level of protection in other plant parts. These experiments indicated that there was a "signal" produced in the induced tissue and that it was translocated to other plant tissues. Translocation of the signal from root to leaf was demonstrated by grafting scions of cucumber, watermelon, and muskmelon onto an induced cucumber rootstock (10). Grafting other scions onto induced rootstocks suggested that the transmissible signal was not cultivar-, genus-, or species-specific (11). This same type of signal may be active in the watermelon-*Fusarium* induction system.

The best inducer of resistance to *F. o. niveum*-2 for Calhoun Gray and Dixielee was *F. o. niveum*-1. The resistance observed was not only local but systemic and nonspecific, in that induced watermelon plants were protected from both Fusarium wilt and anthracnose. The techniques developed in this study to induce plants could easily be incorporated into commercial greenhouse operations.

LITERATURE CITED

1. Biles, C. L. 1988. An isozyme analysis of the induced resistance response to Fusarium wilt of watermelon incited by incompatible vascular wilt fusaria. Ph.D. thesis, Texas A&M University, College Station. 156 pp.
2. Biles, C. L., and Martyn, R. D. 1988. Local and systemic resistance in watermelon induced with *Fusarium oxysporum* ff. sp. (Abstr.) Phytopathology 78:625.
3. Bruton, B. D., Patterson, C. L., and Martyn, R. D. 1988. Fusarium wilt (*F. oxysporum* f. sp. *niveum* race 2) of watermelon in Oklahoma. Plant Dis. 72:734.
4. Cramer, C. L., Ryder, T. B., Bell, J. N., and Lamb, C. J. 1985. Rapid switching of plant gene expression induced by fungal elicitors. Science 227:1240-1243.
5. Davis, D. 1967. Cross-protection in Fusarium wilt diseases. Phytopathology 57:311-314.
6. Dean, R. A., and Kuć, J. 1986. Induced systemic protection in cucumber: Time of production and movement of the signal. Phytopathology 76:966-970.
7. Esposito, R., and Fletcher, A. M. 1961. The relationship of pteridine biosynthesis to the action of copper 8-hydroxy-quinolate on fungal spores. Arch. Biochem. Biophys. 93:369-376.
8. Gessler, C., and Kuć, J. 1982. Induction of resistance to Fusarium wilt in cucumber by root and foliar pathogens. Phytopathology 72:1439-1441.
9. Ishiba, C., Tani, T., and Murata, M. 1981. Protection of cucumber against anthracnose by a hypovirulent stain of *Fusarium oxysporum* f. sp. *cucumerinum*. Ann. Phytopathol. Soc. Jpn. 47:352-359.
10. Jenns, A. E., and Kuć, J. 1979. Graft transmission of systemic resistance of cucumber to anthracnose induced by *Colletotrichum lagenarium* and tobacco necrosis virus. Phytopathology 69:753-756.
11. Kuć, J. 1982. Induced immunity to plant disease. Bioscience 32:854-860.
12. Martyn, R. D. 1985. Differential cross protection of watermelon to Fusarium wilt by related formae speciales. (Abstr.) Phytopathology 75:1304.
13. Martyn, R. D. 1987. *Fusarium oxysporum* f. sp. *niveum* race 2: A highly aggressive race new to the United States. Plant Dis. 71:233-236.
14. Martyn, R. D., and Bruton, B. D. 1989. An initial survey of *Fusarium oxysporum* f. sp. *niveum* races in the United States. HortScience 24. (In press)
15. Mas, P., Molot, P. M., and Riser, G. 1981. Fusarium wilt of muskmelon. Pages 169-177 in: *Fusarium*: Diseases, Biology, and Taxonomy. P. E. Nelson, T. A. Toussoun, and R. J. Cook, eds. Pennsylvania State University Press, University Park. 457 pp.
16. McKeen, C. D., and Wensley, R. N. 1961. Longevity of *Fusarium oxysporum* in soil culture. Science 134:1528-1529.
17. Mohr, H. C. 1986. Watermelon breeding. Pages 37-66 in: Breeding Vegetable Crops. M. J. Bassett, ed. AVI Publishing, Westport, CT.
18. Netzer, D. 1976. Physiological races and soil population level of Fusarium wilt of watermelon. Phytoparasitica 4:131-136.
19. Netzer, D., and Dishon, I. 1973. Screening for resistance and physiological specialization of *Fusarium oxysporum* in watermelon and muskmelon. (Abstr. 941) Int. Congr. Plant Pathol. 2nd.
20. Ott, L. 1984. An Introduction to Statistical Methods and Data Analysis. PWS, Boston. 775 pp.
21. Wymore, L. A., and Baker, R. 1982. Factors affecting cross protection in control of Fusarium wilt of tomato. Plant Dis. 66:908-910.