Induction of Resistance Towards Bacterial Pathogens of Tomato by Exposure of the Host to Dinitroaniline Herbicides

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ABSTRACT

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Growth of tomato seedlings (cv. Bonny Best) in rooting substrate amended with the dinitroaniline herbicide, dinitramine (1 ppm in sand/Pro-mix, 1:1 or 1:2, v/v), caused a suppression in bacterial wilt symptoms caused by the *Pseudomonas solanacearum* strain K60. Dinitramine was not inhibitory to *P. solanacearum* in in vitro assays nor did it restrict multiplication of the pathogen in treated plants. Dinitramine also caused a decrease in the severity of bacterial canker, caused by *Clavibacter michiganense* subsp. *michiganense* JD83-1, but had no significant effect on the severity of bacterial speck, caused by *Pseudomonas syringae* pv. *tomato* DC894H. Resistance was not induced by a similar exposure of plants to the herbicides ethalfluralin or oryzalin. Resistance to Fusarium wilt, caused by *Fusarium oxysporum* f. sp. *lycopersici*, was induced by exposure of seedlings to the herbicides dinitramine, ethalfluralin, and oryzalin. Acetochlor induced resistance towards Fusarium wilt but did not towards bacterial wilt. Exposure of plants to the dinitroaniline herbicide, trifluralin, did not induce resistance towards either bacterial wilt or Fusarium wilt in our experiments.

Herbicides, growth retardants, and other agrochemicals frequently alter the interaction between pathogens and their hosts (5,9,11). Although the mechanism of action remains to be clarified, the increased resistance of plants to fungal wilt diseases after exposure to dinitroaniline and chloroacetimide herbicides has been well documented (3–5,7,9,10). Protection conferred by exposure of plants to dinitroaniline herbicides does not appear to be related to any direct fungitoxic effect of the herbicides on the pathogens (11), but such effects have not been completely ruled out. More likely, the increase in disease resistance is related to changes in host metabolism that alter the normal host-parasite interaction (5,10). Enhanced resistance to *Fusarium* sp. in tomato and cotton plants exposed to the herbicide trifluralin, for instance, was associated with the induced production of antifungal compounds (10). In contrast, resistance to Fusarium wilt disease in melon seedlings treated with dinitroaniline herbicides was related, in part, to a reduced production of ethylene after infection (4). Paclobutrazol treatment of melon seedlings inhibited gibberelic acid synthesis, possibly contributing to resistance (5). In tomato, exposure to dinitroaniline herbicides at concentrations of less than 1 μg per gram of soil is capable of inducing an increase in fungal wilt resistance (9,10).

The induction of resistance after exposure of the host to dinitroanilines has been primarily studied in diseases caused by fungi. Whether such herbicidal compounds are capable of inducing host resistance towards bacterial diseases has not been determined. In the present study, we examined the effect of several dinitroaniline herbicides and acetochlor for the induction of resistance in tomato towards bacterial wilt (*Pseudomonas solanacearum*), bacterial canker (*Clavibacter michiganense* subsp. *michiganense*), and the foliar form of bacterial speck disease (*Pseudomonas syringae* pv. *tomato*). The ability of these compounds to induce resistance towards Fusarium wilt (*Fusarium oxysporum* f. sp. *lycopersici*) of tomato also was determined to confirm and contrast their effectiveness against fungal and bacterial diseases.

MATERIALS AND METHODS

Pathogens. Strain K60, a tobacco strain of the bacterial wilt pathogen, *P. solanacearum*, was obtained from A. Kelman, North Carolina State University. It was grown on triphenyl tetrazolium chloride (TTC) agar (12) or casamino acid-peptone-glucose (CPG) agar. The strain was stored in sterile distilled water at room temperature.

A spontaneous nalidixic acid-resistant (Nal^r) derivative of strain K60, K60.1, was obtained by spreading 0.1 ml of a water suspension (5×10^8 colony forming units (cfu)/ml) of strain K60 onto TTC agar amended with 25 μg/ml of nalidixic acid. Nal^r colonies appeared after 72 h of incubation at 32 C. The mutant appeared, otherwise, identical to the wild type in pathogenicity, and in physiological and morphological characteristics.

C. m. michiganense JD83-1 was obtained from J. Dick, Nabisco Inc., Dresden, Ontario. It was propagated on nutrient broth-yeast extract (NBY) agar (13) at 25 C.

P. s. tomato DC894H was isolated from diseased tomatoes grown near Harrow, Ontario, in 1989. It was maintained on NBY agar at 25 C and stored at −73 C in NBY broth containing 15% glycerol.

F. o. lycopersici, race 0, was originally obtained from Dr. Z. A. Patrick, University of Toronto, Toronto, Ontario. The fungus was maintained on yeast extract agar (7.5 g of yeast extract, 20 g of dextrose, 15 g of agar in 1 L water) and was periodically reisolated from inoculated tomato plants to ensure that virulence was maintained.

Chemicals. The following dinitroaniline herbicides were tested: dinitramine (Cobex; N,N-diethyl-2,6-dinitro-4-trifluoromethylphenyldiamine; Tapa Zaol Chemical Co., Israel), ethalfluralin (Sonalan; N-ethyl-N-(2-methyl-2-propenyl)-2,6-dinitro-4-(trifluoromethyl)benzenamine; DowElanco, Sarnia, Ontario), oryzalin (Surflan; 3,5-dinitro-N^4,N^4-dipropylsulfanilamide; DowElanco, Sarnia, Ontario), and trifluralin (Treflan; α,α,α-trifluoro-2,6-dinitro-N,N-dipropyl-p-toluidine; DowElanco, Sarnia, Ontario). The chloracetamide herbicide, acetochlor (Harness; 2-chloro-N-(ethoxymethyl)-6′-ethyl-o-acetotoluidine; Monsanto Corp., Mississauga, Ontario) was also tested.

Dinitroaniline stock solutions were prepared at a concentration of 2,000 ppm a.i. A stock solution of acetochlor was prepared containing 500 ppm a.i. One-milliliter aliquots of the stock solutions were then diluted in 100-ml vol of water, and the diluted solutions were thoroughly mixed into 1-kg lots of silica sand.

Seeds were allowed to germinate in a rooting substrate mixed with the chemical-amended sand preparations. The rooting substrate, "Pro-mix" BX (New Rochelle, NY), was mixed with the amended sand in the proportions 2:1 (Pro-mix/sand, v/v), unless otherwise noted. Control substrates were produced by mixing Pro-mix with nonamended sand. The resulting substrates were placed in 128-cell transplant plug trays (Landmark Plastic Corp., Akron, OH). Each cell was 3.2 cm^2 and 5.1 cm deep. Two tomato seeds (*Lycopersicon esculentum* Mill. 'Bonny Best') were sown in each cell, and plants were thinned-out to one per cell as they emerged. Developing plants were fertilized once per week with "Plant Pro" 20:20:20 fertilizer solution (Plant Products Ltd., Brampton, Ontario). Plants were harvested 2–4 days after emergence. Adhering rooting substrate was washed off with water. After inoculation, the plants were transferred to 12.7 cm diameter pots and incubated in a growth chamber (Conviron Ltd., Winnipeg, Manitoba) at 31 C, under a 14-h light (140 $\mu E \cdot m^{-2} \cdot s^{-1}$), 10-h dark (24 C) cycle.

Plant inoculation procedure and rooting substrate. Experiments were performed to examine the effect of various inoculation procedures and the rooting substrate type used during incubation on the severity of bacterial wilt disease in nontreated and chemically treated seedlings.

Dinitramine-treated and control plants were either inoculated with a suspension of *P. solanacearum* K60 by one of three procedures: 1) dipping plant roots in a bacterial suspension, 2) drenching the bacterial suspension onto freshly cut root surfaces, and 3) injection of inoculum (10 μl) directly into the lower stem. The concentration of bacteria in the inoculum was 1×10^8 or 5×10^8 cfu/ml for the first procedure and 5×10^8 cfu/ml for the latter two procedures. Dinitramine was tested at two concentrations in the rooting substrate: 1:1 and 1:2 amended sand/Pro-mix (v/v). Occurrence of wilt symptoms and plant death were periodically monitored. Experiments were performed using a completely randomized design.

Effect of chemical treatment on the severity of bacterial wilt and bacterial canker. Tomato seedlings were uprooted from the herbicide-amended substrate after their emergence and, after being rinsed with water, the roots were inoculated by dipping them in a bacterial suspension (5×10^8 cfu/ml) of *P. solanacearum* K60 or *C. m. michiganense* JD83-1.

For bacterial wilt, the number of wilted seedlings was recorded daily, and the percentage of diseased plants was determined. For bacterial canker, disease severity was rated on an arbitrary scale of 0–3, in which 0 = no visible symptoms, 1 = slight wilting, 2 = severe wilting, and 3 = plant killed. Each experiment consisted of five replicates of eight or 10 seedlings each. The experiment was repeated once.

Enumeration of *P. solanacearum* in inoculated tomato stems. Hypocotyl tissue from dinitramine-treated and nontreated plants was harvested and weighed into lots of 0.5 g each. The tissue was suspended in 15 ml of sterile 10 mM potassium phosphate (pH 7.2) buffer, homogenized in a Sorvall Omnimixer (2×90 s), and the suspension was serially diluted. Aliquots were plated onto triplicate plates of TCC-Nal agar and incubated at 32 C. Number of colonies of K60.1 per plate was determined after 3 days and related to the amount of tissue present in the aliquot. The experiment was repeated once.

Toxicity of dinitramine to *P. solanacearum*. The effect of dinitramine on *P. solanacearum* K60 was determined by adding the herbicide to filter paper disks and placing the disks on CPG agar inoculated with 0.1 ml of a 5×10^8-cfu/ml bacterial suspension. Each disk received 50 μl of 0, 1, 10, 50, 100, or 1,000 ppm solutions of dinitramine. Triplicate plates for each concentration were incubated for three days at 32 C and then examined for the presence or absence of zones of bacterial growth inhibition around the disks.

Effect of inoculum concentration on the incidence of bacterial wilt in dinitramine-treated tomato seedlings. Nine days after germination, dinitramine-treated (2 μg/g of dinitramine-amended sand/Pro-mix, 1:2, v/v) and control seedlings were root-dip inoculated with one of the following concentrations of *P. solanacearum* K60: 3.1×10^6, 3.1×10^7, 6.5×10^7, 3.1×10^8, or 3.1×10^9 cfu/ml. Each concentration was tested on five replicates of eight or 10 plants per replicate using a completely randomized design. Plants were incubated for disease development, and the percentage of mortality was determined over time.

Effect of plant age on Fusarium wilt and bacterial wilt expression in dinitramine- or acetachlor-treated tomato plants. Plants germinated in sand/Pro-mix (1:1, v/v) mixture containing dinitramine, acetochlor, or no herbicide were inoculated with *P. solanacearum* or *F. o. lycopersici* at either 4 days (young plants) or 13 days (old plants) after emergence. The experiment was performed as a split-split plot design (age at inoculation = mainplots; pathogen = subplots; dinitramine concentration = sub-subplots). There were five replicates of eight plants per replicate per treatment. Percentage of plant mortality was determined at day 8 post-inoculation for the *P. solanacearum*-inoculated plants, and at 15 days post-inoculation for the *F. o. lycopersici*-inoculated plants. The data were subjected to analysis of variance.

Effect of dinitramine treatment on the severity of bacterial speck of tomato. Sixteen 3-wk-old tomato seedlings were removed from dinitramine-treated rooting substrate and planted into either nontreated sand or rooting substrate. Sixteen plants that had not been exposed to the herbicide served as controls. The leaves of all 32 plants were inoculated with *P. s. tomato* DC89-4H by a procedure that has already been described (6). Plants were examined daily for the development of foliar lesions.

Statistical analysis. Statistical analysis was performed using PC-SAS (SAS Institute, Cary, NC) on a Compaq 386 personal computer.

RESULTS

Plant inoculation procedure and rooting substrate. Inoculation by root wounding or root dipping resulted in 90–100% mortality in nonherbicide-treated plants. In contrast, only 62.5% of the plants died with direct injection (Table 1). Exposure to dinitramine-amended substrate during germination resulted in plant mortality of 20–40% with root-wounding or root-dipping inoculation procedures, and only 12.5% with both herbicide concentrations when the bacteria were injected into the stem (Table 1). The direct root-dip procedure was selected for all subsequent studies, because of its ease of use and effectiveness in inducing disease.

TABLE 1. Effect of inoculation procedure and dinitramine treatment on mortality of tomato from bacterial wilt disease caused by *Pseudomonas solanacearum* K60

Dinitramine treatment (2 ppm)	Percentage of plant mortality[a]			
	Root dip[b] inoculum concentration (cfu/ml)		Root wounding[b]	Stem injection
	1×10^8	5×10^8		
Sand/Pro-mix 1:2, v/v	20	40	20	12.5
Sand/Pro-mix 1:1, v/v	30	20	...[c]	12.5
None	90	100	100	62.5

[a] Percentage of plant mortality was determined 10 days after inoculation. Each value is the mean of eight or 10 replicate plants.
[b] Inoculation by either root dipping or root wounding caused significantly more mortality 10 days after inoculation (orthogonal contrast) than did stem injection.
[c] Not determined.

In experiments on the effect of rooting substrate type on disease incidence, approximately 90% of the plants grown in either herbicide-free sand or Pro-mix were wilted 6 days after stem inoculation with *P. solanacearum* (results not shown). In contrast, 37 and 12% of plants germinated in herbicide-treated medium and transplanted after inoculation to Pro-mix or sand, respectively, developed disease symptoms. Sand was selected as the incubation medium for the remaining experiments because of the severe disease that developed when it was used.

Effect of chemical treatment on the severity of bacterial wilt, Fusarium wilt, and bacterial canker diseases of tomato. Dinitramine prevented symptom development and plant mortality after inoculation with *P. solanacearum* (Fig. 1A). Regression analysis of slopes and intercepts, however, indicated that trifluralin, ethalfluralin, and oryzalin were ineffective in reducing disease (Fig. 1A) compared to the nontreated control. Plant mortality in these treatments reached 75–90% 7 days after inoculation.

Ethalfluralin and oryzalin conferred complete protection from wilting from *F. o. lycopersici*. Trifluralin, however, failed to significantly alter plant mortality when compared to nontreated plants (Fig. 1B).

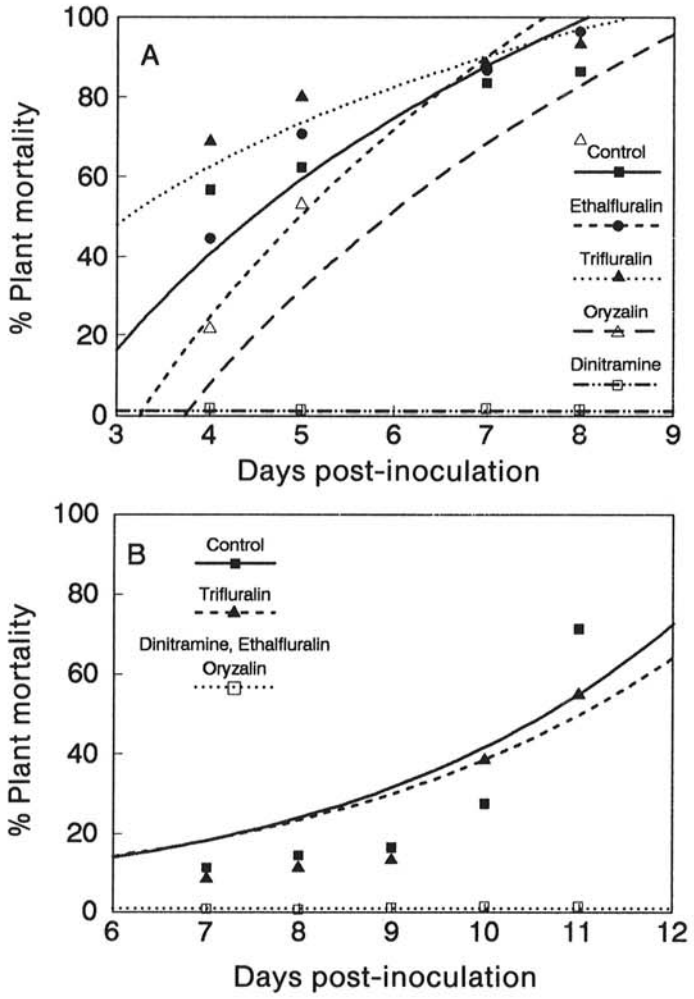

Fig. 1. Effect of selected herbicides on the mortality in tomato cv. Bonny Best inoculated with either *Pseudomonas solanacearum* K60 or *Fusarium oxysporum* f. sp. *lycopersici*. **A,** plants inoculated with *P. solanacearum* K60. Only the regression line for the dinitramine treatment is significantly different from the nontreated control. Regression relationships are of the form $y = a + e^{(-\text{Day})}$. r^2 values for control, ethalfluralin-, trifluralin-, and oryzalin-treated inoculated plants are 0.38, 0.60, 0.31, and 0.35, respectively. Regression lines are based on the observation of 40 plants per treatment. **B,** plants inoculated with *F. o. lycopersici*. No mortality was encountered after dinitramine, ethalfluralin, or oryzalin treatment. Regression lines for the nontreated control and trifluralin-treated plants were not significantly different and are of the form $y = a + e^{(\text{Day})}$, with r^2 values of 0.51 and 0.62, respectively.

Dinitramine-treated tomato plants, inoculated at either 2, 13, or 29 days after emergence with *C. m. michiganense*, showed less severe disease development compared to controls (Fig. 2). Inoculation of seedlings 2 or 13 days after emergence resulted in more severe disease in both the control and treated plants compared to plants that were inoculated at 29 days after emergence. Disease severity gradually increased through time with all treatments. By 21 days after inoculation, there was no difference in either dinitramine-treated or nontreated plants that had been inoculated with canker 2 days after their emergence. Dinitramine-treated plants inoculated 13 days after emergence appeared less diseased than the comparable controls at the end of the experiment (Fig. 2).

Toxicity of dinitramine to *P. solanacearum*. After 3 days of incubation at 32 C, the bacteria grew well on all plates. Zones of inhibition did not appear around any of the herbicide-impregnated disks. Colonization of dinitramine-treated tomato seedlings by the nalidixic acid-resistant mutant of *P. solanacearum*, K60.1. Numbers of bacteria recovered from the dinitramine-treated plants did not differ significantly ($P = 0.05$) from those recovered from nontreated control plants, being approximately

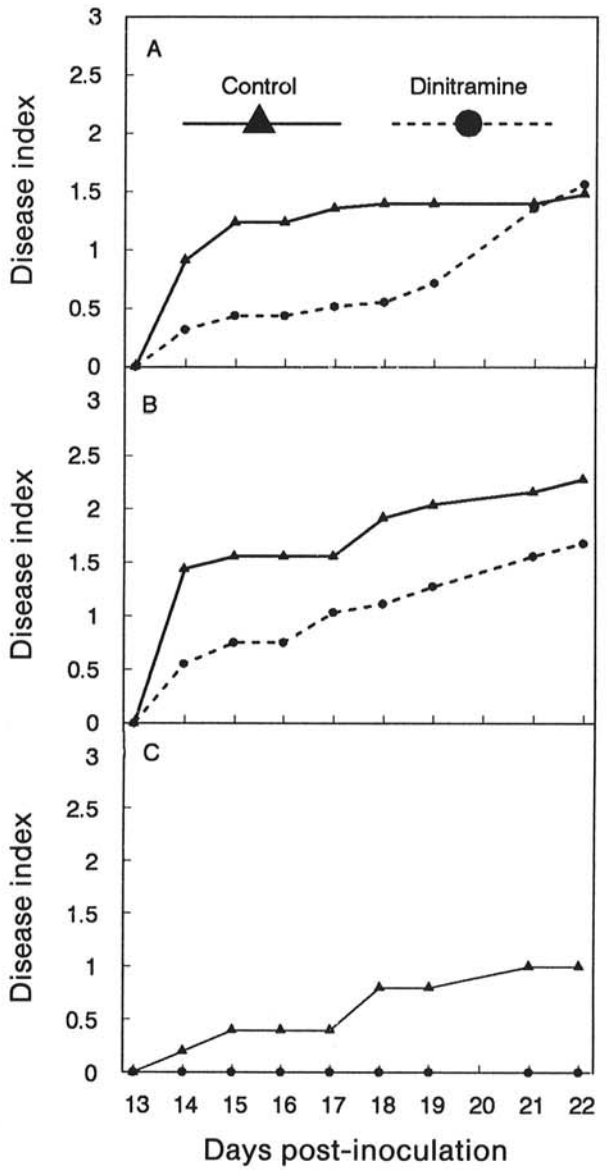

Fig. 2. Effect of dinitramine on the severity of bacterial canker disease of tomato. **A,** plants inoculated 2 days after emergence. **B,** plants inoculated 13 days after emergence. **C,** plants inoculated 29 days after emergence. Markers indicate the mean index obtained from 25 plants per treatment. Ratings were based on an arbitrary scale with 0 = no visible symptoms, 1 = slight wilting, 2 = severe wilting, and 3 = plant killed.

4.0×10^7 and 5×10^7 cfu/g of stem tissue, respectively.

Effect of inoculum concentration on the incidence of bacterial wilt in dinitramine-treated tomato seedlings. Exposure to dinitramine completely prevented mortality when tomato plants were inoculated with *P. solanacearum*, regardless of the inoculum concentration (Fig. 3). In nontreated, inoculated plants, mortality followed a linear relationship from day 4 (the first time at which disease was detectable) to day 8 post-inoculation, and the slopes for the relationships were not significantly different for the various concentrations that were tested. The relationship for the nontreated control plants inoculated with the highest concentration of *P. solanacearum* tested, 3.1×10^9 cfu/ml, however, had a significantly greater intercept than found for the other inoculum concentrations that were tested. This led to a disease incidence of about 80% within 4 days after inoculation at the highest inoculum concentration (Fig. 3). Plants exposed to lower numbers of bacteria reached similar levels of disease at about 6–8 days after inoculation.

Effect of plant age on Fusarium and bacterial wilt expression in dinitramine- or acetochlor-treated tomato plants. Older plants were significantly more susceptible to either pathogen ($P = 0.0054$) than were young plants (Table 2). Also, inoculation with *P. solanacearum* caused significantly more plant mortality ($P = 0.0019$) than did inoculation with *F. o. lycopersici*, regardless of the plant age at inoculation or chemical treatment (Table 2).

Dinitramine was effective in protecting inoculated plants (Table 2) against either pathogen. Dinitramine exposure provided complete protection against *F. o. lycopersici*, and caused a significant reduction in plant mortality from *P. solanacearum*.

Treatment with acetochlor did not significantly reduce mortality compared to the nontreated control for either young or old plants, when they were inoculated with *P. solanacearum*. Acetochlor did cause a significant reduction ($P < 0.05$) in the mortality of young plants inoculated with *F. o. lycopersici*, relative to the control (Table 2). It was ineffective, however, in protecting old plants when inoculated with this pathogen.

Effect of dinitramine on the incidence of bacterial speck disease in tomato seedlings. In contrast to the results obtained with the soilborne pathogens *P. solanacearum* and *C. m. michiganense*, dinitramine had no effect on symptom expression caused by the leaf-spotting pathogen, *P. s. tomato* DC89-4H. Five days after inoculation, all dinitramine-treated and control plants exhibited numerous foliar lesions typical of bacterial speck.

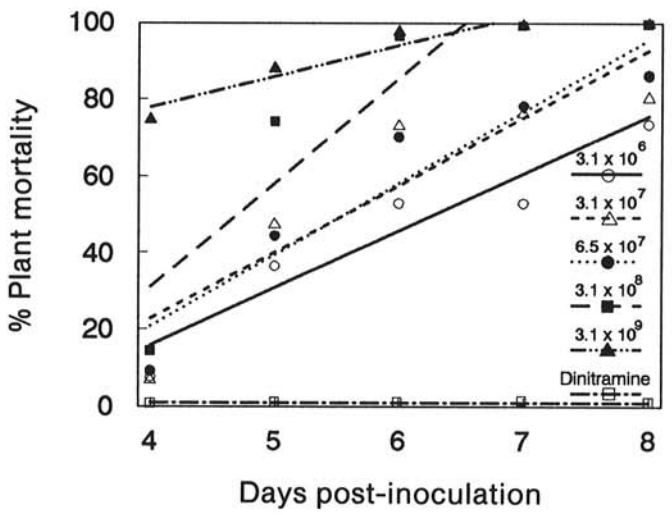

Fig. 3. Effect of inoculum concentration of *Pseudomonas solanacearum* K60 on plant mortality in dinitramine-treated and nontreated tomato plants. Regression lines are of the form $y = a + b$(day). Lines for the nontreated control series have r^2 values of 0.39, 0.58, 0.51, 0.76, and 0.27, for 3.1×10^6, 3.1×10^7, 6.5×10^7, 3.1×10^8, and 3.1×10^9 cfu/ml of inoculum, respectively. Lines were based on observations of 40–50 plants per treatment. No mortality was found at any inoculum concentration after dinitramine treatment.

DISCUSSION

The capacity of dinitroaniline herbicides to protect plants from vascular diseases caused by fungi such as *Verticillium* and *Fusarium* has been clearly demonstrated in previous studies (4,5,7,9,10). In the present study, we established that exposure of tomato plants to dinitramine also provides excellent protection against the wilt-inducing bacterial pathogen, *P. solanacearum*. Our results suggest that this protection was not due to an inhibition of bacterial growth and multiplication. The results, however, do not eliminate the possibility that dinitramine may have had a direct effect on *P. solanacearum*, such as suppressing the biosynthesis of some unknown pathogenicity factor.

Dinitramine appears to suppress the symptoms of bacterial wilt. A similar situation was observed previously by Cohen et al (4) with dinitramine-treated melon plants inoculated with *Fusarium oxysporum* f. sp. *melonis*. Dinitramine-treated plants remained symptomless after inoculation but were found to show the same degree of vascular colonization as nontreated, wilted plants. In contrast to our results with *P. solanacearum*, dinitroanilines (i.e., trifluralin and nitralin) restricted colonization of root tissue and the development of a systemic infection by *F. o. lycopersici* (10).

Efficacy of chemically induced protection against bacterial wilt was influenced by a number of factors, including method of inoculation, duration of treatment, plant age at inoculation, and type of chemical used. For instance, plants transferred from dinitramine-amended rooting substrate into sand after inoculation were found to be better protected from bacterial wilt than were plants transferred into Pro-mix. These results may have been from a more rapid degradation or dilution of the herbicide within Pro-mix-grown plants. Varying the inoculum concentration did not, however, alter the expression of dinitramine-induced resistance, possibly because the infection court may have become saturated with the pathogen at relatively low inoculum concentrations.

Although dinitramine, ethalfluralin, oryzalin, and acetochlor suppressed Fusarium wilt symptoms, only dinitramine prevented disease expression in *P. solanacearum*-inoculated plants. Whether these chemicals have different modes of action is not yet known. The effect of acetochlor on *F. o. lycopersici*-inoculated plants decreased when the length of time between plant exposure to the herbicide and inoculation with the pathogen was extended. Acetochlor is a chloracetamide, and previous studies have shown that these compounds are rapidly detoxified in plant tissues by conjugation with glutathione (2,3,8). Dinitroanilines such as dinitramine, however, appear to be quite persistent in plant tissues (1) and thus could provide long-lasting protection against pathogens.

Treatment with the dinitroaniline herbicide trifluralin (1 μg a.i./g of natural sandy loam soil) also suppresses the development of Fusarium wilt symptoms in tomato (9,10). Grinstein et al (10) reported that ethanol-soluble phytoalexins were produced in trifluralin-treated tomato cvs. Marmande and Rehovot 13 after inoculation with the fungus. However, in our study, this herbicide failed to protect Bonny Best from Fusarium wilt. These contrasting

TABLE 2. Effect of plant age and herbicide treatment on the mortality of tomato plants inoculated with either *Fusarium oxysporum* f. sp. *lycopersici* or *Pseudomonas solanacearum* K60

	Percentage of plant mortality[a]			
	F.o. lycopersici		*P. solanacearum*	
Herbicide treatment	Old[b]	Young	Old	Young
Control	86.6	72.0	100	83.3
Acetochlor	79.8	20.7	100	72.7
Dinitramine	0	0	39.6	17.3

[a] Mortality for *F.o. lycopersici*-inoculated plants was determined 15 days after inoculation and at 8 days after inoculation for *P. solanacearum*. Each value is the mean of 40 plants per treatment. LSD 5% for herbicide treatment means within pathogen and plant age types = 24.26 and to compare any two means = 30.49.

[b] Plants were inoculated 4 days (young) or 13 days (old) after emergence from the herbicide-amended or nontreated rooting substrates.

results suggest that plant protection by this herbicide may depend on the particular tomato cultivar and fungal isolate studied. In a study on the effect of dinitroaniline herbicides on various *Rhizoctonia*-, *Fusarium*-, and *Verticillium*-caused diseases, Grinstein et al (9) concluded that protection is dependent on the specific plant-herbicide pathogen combination tested. Our results with dinitramine and the three bacterial pathogens of tomato, *P. solanacearum*, *P. s. tomato*, and *C. m. michiganense* support this conclusion.

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