Effect of the Herbicide Diphenamid on Damping-off Disease of Pepper and Tomato

J. Katan and Y. Eshel

Department of Plant Pathology and Microbiology; and Department of Field and Vegetable Crops, The Hebrew University of Jerusalem, Faculty of Agriculture, Rehovot, Israel.

This research was partially supported by the Ministry of Agriculture, Israel.

The authors gratefully acknowledge the technical assistance of S. Tsuriel, A. Grinstein, and S. Ilani.

Accepted for publication 5 April 1974.

ABSTRACT

Rhizoctonia solani was the major causal organism for damping-off in directly seeded pepper (*Capsicum frutescens*) fields. The effect of the herbicide diphenamid on the incidence of this disease was tested by sowing peppers in soil inoculated with *R. solani* at various inoculum concns. With each tested isolate, diphenamid increased Rhizoctonia damping-off disease at least at one inoculum concn. This disease increase was not observed, however, in separate experiments carried out with *Pythium*. Results were variable for tomatoes inoculated with *R. solani*. The possible mechanisms involved in the increase in Rhizoctonia disease of pepper were studied by analyzing the effects of the herbicide on the pathogen, the host, and soil microorganisms. Diphenamid is slightly toxic to the pathogen and does not stimulate its growth in vitro, its respiration and glucose utilization in sterile soil, or its virulence to pepper. Pepper seedling resistance to *R. solani* was not affected by pretreatment with diphenamid. The herbicide enhanced colonization of bean stem segments by *R. solani* in natural soil, and suppressed soil microorganism respiration and glucose utilization in glucose-amended soil. Diphenamid also slowed down the decrease in time of *R. solani* colonization and disease incidence. It is suggested that suppression of soil microorganisms contributed to the disease increase in pepper. In contrast with pepper, pretreatment with diphenamid increased tomato seedling resistance to *R. solani*.

Phytopathology 64:1186-1192

Additional key words: Lycopersicon esculentum, biological equilibrium.

Herbicides are commonly used for weed control. As with other pesticides, their biological effects often also extend to nontarget organisms in the environment. These unintended effects may lead to the stimulation or inhibition of activities of nontarget organisms. Many herbicides were found to increase incidence of various diseases, e.g. Rhizoctonia damping-off of cotton, sugarbeets, and other crops (1, 2, 8, 12).

Diphenamid is a common herbicide used for preemergence selective weed control in solanaceous crops. Directly seeded pepper (*Capsicum frutescens* L.) for industrial processing was recently introduced in Israel and fields are commonly treated with diphenamid. Under these conditions a full stand of the crop is essential. It was suspected, however, that diphenamid reduced stand in direct-seeded pepper fields, especially in soils where damping-off pathogens occur.

A shift in disease incidence due to pesticide application is the result of the sum of the positive or negative effects of the herbicide, or its degradation products upon the pathogen, the host, and the surrounding microorganisms. Katan and Eshel (8) suggested four possible mechanisms through which a pesticide may increase or decrease a disease: (i) the effect on pathogen growth or reproduction; (ii) the effect on the pathogen virulence; (iii) the effect on host susceptibility; and (iv) the effect on relationships with other microorganisms.

This study deals with the effect of diphenamid on the incidence of damping-off diseases of pepper, and to a lesser extent, to tomato. The mechanisms involved in the observed increase in the incidence of the disease caused by *Rhizoctonia* in pepper were also studied. A brief report of these results has been published (7).

MATERIALS AND METHODS.—Unless otherwise stated, a loamy sand soil [82.3% sand, 2.3% silt, 15.4% clay, and 0.45% organic matter; pH, 7.4; moisture-holding capacity (MHC), 12.2%] was used. Seedlings grown in this soil were free of damping-off diseases. Colonization, respiration, and glucose utilization experiments were carried out with soil adjusted to 50% MHC and incubated at 27 C. A wettable powder formulation of the herbicide diphenamid (*N, N*-dimethyl-2,2-diphenylacetamid) was applied to the soil either by mixing to a final concn of 10 μg/g, or by spraying on the soil surface at a dosage equivalent to 10 kg/ha. Pepper and tomato cultivars used were Vindale and Rehovot-13, respectively. In direct-seeding experiments, 16 seeds were sown in a plastic pot (10 cm in diam), in six replicates. In transplanting experiments, 20 seedlings were planted in a plastic box (13 × 13 cm), in six replicates. Greenhouse temp ranged between 24 and 30 C.

Isolations and pathogenicity tests.—Diseased seedlings were washed, blotted, surface-disinfested with 1.0% sodium hypochlorite for 1.0 min, rinsed with sterile water, and plated out on potato-dextrose agar (PDA). The fungi and bacteria obtained were tested for pathogenicity to pepper. They were grown on PDA at 27 C for 7 days. The culture in each plate was macerated in a Waring Blendor with 100 ml water. Three ml, or a 1:10 dilution of the suspension, was poured on the surface of planted pepper seeds before they were covered with soil. The test was carried out with either nontreated or diphenamid-treated soils in order to detect pathogenic organisms as well as those pathogenic only in the presence of diphenamid. Pepper was also sown in soil samples collected from fields in which damping-off was observed. This served for the reproduction of damping-off, and for obtaining diseased seedlings under controlled conditions.

Herbicide-disease interaction.—Mycelium of *Rhizoctonia solani* Kuehn [grown for 7 days on thin layers of yeast extract dextrose broth (YDB)] was washed, weighed, homogenized in a Waring Blendor for

30 s, and mixed at various concns with either nontreated or diphenamid-treated soil. Inoculum concn was expressed as mg (wet wt) mycelium/kg soil. *R. solani* isolates derived from diseased pepper or tomato seedlings were designated as P and T isolates, respectively. Unless otherwise stated, peppers and tomatoes were inoculated with P_1 and T_1 *R. solani* isolates, respectively. Diseased seedlings were recorded daily and by the end of the experiment (26 days after sowing), the accumulated percentage of postemergence damping-off was calculated. Preemergence damping-off was calculated by comparing the emergence percentage with that in noninoculated control soils. Inoculation with *Pythium* was carried out similarly, except that the fungus was grown on oat broth.

Effect of diphenamid on growth of R. solani.—An acetone solution of diphenamid was mixed with sterile water and the fine suspension obtained was mixed with cooled melted agar or broth media. The respective media were then inoculated with *R. solani* and incubated at 27 C. Percent inhibition of radial growth or mycelium dry weight at each diphenamid concn was calculated by comparison with the appropriate controls.

Colonization.—The saprophytic activity of *R. solani* was estimated by measuring its capacity to colonize bean stem segments (17). The segments were incubated in *R. solani*-inoculated soil. After each incubation period, the colonization of 100 segments was determined by plating on chloramphenicol-water agar and incubating for 24 h before examining microscopically (17). Results are expressed as percent of segments colonized by *R. solani.*

Respiration and glucose utilization.—Soil was mixed with water or a diphenamid suspension, and a glucose solution. Respiration was measured by a Gilson differential respirometer in 20 g soil samples at 28 C. Results were expressed as μl oxygen consumed/g soil/h. Glucose utilization was estimated by using the anthrone reagent (10) to measure the amount of glucose remaining after incubation.

When sterile soil was used, the procedures were carried out aseptically and the soil was mixed with macerated *R. solani* mycelium. Samples of the mycelial suspension were dried at 80 C for determining inoculum concn. Sterilization of the soil was carried out by autoclaving (121 C at 1 atm for 1.0 h on 2 successive days).

Effect on virulence.—*Rhizoctonia solani* was grown on YDB containing diphenamid at various concns. The mycelium served for inoculating soils in which pepper seeds were sown. The percentage of diseased plants was recorded.

Effect on host susceptibility.—Peppers or tomatoes were sown in noninfested soils with or without diphenamid. The pretreated seedlings obtained were transplanted at the day of emergence to diphenamid-free soil inoculated with *R. solani* at various concns. The percentage of diseased seedlings was recorded.

RESULTS.—*Pepper and tomato damping-off diseases and their interaction with diphenamid.*—Isolations were made from suspected diseased seedlings, either obtained from surveyed pepper fields, or grown in the greenhouse in soils from those fields. Disease incidence varied between 0 and 70%. Diseased seedlings were observed mainly during the first 2 wk after emergence. The total number of isolations made was over 400 and the organism isolated at the highest frequency was *R. solani;* more than 40% of suspected seedlings from the field, and 70% of those from the

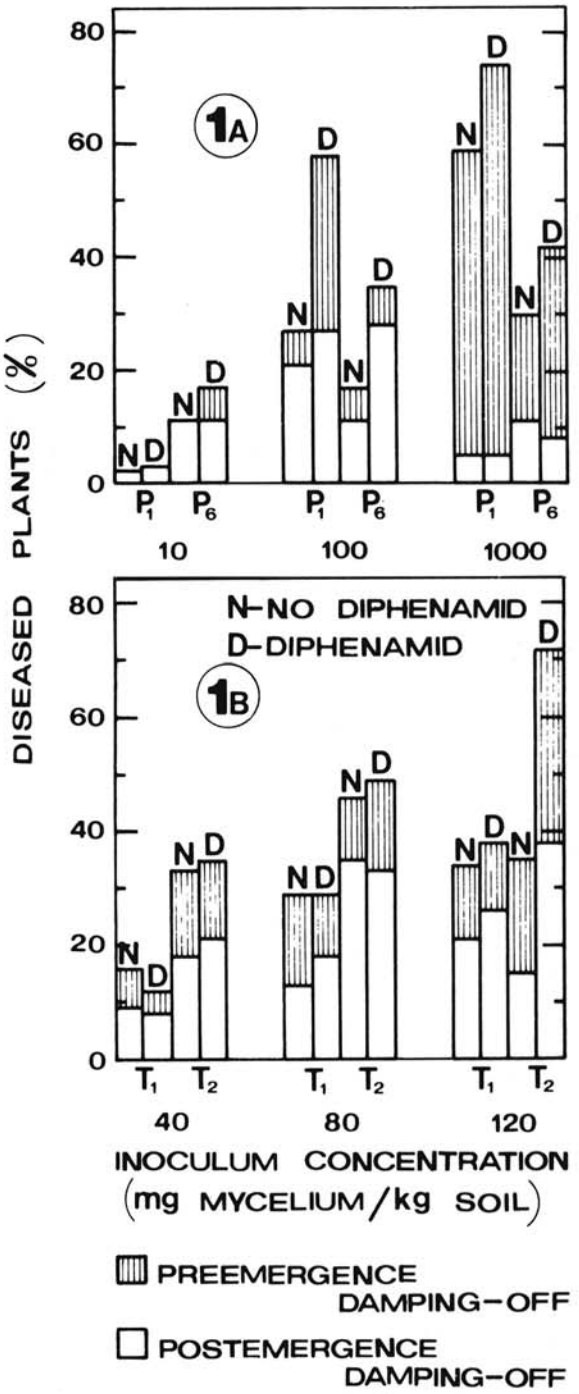


Fig. 1-(A, B). Effect of diphenamid in soil (10 μg/g) on incidence of disease caused by *Rhizoctonia solani* in pepper (1-A) and tomato (1-B). Soils were inoculated with pepper (P_1, P_6) or tomato (T_1, T_2) isolates of the pathogen at three inoculum concns.

TABLE 1. Effect of pentachloronitrobenzene (PCNB) (as Terraclor 75% WP) on disease incidence caused by *Rhizoctonia solani* in pepper in the presence of the herbicide diphenamid. Soil was mixed with 100 mg mycelium/kg soil.

Soil Treatment		
PCNB (μg/g)	Diphenamid (μg/g)	Diseased seedlings (%)
0	0	32
0	10	43
1	10	22
2	10	17
5	10	16
10	10	6
10	0	2
20	10	0
40	10	0
100	10	0

TABLE 2. Effect of diphenamid on the inhibition of linear growth of *Rhizoctonia solani* isolates from pepper (P_1) and tomato (T_1) on Martin's agar medium

Diphenamid Concn (μg/ml)	Inhibition (%)	
	P_1 Isolate	T_1 Isolate
1	−1.0	0.8
20	10.2	9.6
100	20.1	23.8
250	33.1	32.3

greenhouse, yielded this fungus on PDA cultures. Nineteen out of 20 *R. solani* isolates tested wre pathogenic to pepper. Only in rare cases were pathogenic isolates of *Pythium* sp. obtained. In addition, other fungi and bacteria were isolated. These included *Fusarium* spp., *Alternaria* spp., *Sclerotium bataticola,* and *Aspergillus* spp., all nonpathogenic to pepper seedlings when tested in both nontreated and diphenamid-treated soils. No difference was observed in the composition of mycoflora isolated from diseased seedlings grown in soils with or without diphenamid.

In the greenhouse, pepper seeds were sown in soils with or without diphenamid. These soils were artificially infested with various inoculum concns of six P isolates of *R. solani.* Typical results of two isolates are given in Fig. 1-A. These show an interaction between inoculum concn and diphenamid with respect to an increase in disease incidence (preemergence + postemergence damping-off). This increase was the highest and significant at an inoculum concn of 100 mg/kg. With the other four isolates, disease incidence increase varied from 30 to 110% above the control at inoculum concn which varied between isolates and was between 20 to 200 mg mycelium/kg soil. With each isolate, the difference between the diphenamid treatments and the controls was significant ($P = 0.05$) for at least one inoculum concn. Disease incidence also increased when diphenamid was sprayed on soil surface or when a clay soil was used. These experiments were also carried out with tomatoes. Results with two T isolates (Fig. 1-B) show a significant increase ($P = 0.05$) in disease incidence with only one isolate (T_2), and only at the highest inoculum concn. Additional experiments with tomatoes showed variable results: no change, a significant increase or decrease in disease incidence, indicating no consistent response of the tomato plants.

Experiments were also carried out with pepper sown in soils artificially infested with *Pythium* sp. No significant change in disease incidence due to diphenamid was observed. The percentages of diseased seedlings at a concn of 200 mg mycelium/kg soil were 5 and 7, for the nontreated and diphenamid-treated soils, respectively. At inoculum concn of 1,000 mg mycelium/kg soil they were 22 and 25 for the nontreated and diphenamid-treated soil, respectively.

The possibility of controlling disease caused by *R. solani* in pepper, in the presence of diphenamid, with fungicides was examined. A wettable-powder formulation of pentachloronitrobenzene (PCNB) was mixed with inoculated diphenamid-treated soil. Results (Table 1) show 86-100% control at concns of 10 μg/g or more. There was no evidence of phytotoxicity or reduced herbicidal effects. In a similar experiment, benomyl was mixed with the soil at a concn of 10 μg/g and the results were similar to those obtained with 10 μg PCNB/g soil.

Mechanisms involved in disease increase.—

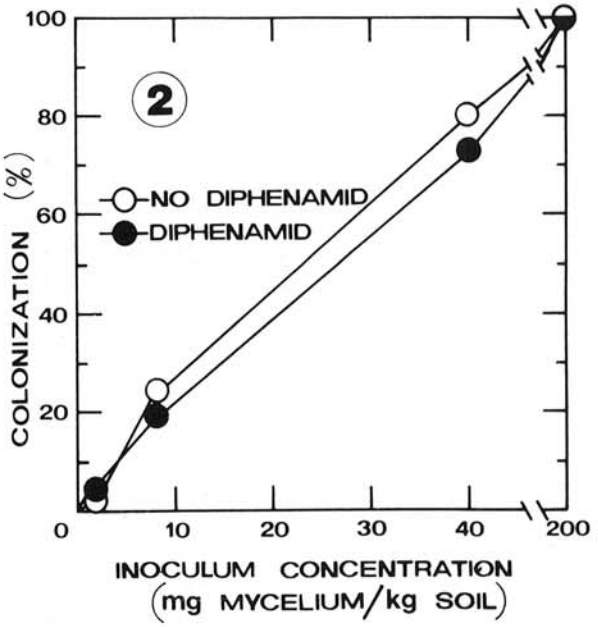
—1) Effect of diphenamid on growth and virulence of *R. solani.*—Growth of *R. solani* on PDA and Martin's agar media, containing diphenamid at various concns, was measured. Results with two isolates on Martin's medium (Table 2) show no stimulatory effect of diphenamid, and a partial toxicity at high concns. The same results were in the various media with four pepper and one tomato isolates. In all cases ED_{50} of diphenamid was above 250 μg/ml, a concn which much exceeds that of fields treated with this herbicide. Rodriguez-Kabana et al. (13) showed that certain herbicides, though toxic to soil fungi in culture, may stimulate their activity in sterile soil. The effect of diphenamid, in sterile soil, on colonization of sterile stem bean segments, respiration and glucose utilization by *R. solani* was therefore studied. Results (Fig. 2, 3) showed a nonsignificant toxic effect of diphenamid on the activity of the fungus.

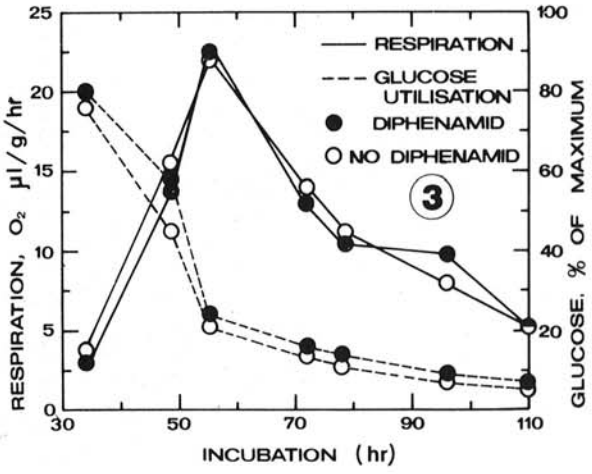
Although diphenamid does not stimulate growth of *R. solani* it is still possible that it may affect its virulence. *Rhizoctonia solani* was grown on YDB containing diphenamid at various concns. The mycelium served for inoculating the soil in which peppers were then sown. Results (Fig. 4) show no significant effect of diphenamid on the virulence at any inoculum or herbicide concn as compared with the nontreated inoculated controls.

—2) Effect of diphenamid on host susceptibility.—Pepper seedlings grown in soil free of *Rhizoctonia* with or without diphenamid, were transplanted to soil inoculated with *R. solani.* Results (Fig. 5-A) showed no significant effect of diphenamid pretreatment, at any inoculum concn on pepper resistance. This experiment was repeated, with similar results, using three other isolates varying in pathogenicity. No significant change was observed either with a clay soil, when diphenamid was applied to soil by spraying 0, 3, and 6 days after sowing, or when using seedlings 1-2 days after emergence. Similar experiments were carried out with tomato seedlings. In contrast to

pepper, diphenamid had a significant effect ($P = 0.05$) in increasing tomato resistance to *R. solani* at all inoculum concns tested (Fig. 5-B). These experiments were repeated with the same results, using either various isolates and inoculum concns, or the spraying method for diphenamid application. This effect on plant resistance was nullified, however, when tomato seedlings were used 1-2 days after emergence instead of being transplanted at the day of emergence.

—3) Effect of diphenamid on the relationships between *R. solani* and soil microorganisms.—The effect of diphenamid on the activities of the pathogen and other microorganisms was followed in natural soil. The saprophytic activity of *R. solani* is known to be correlated with its capacity to colonize plant segments in competition with the other soil microorganisms. Saprophytic activity was increased in time (Fig. 6). A subsequent decrease in saprophytic activity followed the peak in colonization. This is a decolonization phenomenon which has been attributed to the activity of antagonists to *Rhizoctonia* in natural soil (16). Diphenamid increased colonization by *Rhizoctonia* in both stages, namely, it enhanced the initial increase and slowed the subsequent decrease, both significantly ($P = 0.05$). Thus, colonization in diphenamid-treated soil was increased by 70% after 1 day of incubation, and by 105% by the end of the experiment, as compared with the control. This increase in natural soil is in contrast with the slight inhibitory effect on colonization by diphenamid observed in sterile soil (Fig. 2). A trend similar to that observed in Fig. 6 was also obtained using either P_2 and P_3 isolates of the pathogen, or in a clay soil. The increase in colonization by diphenamid, as compared with the control, was 20-90% at the various incubation periods. It was previously shown (16) that activity of *R. solani* in soil gradually decreases, with a similar decrease in disease incidence. This occurred when the pathogen was incubated under moist conditions in natural, but not in a sterile, soil. The slower decrease in colonization by *R. solani* in diphenamid-treated soil (Fig. 6) suggested the possibility that diphenamid may also slow down the decrease in time in incidence of disease caused by *R. solani* in pepper. Natural soil was inoculated with a P_2 isolate of *R. solani* at 50% MHC, and kept in plastic bags in the greenhouse. After each incubation period, soil samples were sown with pepper. Results (Fig. 7) show a significant effect of diphenamid on the rate of disease incidence. A similar trend was obtained with P_1 and P_5 isolates. Disease incidence at the various incubation





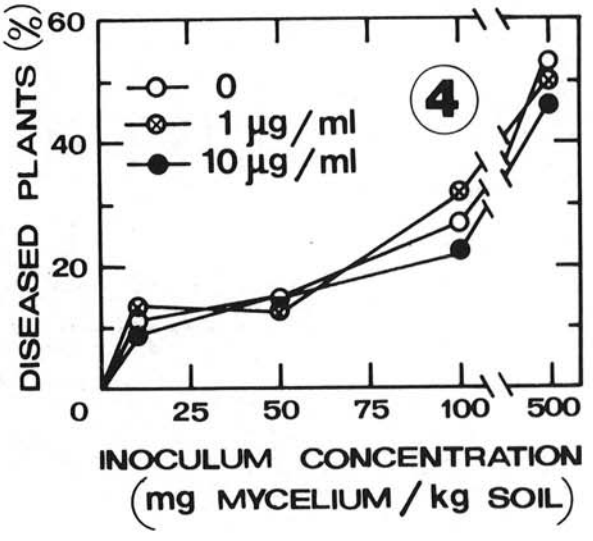


Fig. 2-4. 2) Effect of diphenamid (10 µg/g) in sterile soil on colonization of sterile bean stem segments by *Rhizoctonia solani*. Determinations were made after incubation for 24 h in soils inoculated with the pathogen. At each of the inoculum concns, colonization was determined in 100 segments. **3)** Effect of diphenamid (10 µg/g) on respiration of and glucose utilization by *Rhizoctonia solani* in sterile soil amended with 0.2% glucose. Macerated mycelium (0.1 mg/g, dry weight basis) was mixed aseptically with the soil. **4)** Effect of diphenamid on virulence of *Rhizoctonia solani* to pepper. The fungus was grown in yeast extract dextrose broth containing 0, 1, and 10 µg/ml diphenamid. The mycelium was mixed with the soil at different inoculum concns.

periods was 15-60% higher in the diphenamid-treated than in the nontreated soil.

Natural soil was amended with 0.2% glucose and respiration and glucose utilization were followed (Fig. 8). Diphenamid suppressed respiration of soil microorganisms in the initial period, but increased it later. It also suppressed glucose utilization. A similar trend was also observed with other concns of glucose in soil. The decrease in respiration caused by diphenamid in the first stage ranged between 20-55% and the later increase was 20-40%. Respiration in natural soils not amended with glucose was very low; i.e., 0.3-1.0 μliters/g/h and was not significantly affected by diphenamid.

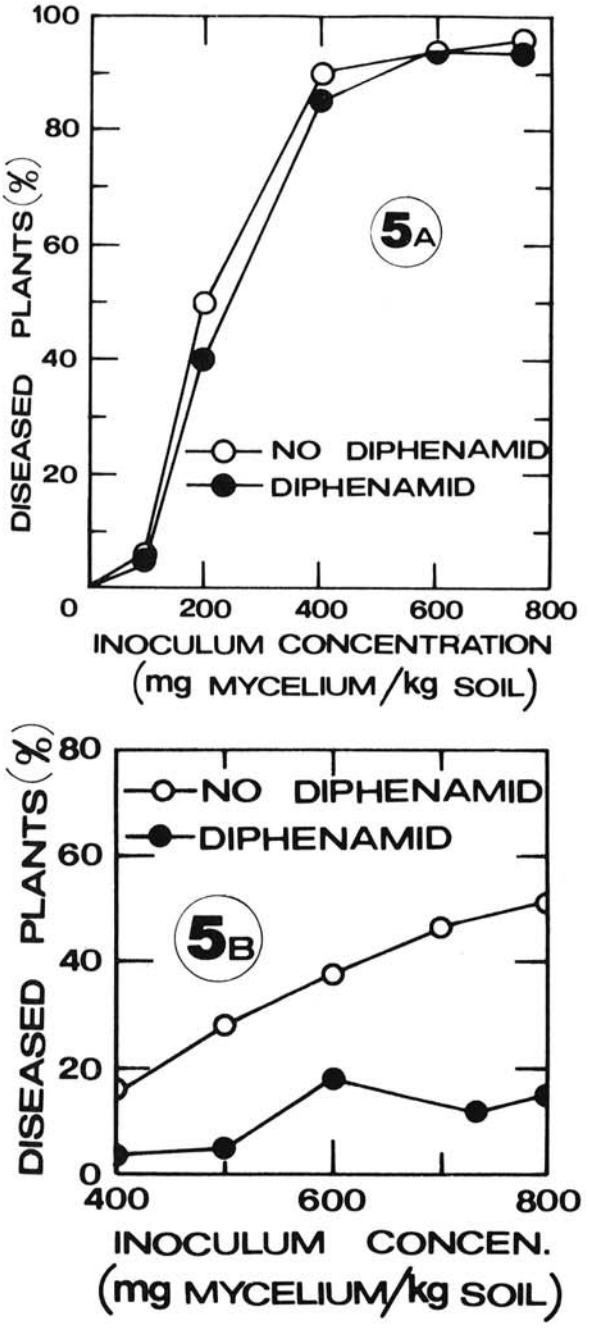


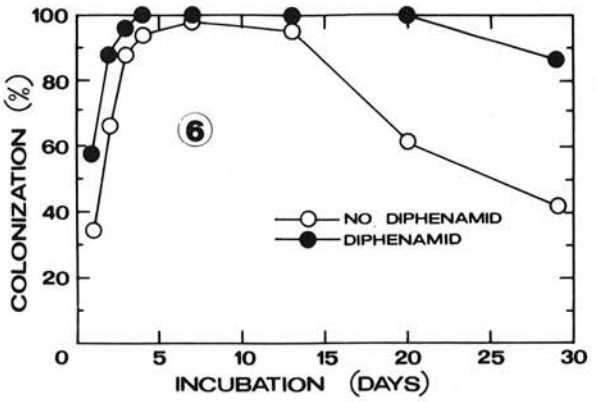
Fig. 5-(A, B). Effect of pretreatment, with diphenamid (10 μg/g) of pepper (5-A) and tomato (5-B) on their susceptibility to *Rhizoctonia solani*. Seeds were sown in soils with or without diphenamid, and seedlings were transplanted to soils inoculated with *Rhizoctonia solani*. Differences between treatments (at each inoculum concn) are significant only with tomato.

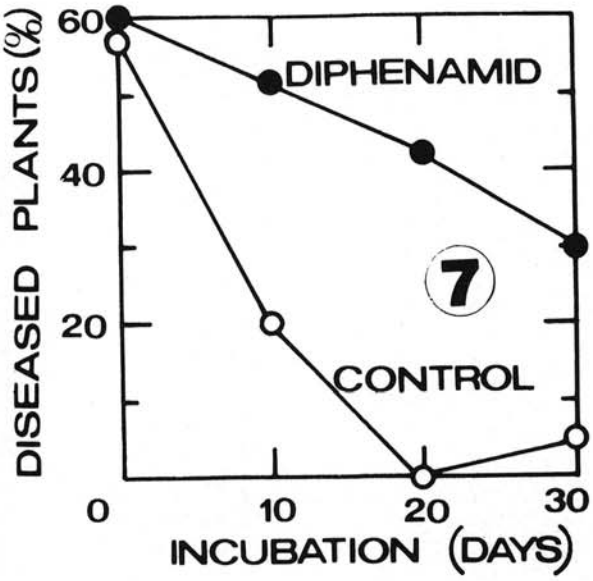
Certain herbicides were found to affect antibiotic production by various microorganisms in culture (5), hence, they may affect antagonism. Twelve isolates of fungi, bacteria and actinomycetes which produced a clear inhibition zone in cultures of *R. solani* were isolated from the soil using the method of Henis et al. (6). Each isolate was inoculated together with *R. solani* in plates containing PDA or Martin's media with or without 10 μg/ml diphenamid. This herbicide had no significant effect on the diam of the inhibitory zone produced.

DISCUSSION.—Damping-off caused by *R. solani* in pepper was increased by diphenamid. This increase was found with all tested pathogen isolates and was especially pronounced with certain inoculum concns. Chandler and Santelmann (2) showed that an interaction injurious to cotton occurred only when the herbicide trifluralin was applied at a certain inoculum concn of *R. solani* in soil.

Herbicides are biocides and are likely to be toxic, in various degrees, to many organisms in the environment. By analyzing the effects of diphenamid on the organisms involved in pepper disease, it was shown that this herbicide can increase the disease in spite of the fact that it is somewhat toxic to the pathogen. It seems that the very specific combination of herbicide, host, and pathogen determines whether, and to what extent, a disease increase (or decrease) occurs. This was shown with diphenamid, which increased pepper damping-off caused by *R. solani,* but not by *Pythium* sp. Host resistance to *R. solani* was increased by diphenamid in tomatoes but not in pepper. Thus, while in pepper suppression of soil microorganism activity was the only effect observed, in tomatoes alterations in both pathogen-microorganism relationship and host resistance occurred simultaneously, affecting the disease in opposite ways. Apparently the balance between these two determines the final effect on disease in tomatoes under various conditions. A possible action of diphenamid (or its degradation products) on the increase of tomato resistance would be through its alteration of the host metabolism, leading to a stimulation of the defense mechanisms. Reduction of Botrytis fabae disease symptoms in broad bean by the herbicide 2,4-D was attributed to a depletion in the carbohydrate content of the leaves (11). Unless the degradation products of diphenamid (14) are much more fungitoxic than the parent compound, its accumulation in tomato tissues, to levels sufficient for the inhibition of *R. solani,* is unlikely because of its low fungitoxicity (Table 1).

Diphenamid altered microbial activities in amended soil. This was indicated by competitive saprophytic ability, respiration and glucose utilization (Fig. 6, 8). Smith et al. (15) working with ammonium sulfamate, and Farley and Lockwood (4) working with PCNB found that populations of microorganisms were affected by these





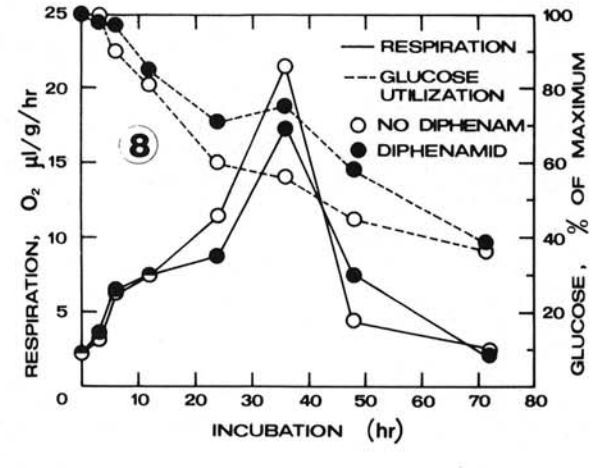


Fig. 6-8. 6) Effect of diphenamid (10 µg/g) on *Rhizoctonia solani* colonization of stem bean segments in natural loamy sand soil inoculated with the pathogen (100 mg mycelium/kg soil). After each incubation period colonization was determined in 100 segments. 7) Effect of diphenamid in soil (10 µg/g) on the incidence of disease caused by *Rhizoctonia solani* in pepper. Soil was inoculated with the fungus (200 mg mycelium/kg) and incubated at 50% moisture-holding capacity. After each incubation period, samples were sown with pepper and the percentage of diseased plants (postemergence + preemergence damping-off) was recorded. 8) Effect of diphenamid (10 µg/g) on soil microorganism respiration and glucose utilization in a loamy sand soil amended with 0.2% glucose.

pesticides only in nutrient-supplemented soils. Diphenamid increased saprophytic activity of *R. solani* which is relatively nonsensitive to this herbicide, and suppressed respiration and glucose utilization of soil microorganisms. Farley and Lockwood (4) found that in a glucose-amended soil, PCNB increased the number of nonsensitive microbes and suppressed soil respiration and glucose utilization. A competition for nutrients in soil occurs also at the infection site, for the limited amounts of root exudates important for the initial growth and penetration of plant tissues by *R. solani*. It is suggested, therefore, that the action of diphenamid on microorganisms at this site and stage, is of great importance in the shift in disease. Diphenamid enhanced the activity of *R. solani* at the parasitic stage, as well as at the saprophytic one in the absence of the host. Thus, diphenamid has a short- and long-term effect on pathogen populations and may also affect their build-up. Mickovski (9) found that diphenamid depressed the number of heterotrophic soil microflora after 10 days of incubation. In some cases this effect lasted as long as 2 mo.

Control of diseases increased by herbicides could be accomplished by using compatible fungicides. *Rhizoctonia* disease of cotton stimulated by trifluralin was controlled by PCNB (12). Another approach would be the shortening of the exposure of the young seedlings to the herbicide. This was shown in pepper by using a late preemergence application of diphenamid rather than an early one (3).

LITERATURE CITED

1. ALTMAN, J., and M. ROSS. 1967. Plant pathogens as a possible factor in unexpected preplant herbicide damage in sugarbeets. Plant Dis. Rep. 51:86-88.
2. CHANDLER, J. M., and P. W. SANTELMANN. 1968. Interactions of four herbicides with Rhizoctonia solani on seedling cotton. Weed Sci. 16:453-456.
3. ESHEL, Y., and J. KATAN. 1972. Effect of time of application of diphenamid on pepper, weeds and disease. Weed Sci. 20:468-471.
4. FARLEY, J. D., and J. L. LOCKWOOD. 1969. Reduced nutrient competition by soil microorganisms as a possible mechanism for pentachloronitrobenzene-induced disease accentuation. Phytopathology 59:718-724.
5. GOUSTEROV, G., R. BRANKOVA, and S. VLAHOV. 1972. The influence of some herbicides on the development and the antibiotical activity of Actinomycete antagonists with anti-fungal activity spectra. Pages 359-363 *in* J. Szegi, ed. Proceedings of the symposium on soil microbiology. Symp. Biol. Hung., 11, Akademiae Kiado, Budapest. 454 p.
6. HENIS, Y., B. SNEH, and J. KATAN. 1967. Effect of organic amendments on Rhizoctonia and accompanying microflora in soil. Can. J. Microbiol. 16:643-650.

7. KATAN, J., and Y. ESHEL. 1972. Increase in damping-off incidence of pepper caused by diphenamid. Weed Sci. Soc. Amer. Abstr. p. 100.
8. KATAN, J., and Y. ESHEL. 1973. Interactions between herbicides and plant pathogens. Residue Rev. 45:145-177.
9. MICKOVSKI, M. 1972. Relationship between herbicide dymid and soil microorganisms. Pages 397-400 *in* J. Szegi, ed. Proceedings of the symposium on soil microbiology. Symp. Biol. Hung., 11, Akademiae, Kiado, Budapest, 454 p.
10. MORRIS, D. L. 1948. Quantitative determination of carbohydrates with Darywood's Anthrone reagent. Science 107:254-255.
11. MOSTAFA, M. A., and S. K. GAYED. 1956. Effect of herbicide 2,4-D on bean chocolate-spot disease. Nature 178:502.
12. PINCKARD, J. A., and L. C. STANDIFER. 1966. An apparent interaction between cotton herbicidal injury and seedling blight. Plant Dis. Rep. 50:172-174.
13. RODRIGUEZ-KABANA, R., E. A. CURL, and J. L. PEEPLES. 1970. Growth response of Sclerotium rolfsii to the herbicide EPTC in liquid culture and soil. Phytopathology 60:431-436.
14. SCHULTZ, D. P., and B. G. TWEEDY. 1971. Uptake and metabolism of N,N-dimethyl-2,2-diphenylacetamide in resistant and susceptible plants. J. Agric. Food Chem. 19:36-40.
15. SMITH, N. R., V. T. DAWSON, and M. E. WEUGEL. 1945. The effect of certain herbicides on soil microorganisms. Soil Sci. Soc. Amer. Proc. 10:197.
16. SNEH, B., J. KATAN, and Y. HENIS. 1972. Colonization of stem segments and chitin particles by Rhizoctonia solani in soil. Phytopathology 62:852-857.
17. SNEH, B., J. KATAN, Y. HENIS, and I. WAHL. 1966. Methods for evaluating inoculum density of Rhizoctonia in naturally infested soil. Phytopathology 56:74-78.