Performance Characteristics of Dicloran, Iprodione, and Vinclozolin for Control of Sclerotinia Blight of Peanut

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ABSTRACT

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A technique using peanut stems was devised to quantify the persistence of three fungicides and their toxicity to *Sclerotinia minor*. Dicloran, iprodione, and vinclozolin were applied in the field to peanut stems until runoff at rates of 10, 3.3, and 2.5 mg/ml, respectively. The persistence of fungicidal activity on stems was quantified weekly via a bioassay involving inoculation of 8.5-cm-long stem segments with mycelial plugs of *S. minor*. Inhibition of resulting lesions showed vinclozolin to be the most persistent fungicide followed by iprodione and dicloran. Comparisons of fungicidal and nonfungicidal treatments indicated that vinclozolin and iprodione act primarily to prevent initial infection. Although dicloran was the least fungicidal, it was the most effective inhibitor of lesion elongation and therefore appears to have a different mode of action. Time, solar radiation, and precipitation were each correlated ($P = 0.01$) with reduced levels of inhibition by the fungicides over time. ED_{50} concentrations of dicloran, iprodione, and vinclozolin were 42.3, 7.6, and 2.5 μg/ml, respectively, according to bioassays performed immediately after treatment of stems in the laboratory. Slopes of the dosage-response curves for both iprodione and vinclozolin were much steeper than that of dicloran, which helps explain the observed differences in activity in the field study.

Additional key words: *Arachis hypogaea*, fungicide persistence

Sclerotinia minor (Jagger) Kohn (4), the cause of Sclerotinia blight of peanut, has become one of the most destructive pathogens of peanuts in Virginia and northeastern North Carolina. Effective fungicides and use patterns are needed to reduce crop losses and slow the continued spread of the fungus. Among the products tested to date, the dicarboximide fungicides have shown the highest degree of fungitoxicity to *S. minor*. Procymidone, a dicarboximide that is no longer being developed, was found to provide excellent control of the disease (9). More recently, iprodione and vinclozolin were shown to give effective control of Sclerotinia blight (7,8), and iprodione was granted registration for this purpose in 1985. Additionally, dicloran and granular PCNB have given disease suppression under field conditions (7,8).

Although the efficacy of these fungicides has been demonstrated, a more complete understanding of factors affecting their performance in the field is needed to develop efficient use patterns. For example, laboratory determinations of the fungitoxicity of dicloran, iprodione, and vinclozolin to *S. minor* indicate that considerably more dicloran would be required to achieve disease control equivalent to that of the other two fungicides (1). Higher rates of dicloran are needed but not to the degree expected on the basis of in vitro studies.

Another area of uncertainty is the timing and method of application. Thorough coverage across the row and penetration to the soil are thought to be important factors in fungicide application. The question of fungicide persistence, which influences treatment interval, has not been addressed quantitatively. Dicloran, iprodione, and vinclozolin are currently recommended for application at the initial appearance of the disease and then at 4-wk intervals until harvest. The high cost per fungicide treatment coupled with the early appearance of disease symptoms in recent years have made this approach expensive. A better understanding of the mode of activity of these fungicides and factors associated with their persistence in the field would be useful in refining current recommendations.

The objectives of this study were to 1) determine the persistence of dicloran, iprodione, and vinclozolin on peanut vines under field conditions; 2) identify the environmental factors associated with decreased fungicide activity over time; and 3) quantify the toxicity of these fungicides to *S. minor* on peanut stems.

MATERIALS AND METHODS

Field persistence of fungicides. The following products were tested: dicloran (Botran 75W), iprodione (Rovral 50W), and vinclozolin (Ronilan 50W). Glucose yeast-extract agar (GYEA) consisting of dextrose, 20 g; yeast extract, 2.0 g; KH_2PO_4, 1.0 g; $MgSO_4 7H_2O$, 0.5 g; agar, 20 g; and distilled water, 1,000 ml, was used for culture maintenance and production of inoculum. The isolate (S-2) of *S. minor* used in bioassays was isolated from a naturally infected peanut plant from Southampton County, Virginia.

A single row of field-grown peanuts, cultivar Florigiant, was divided into 2.4-m plots for fungicide treatment about 15 wk after planting. Border rows were 0.91 m from the treated rows, and standard management practices were followed. The upright vines on one side of the row were pulled back to expose the fairly uniform, prostrate, lateral stems. After removing loose debris, these lateral stems were carefully rinsed with water with a hand-held plant sprayer (Terra Verde Plant and Garden Sprayer). Fungicide suspensions in tap water were then applied to the stems until runoff with a similar sprayer. Concentrations used were 10.0, 3.3, and 2.5 mg/ml for dicloran, iprodione, and vinclozolin, respectively. These are the concentrations shown previously to control Sclerotinia blight of peanut in field trials (1).

The following bioassay was developed to quantify fungicide persistence on the vines. Treated limbs on plants were randomly selected in each plot, cut at the basal end, and brought into the laboratory. All leaves and pegs were trimmed flush with the stem, and an 8.5-cm stem segment was cut from the median portion of each limb. Each stem segment had a node in the center that served as the inoculation site. A 5-mm-

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diameter agar plug with mycelium from the periphery of an actively growing colony of *S. minor* on GYEA was placed at this node directly on the wound created when the stem was trimmed. Inoculated stem segments were incubated in moist chambers at 18 C. By day 2, distinct water-soaked lesions were visible on stems not treated with fungicide. Lesions were measured daily to determine colonization rates. After 4 days of incubation, inhibition by each fungicide was determined by comparing lesion lengths on treated and untreated stems. The ability of each fungicide to prevent infection, i.e., the fungicidal effect, was analyzed by comparing the number of nonzero values in each treatment via Fisher's exact test (3). The Wilcoxon rank sum test (3) was used to determine the ability of each fungicide to inhibit colonization once a lesion was initiated, i.e., the inhibition effect. This test compared only the nonzero values for lesion length within each treatment. A rank LSD procedure based on the Kruskal-Wallis test was used to evaluate the experimentwise error rate of each data set (5).

Bioassays for fungicide activity were conducted immediately after treating the stems and then at 1-wk intervals to measure changes in fungicide activity over a period of 4 wk. This test was conducted once in 1984 with six replicates per treatment and twice in 1985 with eight and nine replicates.

Environmental factors. Meteorological conditions during the experiment were recorded at the field site by a computerized weather monitoring unit of Virginia's Agro-Environmental Monitoring System (11). Solar radiation and rainfall were given primary emphasis. Cumulative data for both solar radiation and rainfall were obtained on each bioassay date, and the product moment (Pearson) correlation was found between each and the decrease in fungicide activity over time. The correlation between time and the change in fungicide activity was also determined.

ED_{50} determinations. Uniform, lateral limbs of 13-wk-old, greenhouse-grown peanut plants (cultivar Florigiant) were cut and trimmed as described earlier. Stem segments were rinsed, submerged for 1 min in varying concentrations of fungicide suspended in distilled water, and then inoculated as before with isolate S-2 of *S. minor*. Lesion lengths were measured at days 2–7, and lesion expansion was plotted over time for each fungicide dosage level. Percent inhibition at each fungicide concentration was determined by comparing the slopes of lines for lesion expansion on treated and untreated stems. Levels of inhibition were then plotted against fungicide concentration, and linear regression analyses were used to determine dosage levels for 50% inhibition of lesion expansion (ED_{50} values). Treatments were replicated four times, and the test was repeated with stems from field-grown Florigiant peanuts.

RESULTS

Field persistence of fungicides. None of the fungicide-treated stems assayed immediately after treatment developed lesions, but the activity of all fungicides decreased in subsequent assays during the experiment (Fig. 1). Distinct differences in fungicides were found. Fisher's exact test demonstrated that, at 1 wk after application, dicloran did not differ from untreated controls in preventing lesion formation (Table 1). Iprodione was significantly fungicidal for 2 wk and vinclozolin for 3 wk. None of the fungicide treatments in the 1984 test prevented lesion formation at 4 wk. Results in both 1985 tests were similar, except iprodione and vinclozolin remained significantly fungicidal for 4 wk in one of those tests.

Dicloran was least effective in preventing lesion formation but gave the highest level of inhibition of lesion expansion according to the Wilcoxon rank sum test (Table 2). This inhibition of lesion elongation was significant ($P = 0.01$) throughout all 4 wk of the test. In contrast, once lesions formed on stems treated with iprodione or vinclozolin, their length was not significantly different from that on untreated stems. The same trends were evident in both the

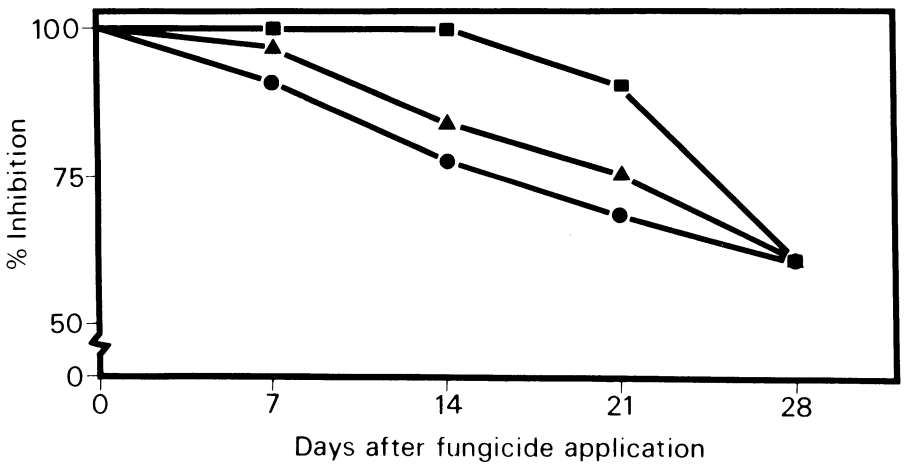

Fig. 1. Persistence of dicloran (●), iprodione (▲), and vinclozolin (■) on field-treated peanut stems based on inhibition of lesion expansion in bioassays with *Sclerotinia minor*.

Table 1. Efficacy of dicloran, iprodione, and vinclozolin in suppressing lesion elongation on peanut stems bioassayed with *Sclerotinia minor* (1984 test)

Treatment	Rate[y] (mg/ml)	Day of bioassay after fungicide application				
		0	7	14	21	28
Check		6 a[z]	6 a	6 a	6 a	6 a
Dicloran	10.0	0 b	3 ab	3 ab	6 a	4 a
Iprodione	3.3	0 b	1 ab	2 ab	5 a	5 a
Vinclozolin	2.5	0 b	0 b	0 b	2 b	4 a

[y] Fungicide suspensions in tap water were applied until runoff with a plant sprayer to simulate commercial use rates of 3.37, 1.12, and 0.84 kg/ha for dicloran, iprodione, and vinclozolin, respectively.
[z] Number of stems (six per treatment) with lesions caused by *S. minor*. Values in columns followed by the same letter(s) are not significantly different at $P = 0.05$ according to Fisher's exact test.

Table 2. Efficacy of dicloran, iprodione, and vinclozolin in suppressing lesion elongation on peanut stems bioassayed with *Sclerotinia minor* (1985, test 2)

Treatment	Rate[x] (mg/ml)	Day of bioassay after fungicide application				
		0	7	14	21	29
Check		57.3 a[y]	60.9 a	62.1 a	60.0 a	61.3 a
Dicloran	10.0	–[z]	15.2 b	21.8 b	21.8 b	17.0 b
Iprodione	3.3	–	–	37.5 ab	38.5 ab	25.5 ab
Vinclozolin	2.5	–	–	–	30.0 ab	37.0 ab

[x] Fungicide suspensions in tap water were applied until runoff with a plant sprayer to simulate commercial use rates of 3.37, 1.12, and 0.84 kg/ha for dicloran, iprodione, and vinclozolin, respectively.
[y] Lesion length (mm) at 4 days after initiation of bioassay with *S. minor*. Six stems per treatment were used to compute means. Means in columns followed by the same letter(s) are not significantly different according to the Wilcoxon rank sum test and the Kruskal-Wallis test, both at $P = 0.01$.
[z] – = No lesions formed.

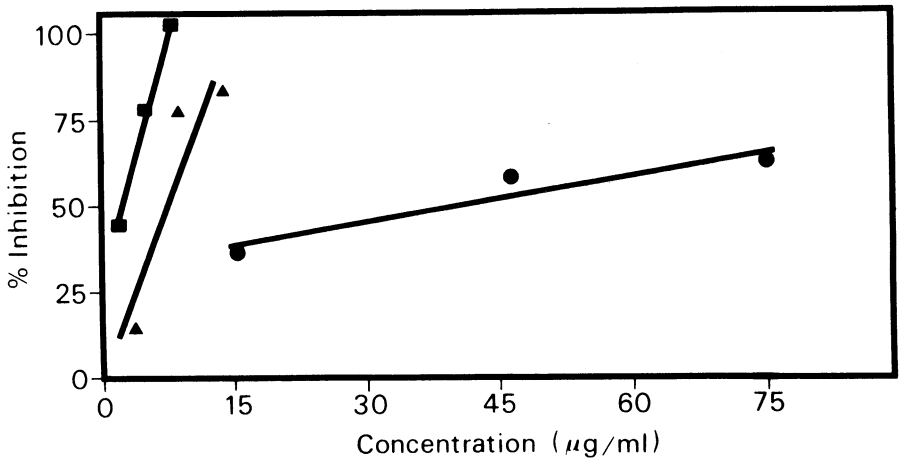

Fig. 2. Dosage-response effect of dicloran (●), iprodione (▲), and vinclozolin (■) on lesion expansion in bioassays with *Sclerotinia minor*.

1984 and the other 1985 test, except dicloran remained significantly inhibitory to lesion expansion only 3 wk in 1984.

The Kruskal-Wallis one-way ANOVA by ranks was significant at $P = 0.005$ for all data sets.

Environmental factors. Accumulated solar radiation for days 7, 14, 21, and 28 varied from a low of 2,248, 5,600, 8,849 and 11,900 langleys, respectively, in the first 1985 test, to a high of 2,886, 6,326, 9,918, and 13,103 langleys in the second 1985 test. Accumulated precipitation for the same periods varied from a low of 1.76, 2.37, 2.37, and 2.37 cm, respectively, in the second 1985 test to a high of 2.39, 3.13, 3.16, and 9.29 cm in the first 1985 test. The correlation coefficients relating these environmental factors as well as time to the length of lesions in bioassays were positive ($P = 0.01$), but none indicated a high degree of correlation.

ED_{50} determinations. According to measurements of lesion length in stem bioassay tests, ED_{50} values for dicloran, iprodione, and vinclozolin were 42.3, 7.6, and 2.5 μg/ml, respectively. All curves fit a linear model with R^2 values between 0.90 and 0.99 (Fig 2). Slopes were calculated to be 0.45, 6.7, and 9.3 for dicloran, iprodione, and vinclozolin, respectively. The test was repeated with similar results.

DISCUSSION

Although all three fungicides have been reported to suppress disease development in the field (1,7,8), results of the current study suggest that dicloran acts by a mechanism distinctly different from that of either iprodione or vinclozolin. The activity of dicloran appears to be fungistatic, as indicated by the fact that it significantly inhibited lesion expansion for 3 wk and was fungicidal only immediately after application. These data are further supported by laboratory studies on fungicide-amended agar wherein up to 100 μg/ml of dicloran was not fungicidal to *S. minor* (*unpublished*). Concentrations of less than 10 μg/ml of iprodione or vinclozolin were fungicidal in similar tests with fungicide-amended agar. Dicloran is an aromatic hydrocarbon, whereas iprodione and vinclozolin are both dicarboximides. All three fungicides have a similar spectrum of microbial activity, but their primary mode of action has not been defined (10). Data from the current study suggest that there may be fundamental differences in the mode of action of dicloran and the two dicarboximide fungicides.

The ED_{50} values for each fungicide on excised peanut stems were higher than that reported in previous studies with fungicide-amended GYEA, but the relative toxicity remained similar to previous determinations in agar media (1). Of equal significance in these tests is the difference in slope of the dosage-response curves of the fungicides. The slopes for iprodione and vinclozolin were about 14 and 20 times steeper, respectively, than that for dicloran. This means that to achieve a similar change in the percent inhibition by these fungicides, much larger changes in concentration are required for dicloran than for iprodione or vinclozolin. For example, a decrease from 75.7 to 20.5 μg/ml of dicloran resulted in the same change in activity, i.e., from 65 to 40% inhibition, as did a decrease from 4.1 to 1.4 μg/ml of vinclozolin (Fig. 2). This may explain the superior activity of dicloran in suppressing lesion elongation as opposed to the "all-or-nothing" phenomenon evident with iprodione and vinclozolin (Table 2).

It should also be noted that this study evaluated only the effect of fungicide that remains on the plant. Because *S. minor* is a soilborne pathogen, it may be that the soil surface is also an important target for fungicide treatment. Studies with PCNB indicate that the granular formulation provides greater suppression of Sclerotinia blight of peanut in field trials than spray applications of the wettable powder (8). The increased interception of fungicide by leaves and reduced deposition of fungicide on soil as well as plant tissues at the soil surface are thought to account for the reduced efficacy of spray applications of PCNB.

Many factors are known to influence the persistence of a pesticide on a plant. Sunlight is thought to be one of the most important physical determinants of the fate of pesticides in nature (6). Burchfield (2) lists 1) decomposition by hydrolysis, photolysis, microbes, etc.; 2) volatility; 3) tenacity or resistance to displacement; and 4) plant growth. Some of these factors are predictable and dependent on the physicochemical traits of a fungicide. Examples include volatility, tenacity, and decomposition. The effects of plant growth are also predictable but should not have had a major impact on results of this study, because the plants were 15 wk old at the time of fungicide application. Environmental influences, however, may vary widely but can be monitored. A better understanding of the factors affecting a given fungicide in the field would be useful in predicting the duration of disease control. This research has provided information on the persistence and mode of activity of fungicides for control of Sclerotinia blight. Although some trends were established, more work is needed to construct models that predict fungicide performance under field conditions.

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