Sclerotinia Blight of Peanut: Relationship Between in vitro Resistance and Field Efficacy of Dicarboximide Fungicides

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We thank G. M. Arrington, M. B. Britt, and J. L. Ratliff for technical assistance.

Accepted for publication 8 January 1987 (submitted for electronic processing).

ABSTRACT

Brenneman, T. B., Phipps, P. M., and Stipes, R. J. 1987. Sclerotinia blight of peanut: Relationship between in vitro resistance and field efficacy of dicarboximide fungicides. Phytopathology 77:1028-1032.

Two isolates of *Sclerotinia minor* with in vitro resistance to iprodione and vinclozolin were pathogenic to peanut in field microplots and survived as well as a fungicide-sensitive field isolate. Dicloran, iprodione, and vinclozolin were applied to peanut plants for 3 yr at annual rates of 8.41, 3.36, and 2.52 kg/ha to control Sclerotinia blight. Disease caused by in vitro fungicide-resistant isolates was suppressed 19, 33, and 87% by dicloran, iprodione, and vinclozolin, respectively, compared with 15, 24, and 76% for the sensitive isolate. Isolates recovered from tissue samples still grew on fungicide-amended media, indicating that in vitro resistance and in vivo resistance were not equivalent. Fungicide treatments reduced sclerotial populations of all isolates in soil and reduced the viability of sclerotia recovered from plots infested with sensitive isolates but not from those infested with resistant isolates. Nine isolates of *S. minor* with in vitro resistance to iprodione or vinclozolin were found to have cross-resistance to these fungicides as well as to dicloran and pentachloronitrobenzene. This work illustrates the potential risks associated with in vitro evaluation of fungicide resistance and suggests that the threat of dicarboximide resistance in *S. minor* may not be as great as originally thought, since disease control is still achieved under field conditions.

Additional key words: Arachis hypogaea.

Sclerotinia blight of peanut (*Arachis hypogaea* L.) was first observed in 1971 (14) and has since become a major disease of peanuts in Virginia. Early testing showed the dicarboximide fungicides to be effective in controlling the disease (4,7,11,13). However, in vitro studies (16) with the dicarboximide fungicide procymidone showed that actively growing mycelium of the pathogen, *Sclerotinia minor* (Jagger) Kohn (9), developed resistance to at least 100 μg/ml of procymidone at a frequency of 2.3% in the population evaluated. Resistance was stable in the absence of the fungicide, and isolates were found to be cross-resistant to iprodione and vinclozolin, two additional dicarboximides, as well as to dicloran. Although procymidone showed excellent potential for the control of Sclerotinia blight (13), registration efforts were terminated in 1980. Since 1985, iprodione has been the only fungicide with full registration for use on peanut to control Sclerotinia blight. Similar registration for vinclozolin continues to be sought. More recent studies (4) showed that in vitro resistance to iprodione and vinclozolin occurred at a frequency of 1.8% on fungicide-amended medium. Fungicide-resistant isolates in that study were capable of growing at up to 1,000 μg/ml of the fungicide to which resistance originated.

The appearance of fungicide-resistant isolates of *S. minor* is cause for concern because dicarboximide-resistant isolates of numerous other fungi have been reported (2). Although such reports usually concern tests conducted in the laboratory, there are also reports of field resistance and a loss of disease control (6,10). Much work has been done with *Botrytis cinerea* Pers. ex Fr. on a variety of crops, and results have indicated that resistant isolates could still be controlled by regular fungicide applications (8). It has been suggested that resistant isolates may be less ecologically fit than sensitive isolates or that the level of resistance was too low for expression under field conditions (1,8). Such information is essential to the development of appropriate use patterns for fungicides. This is particularly true for control of Sclerotinia blight of peanut, where the widespread use of dicarboximides is just beginning.

The objectives of this study were to characterize the dicarboximide-resistant isolates of *S. minor* obtained in earlier studies (4) for pathogenicity to peanuts treated and not treated with fungicides; for survival and regeneration of sclerotia in soils cropped to peanuts over a period of 3 yr; for competition with fungicide-sensitive field isolates in both the presence and the absence of fungicides; and for cross-resistance to other fungicides with utility in Sclerotinia blight control.

MATERIALS AND METHODS

Pathogenicity and response to fungicides. The following fungicides were used: dicloran (Botran 75W, NOR-AM Chemical Co., Wilmington, DE), iprodione (Rovral 50W, Rhône-Poulenc Inc., Monmouth Junction, NJ),. and vinclozolin (Ronilan 50W, BASF Wyandotte Corp., Parsippany, NJ). Field studies began in 1983 with the establishment of microplots constructed of fiberglass barriers (0.3 cm thick, 60 cm high, 77 cm in diameter) inserted 45 cm deep in the soil, a Dragston fine sandy loam. The field had previously been in a corn-peanut rotation and had no history of Sclerotinia blight. Sclerotia of *S. minor* for infestation of the soil were produced in a sterilized medium consisting of field soil amended with cornmeal (5% w/w). After 2 wk of incubation at 25 C, sclerotia were washed on a 325-μm-mesh sieve to remove the growth medium. Standardized quantities of 1,800 sclerotia were mixed into the upper 8 cm of soil in each microplot, resulting in a density of approximately four sclerotia per 100 g of soil. Infestation was done just before Florigiant peanut seed were planted. Plots were planted annually between May 9 and May 14, with plant densities standardized at three per plot (after emergence) to simulate field densities. Plots were maintained over 3 yr (1983–1985), and standard management practices were followed with the exception of weed control, which was primarily

manual. Plots were irrigated as needed to reduce plant stress and promote disease development. Applications of chlorothalonil (Bravo 500) or benomyl (Benlate 50W) plus sulfur (Super Six) were made according to the Virginia leaf spot advisory program to control Cercospora leaf spot. All cultural practices were performed in a manner to prevent transfer of inoculum between plots.

One fungicide-sensitive isolate of *S. minor* (S-2) and two dicarboximide-resistant isolates (R-2B and R-2C) were used to infest soil in microplots during the spring of 1983. ED_{50} values for isolate S-2 on fungicide-amended agar had previously been determined to be 1.12, 0.13, and 0.06 μg/ml for dicloran, iprodione, and vinclozolin, respectively (4). ED_{50} values for the fungicide-resistant isolates were not calculated, since growth was often not different ($P = 0.05$) among concentrations of 1, 100, 500, and 1,000 μg/ml of fungicide (4).

A randomized complete block design was used with four replications of the following treatments per isolate: dicloran, 3.37 kg/ha followed by two applications at 2.52 kg/ha; iprodione, three applications at 1.12 kg/ha; vinclozolin, three applications at 0.84 kg/ha; and no treatment. First treatments were applied about the second week of July, with subsequent applications at about 4-wk intervals. A CO_2-pressurized backpack sprayer with a single $D_2$13 (disk-core) nozzle and 345 kPa pressure was used to deliver sprays at a volume of 375 L/ha.

Disease data were recorded three times during the growing season. The first two were disease incidence ratings taken in late July and August, respectively. These were based on the number of infection sites as determined by the presence of active mycelial growth. At harvest in early October, the severity of disease for each plant was rated on a scale of 0 (no disease) to 10 (plant dead). Samples of diseased tissue were collected from each plot at harvest. After soaking in 0.5% NaOCl for 1 min, tissue was placed on glucose yeast-extract agar (GYEA) amended with 100 μg/ml of both chloramphenicol and chlortetracycline HCl. Actively growing colonies of *S. minor* were then tested for their ability to grow on GYEA amended with dicloran (8 μg/ml), iprodione (2 μg/ml), and vinclozolin (2 μg/ml). Although the dicarboximide-resistant isolates of *S. minor* found in previous studies were all resistant to much higher concentrations of fungicide (4,16), these concentrations were chosen to enable detection of isolates with lower levels of fungicide resistance.

Peanut plants were dug and inverted to dry about the first week of October each year. The yield of peanuts per microplot was determined about 2 wk later and expressed on a basis of 4–6% moisture (w/w).

Competition of fungicide-sensitive and fungicide-resistant isolates. Microplots for this study were established in the spring of 1984. Soybeans had been grown at this site in 1983, and the soil type was a Goldsboro fine sandy loam. Management was similar to that described previously. The soil was infested with 900 sclerotia each from a sensitive and a resistant isolate of *S. minor*, resulting in a density of about four sclerotia per 100 g of soil. Three pairs of isolates were utilized (S-2 + R-2C, S-5 + R-5B, and S-1 + R-1D). Plots either were not treated or were given three applications of vinclozolin (0.84 kg/ha) annually according to the schedule described previously. A randomized complete block design was used with four replications. Ratings of disease severity, sampling for resistance, and harvesting were done as described previously.

Determination of sclerotial populations. Populations of sclerotia in soil within microplots were determined in June 1985. Twelve soil cores (2 × 8 cm) were taken from each microplot. After thorough mixing, samples were screened over 3-mm mesh to remove plant debris. Samples equivalent to 100 g dry weight of soil were processed with a semiautomatic elutriator (6.75-min elutriation period) as previously reported (18) for quantitative recovery of sclerotia from soil.

Sclerotia were counted, surface-disinfested 2 min in 0.5% NaOCl, placed on GYEA amended with 100 μg/ml each of chloramphenicol and chlortetracycline HCl, and incubated at 20 C to determine viability. Colonies of *S. minor* resulting from germinating sclerotia were transferred to GYEA amended with dicloran (8 μg/ml), iprodione (2 μg/ml), or vinclozolin (2 μg/ml) to test for resistance. The incidence of other fungi associated with the sclerotia in these assays was recorded.

Cross-resistance. Fungicide suspensions were prepared in sterile distilled water and pipetted into flasks containing autoclaved GYEA cooled to 70 C. The final concentration of all fungicides was 100 μg/ml. PCNB (Terraclor 75W, Uniroyal Chemical Co., Naugatuck, CT) was evaluated in a similar but separate experiment. The medium was stirred during addition of fungicide and for 1 min thereafter, then aliquots were dispensed at 23 ml per petri dish (85 mm diameter).

The nine isolates of *S. minor* resistant to dicarboximide fungicides (obtained during earlier in vitro sensitivity testing) were evaluated along with the three sensitive field isolates from which they originated (4). Petri plates with fungicide-amended or nonamended medium were seeded at the perimeter with a 5-mm-diameter agar plug with mycelium from the periphery of an actively growing colony of *S. minor* on GYEA. Plates were incubated at 25 C in darkness, and linear growth (mm) was measured at 24-hr intervals.

RESULTS

Pathogenicity and response to fungicides. Figure 1 shows the mean disease severity rating at harvest of control and fungicide-treated plots for the 3 yr of testing. Disease caused by in vitro fungicide-resistant isolates was suppressed 19, 33, and 87% by dicloran, iprodione, and vinclozolin, respectively, compared with 15, 24, and 76% for the sensitive isolate. All isolates were pathogenic to peanut, and no differences in virulence of fungicide-sensitive and fungicide-resistant isolates were noted. Similar results with these and other isolates were obtained in growth-chamber studies (*unpublished*). Disease caused by all three isolates

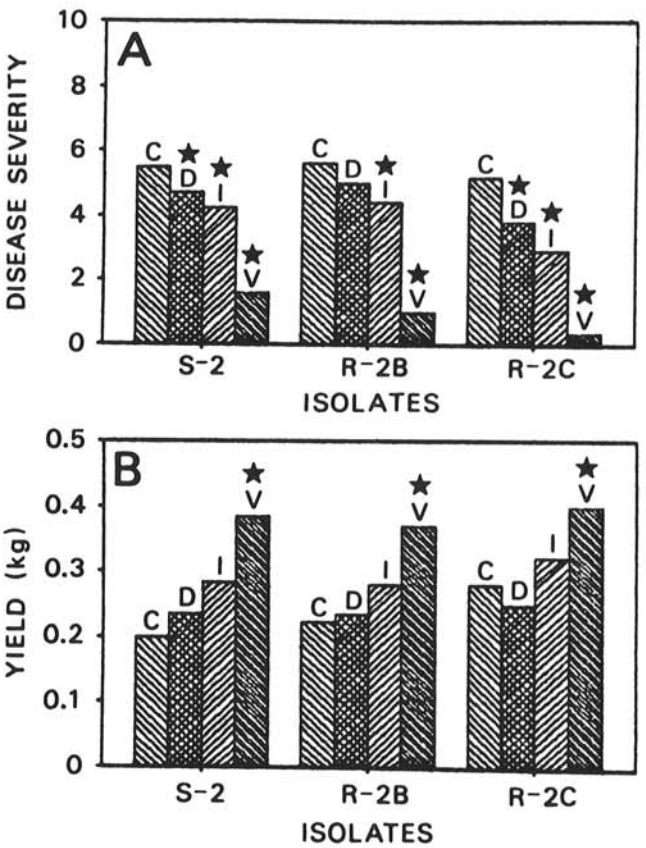

Fig. 1. Effect of fungicides on **A,** severity of Sclerotinia blight of peanut at harvest (0 = no disease, 10 = plant dead) and **B,** pod yields (kilograms per microplot) in microplots infested with either a fungicide-sensitive (S-2) or a fungicide-resistant (R-2B or R-2C) isolate of *Sclerotinia minor*. All data are the mean of 3 yr, each with four replications. C = check, D = dicloran, I = iprodione, and V = vinclozolin; ★ indicates significant difference from the control at $P = 0.05$ according to Duncan's multiple range test.

was suppressed about equally by fungicides, regardless of an isolate's in vitro sensitivity. This trend was also evident in disease incidence ratings taken earlier in the season. Yield data indicated no apparent differences in the aggressiveness of isolates, and consistent increases resulted from use of the dicarboximide fungicides (Fig. 1). Despite these trends, isolates recovered in tissue samples at harvest retained their original sensitivity or resistance on fungicide-amended GYEA.

Population densities of sclerotia in soil after 2 yr were suppressed ($P = 0.05$) by fungicide treatment (Table 1). Where no fungicides were applied, sclerotial densities of resistant isolates in soil were generally lower than those of the sensitive isolate. Most sclerotia recovered from microplot soils were found to retain their original fungicide sensitivity or resistance. In vitro resistance, however, was detected in one isolate in a plot infested with a sensitive isolate and treated with iprodione for 2 yr. Not all sclerotia recovered from plots infested with resistant isolates were still resistant.

Fungicide treatments decreased the viability of sclerotia from soil infested with sensitive isolates of *S. minor* but had no apparent effect on viability of sclerotia in soil infested with resistant isolates (Fig. 2). Both resistant isolates behaved similarly, and results for them are combined. Numerous fungi were found associated with sclerotia after surface-disinfestation with NaOCl. Species of *Fusarium, Trichoderma,* and *Verticillium* were most commonly isolated. An analysis of population changes in these genera showed that *Trichoderma* spp. were associated with 8.9, 9.5, and 8.1% of sclerotia in plots treated with dicloran, iprodione, and vinclozolin, respectively, compared with 3.0% in the untreated plots. The incidence of other fungi did not appear to be affected by fungicide treatments.

TABLE 1. Populations of sclerotia of *Sclerotinia minor* in microplot soils after 2 yr of peanut culture and fungicide treatment

Isolate[w] Treatment[x]	Sclerotia per 100 g of soil	Resistant[y] (%)
S-2		
Untreated	52 a[z]	0
Dicloran (8.41 kg/ha)	28 bcd	0
Iprodione (3.36 kg/ha)	30 bc	4
Vinclozolin (2.52 kg/ha)	10 de	0
R-2B		
Untreated	44 ab	96
Dicloran (8.41 kg/ha)	25 cd	96
Iprodione (3.36 kg/ha)	22 cde	92
Vinclozolin (2.52 kg/ha)	6 e	85
R-2C		
Untreated	29 bcd	96
Dicloran (8.41 kg/ha)	25 cd	100
Iprodione (3.36 kg/ha)	11 cde	95
Vinclozolin (2.52 kg/ha)	6 e	80

[w] S = in vitro fungicide-sensitive field isolate and R = in vitro fungicide-resistant isolate of *S. minor*.
[x] Cumulative annual rates of fungicide applied.
[y] A maximum of 64 sclerotia per treatment were sampled.
[z] Means followed by the same letter(s) are not significantly different at $P = 0.05$ according to Duncan's multiple range test.

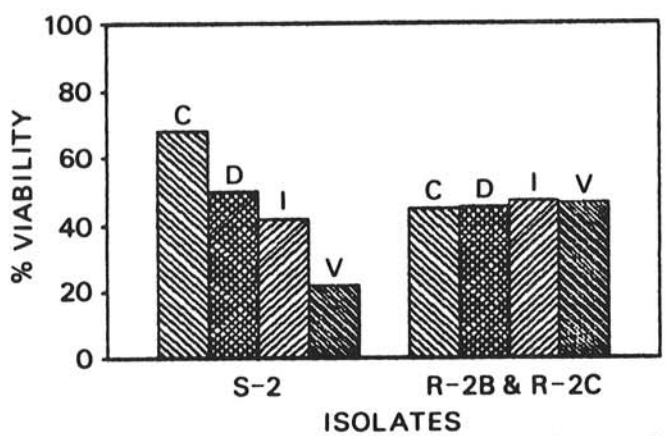

Fig. 2. Effect of fungicides on the viability of sclerotia from microplot soils infested with in vitro fungicide-sensitive (S-2) and fungicide-resistant (R-2B and R-2C) isolates of *Sclerotinia minor*. C = check, D = dicloran, I = iprodione, and V = vinclozolin; means of R-2B and R-2C are given.

Competition of dicarboximide-sensitive and dicarboximide-resistant isolates under field conditions. Plants in microplots used for competitive pathogenicity tests (paired isolates) showed less disease than plants in plots where pathogenicity and response to fungicides were tested for single isolates. Applications of vinclozolin, however, resulted in disease suppression of 73–93%, compared with the untreated plots. Tissue biopsies revealed differences in the competitive pathogenicity of isolate pairs. Where no fungicide was applied, the percent recovery of in vitro resistant isolates varied from 0 to 64% (Table 2). The percent recovery of in vitro resistant isolates increased dramatically with vinclozolin treatment of plots and ranged from 67 to 100%; the total number of isolates recovered was low, however, as a result of excellent disease control.

Populations of sclerotia in plots were quite low, even where fungicide was not applied (Table 3). Neither isolate S-1 nor isolate R-1D appeared to be adapted for survival, as only 0.8 sclerotium per 100 g of soil was recovered in untreated plots, and none of these was viable. Higher numbers of sclerotia were recovered from soil infested with the other isolate pairs, but populations were low considering an initial infestation level of four sclerotia per 100 g of soil. The percent viability of sclerotia from plots varied widely among isolates.

The resistant isolates were competitive with sensitive isolates and in two instances accounted for greater than 50% of sclerotia recovered from soil when fungicides were not used (Table 3). A shift toward greater recovery of sclerotia from resistant isolates than from sensitive isolates where fungicides were applied was not evident. Detection of such a shift, however, may require an increase in sample size.

TABLE 2. Fungicide sensitivity of isolates obtained from diseased plants in microplots infested with equal numbers of sclerotia from in vitro sensitive and in vitro resistant isolates of *Sclerotinia minor*

Treatment Isolate pair[a]	1984 Recovered (no.)	1984 Resistant (%)	1985 Recovered (no.)	1985 Resistant (%)
Untreated				
S-2 + R-2C	22	64	8	63
S-5 + R-5B	21	43	10	20
S-1 + R-1D	10	0	2	0
Vinclozolin[b]				
S-2 + R-2C	1	100	2	100
S-5 + R-5B	6	100	0	···
S-1 + R-1D	6	67	1	100

[a] S = in vitro fungicide-sensitive field isolate and R = in vitro fungicide-resistant isolate of *S. minor*.
[b] Cumulative annual rates were 2.52 kg/ha.

TABLE 3. Populations of sclerotia in microplots 2 yr after infestation with fungicide-sensitive and fungicide-resistant isolates of *Sclerotinia minor*

Treatment Isolate pair[a]	Recovered (no.)	Sclerotia per 100 g of soil	Viable (%)	Resistant (%)
Untreated				
S-2 + R-2C	11	2.8	82	67
S-5 + R-5B	24	6.0	44	70
S-1 + R-1D	3	0.8	0	···
Vinclozolin[b]				
S-2 + R-2C	1	0.3	100	0
S-5 + R-5B	4	1.0	25	100
S-1 + R-1D	1	0.3	100	0

[a] S = in vitro fungicide-sensitive field isolate and R = in vitro fungicide-resistant isolate of *S. minor*.
[b] Cumulative annual rates were 2.52 kg/ha.

Cross-resistance. Fungicide-resistant isolates of *S. minor* were capable of growth on media amended with dicloran, iprodione, or vinclozolin (Table 4). A similar but separate test indicated that cross-resistance existed to PCNB as well. Dicloran was the most inhibitory to resistant isolates, followed by iprodione, then vinclozolin. This is somewhat surprising because dicloran is the least fungitoxic to sensitive field isolates (4), although similar results have been reported for *S. minor* (17) and *Monilinia fructicola* (19).

TABLE 4. Cross-resistance of isolates of *Sclerotinia minor* to three fungicides[a]

Isolate[b]	Mycelial growth (mm/day)			
	Untreated	Dicloran	Iprodione	Vinclozolin
S-1	24.0	0.6	0.0	0.0
R-1A	14.0	4.0	3.3	5.7
R-1B	13.8	4.4	3.5	5.3
R-1C	16.8	4.0	3.3	9.0
R-1D	16.6	4.0	1.8	5.3
S-2	8.6	0.8	0.0	0.0
R-2A	14.9	4.5	6.6	10.2
R-2B	15.1	4.5	6.2	5.6
R-2C	15.0	4.1	7.0	8.9
S-5	11.9	0.5	0.0	0.0
R-5A	14.1	3.7	3.0	7.6
R-5B	14.1	5.5	8.2	10.3
Mean of sensitive	14.8	0.6	0.0	0.0
Mean of resistant	14.9	4.3	4.8	7.5

[a] Glucose yeast-extract agar amended with 100 μg/ml of fungicides.
[b] S = in vitro fungicide-sensitive field isolate and R = in vitro fungicide-resistant isolate of *S. minot*. A, B, C, and D designate origin of specific isolate (4).

DISCUSSION

The development of fungicide resistance is a problem of increasing importance to modern agriculture. This is a relatively recent phenomenon and usually involves one of the more selective fungicides that have a single-site mode of action, although the problem is not limited to this class of compounds (20). Because of the potentially disastrous effects, fungicide resistance has become a major consideration before seeking to register a new compound. Such determinations are generally based on laboratory studies or early monitoring results combined with more theoretical considerations (20).

There has been concern about the possibility of dicarboximide-resistant isolates of *S. minor* becoming a problem when such fungicides are widely used in peanut culture (4,17). Until now, the absence of field data made accurate assessment of the situation difficult. Our findings indicate that isolates of *S. minor* selected on the basis of in vitro resistance are capable of surviving and competing pathogenically with sensitive isolates. This occurred even in the absence of the fungicides but was enhanced when they were applied. Dicarboximide resistance does not appear to be detrimental to the survival of the fungus. In fact, a supplemental study with sclerotia from two resistant isolates of *S. minor* showed them to be even more tolerant of high-temperature stress than sclerotia from their sensitive parent isolate (3). This is in contrast to the situation with dicarboximide-resistant isolates of *B. cinerea*, which often have decreased vigor and rapidly revert to their sensitive state when the selective influence of the fungicide is removed (12). Furthermore, such isolates of *B. cinerea* show an unusual degree of sensitivity to osmotic stress (1). Although this was the case with some isolates of *S. minor* resistant to the dicarboximides, the trend was not as distinct as reported for other fungi (*unpublished*). Considering these traits, as well as the stability of dicarboximide resistance in *S. minor*, such isolates might persist in nature for a period of years.

Applications of iprodione and vinclozolin have been reported to increase the longevity of sclerotia of *S. minor* in soil (5). This could have a detrimental effect on long-term disease control by allowing higher populations of sclerotia to develop. The trend was reversible, however, if *Trichoderma* spp. were added to the soil. The data presented in this study demonstrate that the viability of sclerotia in Virginia either is not affected or is reduced by applications of these fungicides (Fig. 2). This discrepancy might be explained by the high populations of *Trichoderma* spp. that appear to reside in peanut field soil (15). The fact that fungicide applications substantially increased the recovery of *Trichoderma* spp. from sclerotia supports this theory.

The overall implications of this research are twofold. First, although the threat of field resistance in *S. minor* may appear to be less severe than originally thought, it remains a very real biological phenomenon that should not be ignored. This is supported by the development of in vitro resistance in one isolate of *S. minor* under selection pressure in the field. Further studies are being conducted on that isolate to determine its significance.

Considering the current lack of fungicides with alternate modes of action, the dicarboximides should be used only as part of an integrated control program utilizing several disease-suppressive inputs. Such an integrated program should include measures to delay onset of the disease, such as planting late, reducing seeding rates, and using 0.91-m spacing of single rows, as well as selecting a suitable cultivar and avoiding unwarranted injury to vines (P. M. Phipps, *unpublished*). An active survey program should also be conducted annually to detect any changes in sensitivity of the fungus, particularly where a loss of disease control is suspected.

The second implication concerns the methodology used for fungicide resistance screening. Dicarboximide-resistance in *S. minor* can be induced readily in the laboratory, but such isolates still respond to fungicide treatment in the field. This phenomenon, not fully understood, may be due to the profound differences between growth on a nutrient-rich agar medium and growth as a parasite on a living plant. It is possible that similar situations exist with other pathogen-pesticide interactions. The speed and convenience of many in vitro screening procedures make them attractive, particularly if results correlate well with findings in the field. The limitations of such studies must always be considered, however, particularly in the absence of field data.

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