Integration of Genotype and Age-Related Resistances to Reduce Fungicide Use in Management of Alternaria Diseases of Cotton and Potato

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ABSTRACT

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Concepts for the integration of genotype resistance, age-related resistance, and fungicide for the suppression of Alternaria diseases were developed and evaluated by a computer simulation model and in the field. The model reflects the effects of environment, genotype resistance, and fungicide efficacy on *Alternaria solani* in potatoes. We found that changes in host resistance, with age and among genotypes, could be compensated for by adjusting the intensity of fungicide applications, i.e., by increasing the frequency of sprays toward the end of the season and spraying moderately resistant cultivars at longer intervals than susceptible cultivars. The time during the growing season when application of a systemic spray within a routine application of protectant sprays would most effectively suppress *Alternaria* spp. was examined in simulation experiments and in the field. The efficacy of a single systemic spray was highest when applied toward the end of the season when host susceptibility increased. The concepts for integration were evaluated in six field trials, three involving *A. macrospora* in cotton and three involving *A. solani* in potatoes. In most cases, the disease suppression achieved by the integration treatment did not differ significantly from that achieved by application of either protectant fungicides (maneb, mancozeb, or chlorothalonil) on a 7-day schedule or systemic fungicides (tebuconazole or difenoconazole) on a 14-day schedule, although up to five fewer sprays were applied in the integration treatment.

Additional keywords: disease control, *Gossypium barbadense*, IPM, *Solanum tuberosum*.

Early blight, caused by *Alternaria solani* Sorauer, is one of the main causes of defoliation of potato (*Solanum tuberosum* L.) (5, 19,20) and Alternaria leaf spot, caused by *Alternaria macrospora* A. Zimmerm., is a major disease that limits economic production of Pima cotton (*Gossypium barbadense* L.) in Israel and elsewhere (2,20,27,31). Severe epidemics of early blight may restrict potato yields by up to 20 to 30% (6,25), and Alternaria leaf spot may restrict cotton yield by up to 20 to 40% (2,21,24,27). To suppress Alternaria diseases, fungicides often are applied to the foliage. During a typical growing season, potato and Pima cotton fields are sprayed with fungicide 5 to 10 times. Until recently only protectant fungicides were available for the suppression of Alternaria diseases. Tebuconazole (Folicur, Bayer AG, Leverkusen Bayerwerk, Germany) and difenoconazole (Score, Ciba, Basle, Switzerland) are new systemic fungicides that are available for use. Application of these fungicides in potato and cotton crops resulted in a longer period of effectiveness and sometimes led to superior disease suppression and to yields higher than those achieved by application of protectant fungicides (25,27,31).

Experience with late blight of potatoes has indicated that integration of several factors to suppress disease can be efficacious and, therefore, may be successful with various Alternaria diseases. Genotype resistance of potatoes combined with fungicide applications has shown additive effects in reducing late blight epidemics (11,12). Consequently, recommendations to potato growers in the northeastern United States are to adjust the frequency of late blight fungicide applications according to the resistance of the cultivar: Susceptible cultivars are sprayed every 7 days, moderately susceptible cultivars every 10 days, and moderately resistant cultivars every 14 days (13). In another study, the relative contribution of genotype resistance and protectant fungicides in potato early blight suppression were estimated by a computer simulation model (28). Shtienberg and Fry (28) found that spraying a cultivar moderately resistant to *A. solani* on a 17-day schedule suppressed disease at levels similar to those achieved by spraying a susceptible cultivar on a 7-day schedule. It was suggested that in areas where both early and late blights threaten the crop, cultivars moderately resistant to both diseases should be sprayed on a 14-day schedule, whereas cultivars susceptible to one disease or both, should be sprayed on a 7-day schedule. The appropriateness of these suggestions was corroborated recently in the field (32).

The response of potato and cotton to *Alternaria* spp. changes as the host ages. Immature potato plants are relatively resistant to early blight. However, after initiation of tuberization, susceptibility increases gradually, and mature potato plants are very susceptible to *A. solani* (18–20,22,33). Cultivars differing in genotype resistance usually follow a similar pattern of changes in age-related resistance. Cotton plants show two peaks of susceptibility. The cotyledons are highly susceptible to *A. macrospora*, five to

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nine times more than the first true leaves (1). Between the cotyledon stage and the initiation of flowering, plants are relatively resistant to the pathogen. After flowering, host susceptibility increases gradually and supports a rapid increase in disease development (31).

Adjustment of fungicide applications according to age-related resistance was considered mainly with respect to initiation of spraying. Early sprays had little, if any, effect on overall suppression of Alternaria diseases (6,17,18,26,27,29). Various models have been developed to predict the optimal timing for initiation of spraying. Most models involve prediction of host physiological age (10,17, 26,30).

Results of previous studies (23,27,28,31,35) enable us to develop concepts for the integration of genotype and age-related resistances in management of Alternaria diseases to reduce fungicide use. The concepts are as follows: application of fungicides is not needed in plants at the vegetative stage because they are relatively resistant. Accordingly, spraying should be initiated only when host response to *Alternaria* spp. shifts toward increased susceptibility, i.e., at the initiation of the reproductive stage (23,26). The frequency of subsequent sprays should be determined according to the genotype resistance of the cultivar and the efficacy of the fungicide, in relation to changes in age-related resistance. Accordingly, protectant fungicides should be applied initially at relatively long intervals that are shortened as the crop ages. Toward the end of the season, more effective control, by a systemic fungicide, is recommended (26). Genotype resistance will be considered by spraying moderately resistant cultivars less frequently than susceptible ones (28,30,32). Elements of these concepts have been utilized, in part, in the Potato Disease Management Program (PDM) developed in Wisconsin for the management of potato early and late blights (34). According to PDM, spraying intensity against early blight is increased during the season by adjustment of the rate of fungicide (i.e., beginning with a low rate and continuing later in the season with progressively higher rates). PDM, however, does not consider genotype resistance or the efficacy of the fungicide.

In this study we examined the concepts outlined above for the integration of genotype resistance, age-related resistance, and fungicide efficacy in management of Alternaria diseases. Experiments were conducted by a computer simulation model and in the field. Field trials were performed in Israel and involved cotton and potato crops. A preliminary report has been published (15).

MATERIALS AND METHODS

Simulation studies. Various aspects related to integration of genotype resistance, age-related resistance, and fungicide were studied by a computer simulation model (16). The model reflects the effects of environment and cultivar resistance on the development of *A. solani* in potatoes (16,26,28) and includes the initial deposition of the fungicide chlorothalonil (3) and its subsequent weathering, redistribution, loss, and efficacy (4). Predictions of the model were validated in the field and were accurate (16). The model (written in the "C" language) was operated on a DOS-PC equipped with a "C" compiler. Because the cultural practices in Israel differ markedly from those in the northeastern United States (where the model was developed and validated) and because the model was not validated in Israel, the input data and initial parameters used for driving the model reflect the crop, disease, and weather characteristics typical for the northeastern United States. Consequently, the model was used only for addressing epidemiological questions and not for developing guidelines for disease management. This has been done in the past (26,28,29,32). Initializing parameters for the experiments presented in this report were as follows: length of season (from date of planting until vine killing) was 105 days; median emergence occurred on day 20 after planting; initial disease was 1 lesion per 10 plants 30 days after planting. Application of the protectant fungicide chlorothalonil was simulated, at a rate of 1.34 kg a.i./ha. Sprays were initiated at the date of disease onset. The simulation experiments reported here used 9 years of actual meteorological data (1977 to 1981 and 1984 to 1987) recorded at Freeville, NY, and were executed with cultivars susceptible (for example, Norchip), moderately susceptible (Kennebec), or moderately resistant (Rosa) to *A. solani*. The area under the defoliation progress curve (AUDPC) (27) was used as a measure of epidemic intensity. The period used for calculating AUDPC was from the date of disease onset until the end of the season. AUDPC units are proportion days.

The assumption that changes in host resistance with age can be compensated for by adjusting the frequency of fungicide application was examined for the susceptible cultivar. The following treatments were included in this set of runs: i) untreated control; ii) fungicide applied on a 7-day schedule, six sprays total; iii) fungicide applied at variable intervals determined in relation to changes in age-related resistance of the host, i.e., starting with long intervals that were subsequently shortened; spraying intervals were 11, 9, 7, 6, and 5 days, six sprays total. In the next two treatments (iv and v) fungicide was applied at variable intervals but not in relation to changes in host resistance: iv) starting at short intervals that became longer with crop age; spraying intervals were 5, 6, 7, 9, and 11 days, six sprays total; and v) starting at long intervals that were shortened and then were lengthened again; spraying intervals were 9, 6, 5, 7, and 11 days, six sprays total. In all treatments the last spray was applied 20 days before the end of the season because a previous study showed that sprays applied later than that date do not contribute substantially to disease control (26).

In another set of experiments, the integration of genotype resistance, age-related resistance, and protectant fungicides was examined. The hypothesis was that spraying a cultivar with some level of resistance to *A. solani* less frequently than spraying a susceptible cultivar would not affect disease suppression substantially. Simulation experiments included the three types of cultivars, i.e., susceptible, moderately susceptible, and moderately resistant. Sprays were timed at variable intervals adjusted to compensate for changes in age-related resistance as described above. The actual intervals were determined separately for each cultivar as follows. For the susceptible cultivar, spraying intervals were 11, 9, 7, 6, and 5 days, six sprays total. For the moderately susceptible cultivar, spraying intervals were 14, 10, 8, and 7 days, five sprays total. For the moderately resistant cultivar, spraying intervals were 17, 14, and 10 days, four sprays total. For comparison, experiments also included treatments in which sprays were applied on a 7-day schedule (seven sprays total) and an untreated control.

Simulation experiments also were performed to determine when a single application of a systemic fungicide within routine applications of protectant sprays would most effectively suppress early blight. The fungicide module of the simulator was developed and validated for chlorothalonil, a protectant fungicide (3,4), and not for the newly registered systemic fungicides. The systemic fungicides (tebuconazole and difenoconazole) are more effective than the protectants. To mimic the effects of a more effective fungicide in the model, chlorothalonil was applied on two consecutive days. Although this treatment is obviously not similar to the application of a systemic spray, it may give some indication of what would happen when a more effective control is applied. This treatment will be referred to as the "double-protectant treatment." The double-protectant treatment was applied once in each run, at different times during the season. Its contribution was expressed in terms of the relative control efficacy, i.e., the improvement in disease suppression achieved by the treatment in relation to routine 7-day protectant sprays. In the routine treatment, a total of six sprays was applied. The procedure used for calculating the relative control efficacy was as follows.

Efficacy of fungicidal control achieved by the different treatments was determined in relation to that achieved by the 7-day protectant treatment. AUDPC values in the 7-day treatment (A_7) and in the untreated control plots (A_C) were used to evaluate the control efficacy achieved by the 7-day treatment (C_7): $C_7 = 1 - A_7/A_C$.

Thus, greater control efficacy is associated with more effective disease suppression. Similarly, the control efficacy achieved by the tested treatment (C_T) was calculated in relation to AUDPC values in the untreated plots. The control efficacy in each of the tested treatments then was related to that achieved in the 7-day treatment (relative control efficacy, R_T) as follows: $R_T = (C_T - C_7)/C_7 \times 100$.

Field studies. Field trials were performed to evaluate the applicability and significance of the integration concepts. Disease suppression in plots treated according to the concepts of integration was compared with that achieved by routine applications of protectant or systemic fungicides. Experiments involved cotton and potato crops. For cotton, three field trials were conducted, two in 1991 (trials 1 and 2) and one in 1992 (trial 3). Experiments were conducted in commercial fields located in the coastal (trials 1 and 3) or inland (trial 2) plains of Israel. These regions differ with respect to soil type and, to some extent, microclimate. Cultivar S-5 was sown in trial 1, and cultivar F-177 was sown in trials 2 and 3; both are susceptible to *A. macrospora*. Seeds were sown to a depth of 2 to 4 cm during the first (trials 1 and 2) or third (trial 3) weeks of April; there were 10 seeds per meter of row, with 1-m spacing between rows. The previous crop was also Pima cotton. The crop was drip-irrigated and maintained according to commercial cultural practices. Experimental plots in each trial were arranged in a randomized complete block design with four replicates per treatment. Each experimental plot was 18 × 25 to 35 m (trials 1 and 2) or 18 × 100 m (trial 3). Fungicides (in 90 to 100 L of water per ha) were applied by means of a tractor-mounted boom sprayer with cone-jet X3 nozzles (Degania Sprayers, Degonia, Israel) at a pressure of 350 kPa. Sprays did not contain spreader, sticker, or adjuvant.

Experiments with potatoes were conducted in the northern Negev region of Israel. Certified potato seeds (whole tubers, each weighing 50 to 100 g) were machine-planted during the spring season, during the last week of February 1991 (trial 4), or planted by hand during the autumn, during the second week of September 1992 and 1993 (trials 5 and 6). Cultivars Nicola and Alpha were planted in trial 4, Cara and Alpha in trial 5, and Alpha in trial 6. Cara is moderately resistant, Alpha is moderately susceptible and Nicola is susceptible to *A. solani*. Cultivar response was determined in field trials (D. Shtienberg, *unpublished data*). Plots consisted of four 7-m-long rows. Interrow spacing of plants was 0.96 m, and intrarow spacing of plants was 25 to 30 cm. Plots were separated from each other by fallow areas approximately 1 m wide. Fungicides (in 260 to 300 L of water per ha) were applied by means of a motorized backpack-sprayer at a pressure of 275 kPa with cone-jet X6 nozzles. Sprays did not contain spreader, sticker, or adjuvant.

In each trial one protectant and one systemic fungicide were used. Protectant fungicides included maneb (Manebgan, 50% FC, Agan Ltd., Ashdod, Israel) at a rate of 2.0 kg a.i./ha; mancozeb (Manzidan, 80% WP, Rohm and Haas, Croydon, England) at a rate of 2.4 kg a.i./ha; and chlorothalonil (Bravo, 50% SC, ISK Bioteck, Painesville, OH) at a rate of 1.5 kg a.i./ha. Systemic fungicides included tebuconazole (Folicur, 25% EC, Bayer AG) at a rate of 0.25 kg a.i./ha; and difenoconazole (Score, 25% EC, Ciba) at a rate of 0.25 kg a.i./ha.

Treatments. The following four treatments were included in all trials: i) untreated control; ii) protectant fungicide (maneb, mancozeb, or chlorothalonil) applied on a 7-day schedule; iii) systemic fungicide (tebuconazole or difenoconazole) applied on a 14-day schedule; and iv) a protectant and a systemic fungicide applied at variable intervals adjusted according to changes in age-related resistance of the host. This treatment will be referred to hereafter as the "integration treatment." Details on the type of fungicides, number of applications, and spraying intervals are presented in Table 1. Experimental plots were not inoculated artificially with *Alternaria* spp. because inoculum was present naturally at the test sites: infested debris from previous crops (cotton) or airborne spores from adjacent fields (potato). In the cotton trials, spraying was initiated according to the recommended threshold (i.e., when an average of 1 lesion per 10-m row of plants was detected on true leaves) but not earlier than flowering. This happened 61, 56, and 68 days after planting in trials 1, 2, and 3, respectively. In the potato trials, spraying was initiated soon after the appearance of *A. solani* lesions in the canopy but not earlier than initiation of tuberization. This happened 71, 56, and 43 days after planting in trials 4, 5, and 6, respectively.

The time during the growing season at which a single application of a systemic spray within routine applications of protectant sprays would most effectively suppress early blight was examined in trial 6, in addition to treatments 1 through 4 indicated above. A single application of difenoconazole (0.25 kg a.i./ha) was applied (in different treatments) 43, 65, or 86 days after planting. These treatments will be referred to as difenoconazole-1, -2 or -3, respectively. Mancozeb (2.4 kg a.i./ha) was applied during the rest of the season on a 7-day schedule, eight sprays total. After the application of difenoconazole in each of the treat-

TABLE 1. The number of fungicide applications and spraying intervals in trials conducted to evaluate the efficacy of various treatments on *Alternaria macrospora* in cotton and *A. solani* in potato

Fungicide treatment	Spray interval (days)	Number of applications and fungicides[y]							
		Trial 1	Trial 2	Trial 3	Trial 4 (cv. Nicola)	Trial 4 (cv. Alpha)	Trial 5 (cv. Alpha)	Trial 5 (cv. Cara)	Trial 6 (cv. Alpha)
Cotton									
Control	–	0	0	0					
Protectant	7	10M	10M	12M					
Systemic	14	5T	6T	6T					
Integration	Variable[z]	3M+3T	3M+3T	3M+3T					
Potato									
Control	–				0	0	0	0	0
Protectant	7				8C	8C	8Mz	8Mz	10Mz
Systemic	14				4T	4T	5T	5T	5D
Integration	Variable[z]				2C+3T	2C+2T	2Mz+3T	2Mz+2T	3Mz+2D

[y] Protectant fungicides: M = maneb (2.0 kg a.i./ha); C = chlorothalonil (1.5 kg a.i./ha); and Mz = mancozeb (2.4 kg a.i./ha). Systemic fungicides: T = tebuconazole (0.25 kg a.i./ha) and D = difenoconazole (0.25 kg a.i./ha).

[z] Protectant (P) and systemic (S) fungicides were applied at variable intervals as follows: Trials 1, 2, and 3: P, →12 to 14 days P, →7 to 10 days P, →5 to 7 days S, →14 to 17 days S, →12 to 14 days S. Trials 4 (cv. Nicola) and 5 (cv. Alpha): P, →14 days P, →7 to 10 days S, →14 to 17 days S, →12 to 14 days S. Trials 4 (cv. Alpha) and 5 (cv. Cara): P, →14 to 17 days P, →14 days S, →17 to 21 days S. Trial 6: P, →14 days P, →7 days P, →7 days S, →14 days S.

ments, a 14-day interval was maintained before application of the next mancozeb spray.

Disease assessment. Disease was assessed visually independently by two individuals, and the average scores were recorded. Assessments were made every 7 to 14 days from the appearance of disease symptoms in the field until the end of the season. For cotton, disease-induced defoliation was assessed on 10 (1991) or 20 (1992) arbitrarily chosen plants in each experimental plot as follows: The disease severity of attached leaves and proportion of shed leaves were assessed separately for the lower (<30 to 50 cm), middle (30 to 50 to 70 to 90 cm) and upper (>70 to 90 cm) levels of the canopy. Ranges are given because the height of the crop varied slightly among trials. Disease severity was determined with the aid of a disease-assessment scale (9). Leaf abscission sites on the main stem and branches were easily distinguished, and leaf shedding was determined with the aid of a defoliation-assessment key (27). These assessments served as the basis for estimates of the defoliation induced by Alternaria leaf spot (calculated as a weighted sum of estimates of the disease severity of attached leaves and the proportion of shed leaves [25]). For potatoes, defoliation in the two middle rows of each plot was estimated using a modification of a blight-assessment key (11). Disease records were used to calculate the AUDPC values for each of the treatments. Results were subjected to statistical analysis, and where F values showed significant differences, Fisher's protected LSD test was applied at $P = 0.05$.

RESULTS

Simulation studies. Simulation experiments were conducted to study components of the integration treatment. The intensity of the simulated epidemics for the susceptible cultivar varied substantially among years, ranging from an AUDPC value of 11 in 1985 to 21.2 in 1981. Application of chlorothalonil on a 7-day schedule decreased early blight substantially (Table 2). However, improved disease suppression was achieved by scheduling sprays in accordance with changes in host resistance with age. AUDPC values among the 7-day and the integration treatments differed significantly ($P = 0.02$) as determined by a t test for paired observations (years). Timing fungicide applications at variable intervals, and not in accordance with changes in host resistance with age, did not affect disease suppression compared with the 7-day treatment (Table 2).

Simulation runs were performed for the three types of cultivars to test the possibility of integrating genotype resistance, age-related resistance, and fungicide in disease control. As expected, disease was more severe for the susceptible cultivar than for the moderately susceptible or the moderately resistant cultivars. Genotype resistance in the simulator reflects the level of resistance currently available in potato cultivars grown in the northeastern United States. For the three types of cultivars, application of chlorothalonil on a 7-day schedule reduced AUDPC values significantly, compared with the untreated control (Table 3). Although one, two, and three fewer sprays were applied in the integration treatment compared with the 7-day treatment, respectively, for the susceptible, moderately susceptible, and moderately resistant cultivars, differences in AUDPC among these treatments were not significant (Table 3).

Disease suppression achieved by some of the double-protectant treatments was better than that achieved by the 7-day protectant sprays, as reflected by values of the relative control efficacy. The contribution of the double-protectant treatment increased gradually when the treatment was applied later in the season. Similar effects were observed for the three types of cultivars; however, the most pronounced contribution of the double-protectant treatment (24%) was achieved for the susceptible cultivar when applied 85 days after planting (Fig. 1).

The simulation experiments described above were repeated using different values of initial parameters. These were length of season, initial disease level, disease onset date, and spraying intervals. In general, trends in all simulation experiments were similar to those described above (Tables 2 and 3; Fig. 1). Accordingly, results are not shown in this report.

Field studies. Alternaria leaf spot appeared in the cotton trials at or soon after flowering (56 to 68 days after planting). Disease developed relatively slowly at first, and only at 50 to 70 days after disease onset (100 to 150 days after planting) did defoliation levels reached 1%. However, defoliation increased rapidly thereafter, and by the end of the season, defoliation induced by *Alternaria* sp. in the untreated plots exceeded 80% in all trials. A mild epidemic developed in trial 1 and relatively severe ones in trials 2 and 3 (Fig. 2). Defoliation in all three plots treated with maneb on a 7-day schedule or with tebuconazole on a 14-day schedule was significantly lower than that of the control plots throughout the entire growing season. However, differences between these two treatments were insignificant. Effects of the integration treatment resembled those of the routine fungicide treatments, although only six sprays (three maneb plus three tebuconazole) were applied. Similar effects of the treatments were observed when the intensity of the epidemics was expressed in terms of AUDPC values (Fig. 2).

Alternaria epidemics in the potato trials followed a pattern similar to that described for the cotton trials. Initial disease symptoms were observed relatively early, and disease developed slowly at first, followed by rapid disease progress. By the end of the season, defoliation induced by *A. solani* exceeded 90% in the untreated control plots in all trials (Figs. 3 and 4A). Disease suppression achieved by the 7-day protectant (chlorothalonil or mancozeb) or the 14-day systemic (tebuconazole or difenocon-

TABLE 2. Effect of various fungicide treatments on the suppression of *Alternaria solani* in potato[y]

Fungicide treatment	Number of sprays and spray intervals (days)	AUDPC[z]
Untreated control	0	15.7 (±1.0)
Fixed intervals	6 (7,7,7,7,7)	8.8 (±1.3)
Integration treatment	6 (11,9,7,6,5)	7.6 (±1.2)
Variable intervals	6 (5,6,7,9,11)	9.0 (±1.3)
Variable intervals	6 (9,6,5,7,11)	8.9 (±1.4)

[y] Experiments were performed by a computer model simulating disease development and fungicide effects in a susceptible cultivar.

[z] AUDPC = area under the disease progress curve; AUDPC units are proportion days. Results are averages of nine growing seasons (±SE).

TABLE 3. Integration of genotype resistance, age-related resistance, and protectant fungicide in the management of potato early blight[y]

Cultivar response to *A. solani*	Fungicide treatment	Number of sprays and spray intervals (days)	AUDPC[z]
Susceptible	Untreated control	0	15.7 a
	7-day	7 (7,7,7,7,7,7)	8.7 b
	Integration treatment	6 (11,9,7,6,5)	7.6 b
Moderately susceptible	Untreated control	0	9.7 b
	7-day	7 (7,7,7,7,7,7)	2.7 c
	Integration treatment	5 (14,10,8,7)	3.2 c
Moderately resistant	Untreated control	0	8.2 b
	7-day	7 (7,7,7,7,7,7)	3.2 c
	Integration treatment	4 (17,14,10)	4.3 c

[y] Experiments were performed by a computer simulation model reflecting the response of susceptible, moderately susceptible, and moderately resistant cultivars to *Alternaria solani*.

[z] AUDPC = area under the disease progress curve; AUDPC units are proportion days. Results are averages of nine growing seasons. Numbers followed by different letters differ significantly ($P < 0.05$) according to Fisher's protected LSD test.

azole) treatments was significant at most assessment dates. Differences among the protectant and systemic treatments were not significant in trials 4 and 6, but superior disease suppression was achieved by tebuconazole compared with mancozeb in trial 5 (Fig. 3). Three to five fewer sprays were applied in the integration treatment than in the 7-day protectant treatment, and a similar number (one more or one less) of sprays was applied compared with the number in the 14-day systemic treatment. In general, disease suppression achieved by the integration treatment resembled that achieved by the 7-day protectant and the 14-day systemic treatments (Figs. 3 and 4A). The only exception to these results was in trial 4, in which disease suppression by the 14-day tebuconazole treatment was significantly better than that of the integration treatment. However, differences between the integration treatment and the 7-day protectant treatment were nonsignificant (Fig. 3C and D).

The time in the growing season at which a single application of a systemic spray within a routine application of protectant sprays would most effectively suppress early blight was examined in trial 6. A trend toward improved disease suppression was observed when the single difenoconazole spray was delayed during the season. By the end of the season, *Alternaria*-induced de-

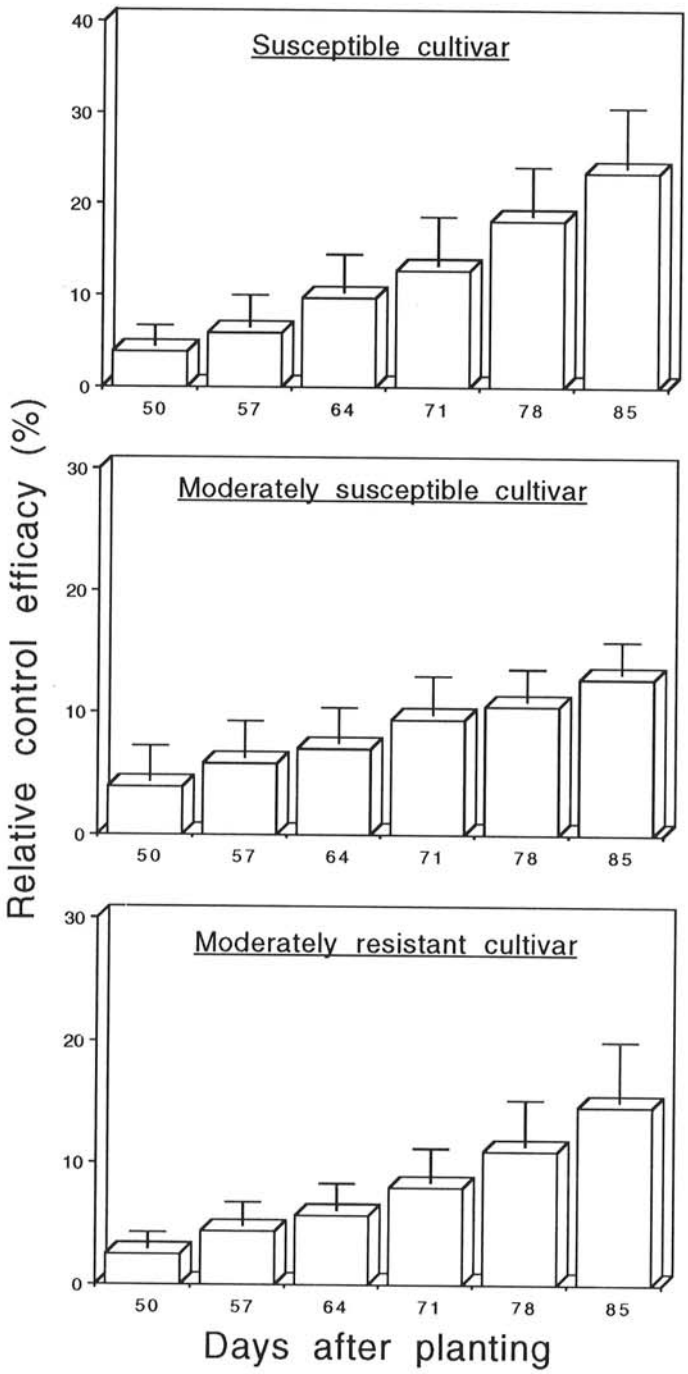

Fig. 1. The contribution of a double-protectant fungicide treatment (mimicking the effect of a systemic fungicide) applied at different times during the growing season to the suppression of *Alternaria solani* in potato. The value of the relative control efficacy indicates the suppression of early blight achieved by a certain treatment in relation to that achieved by a 7-day treatment with chlorothalonil. Experiments were performed by a computer simulation model. Results are averages of nine growing seasons; bars indicate the SE.

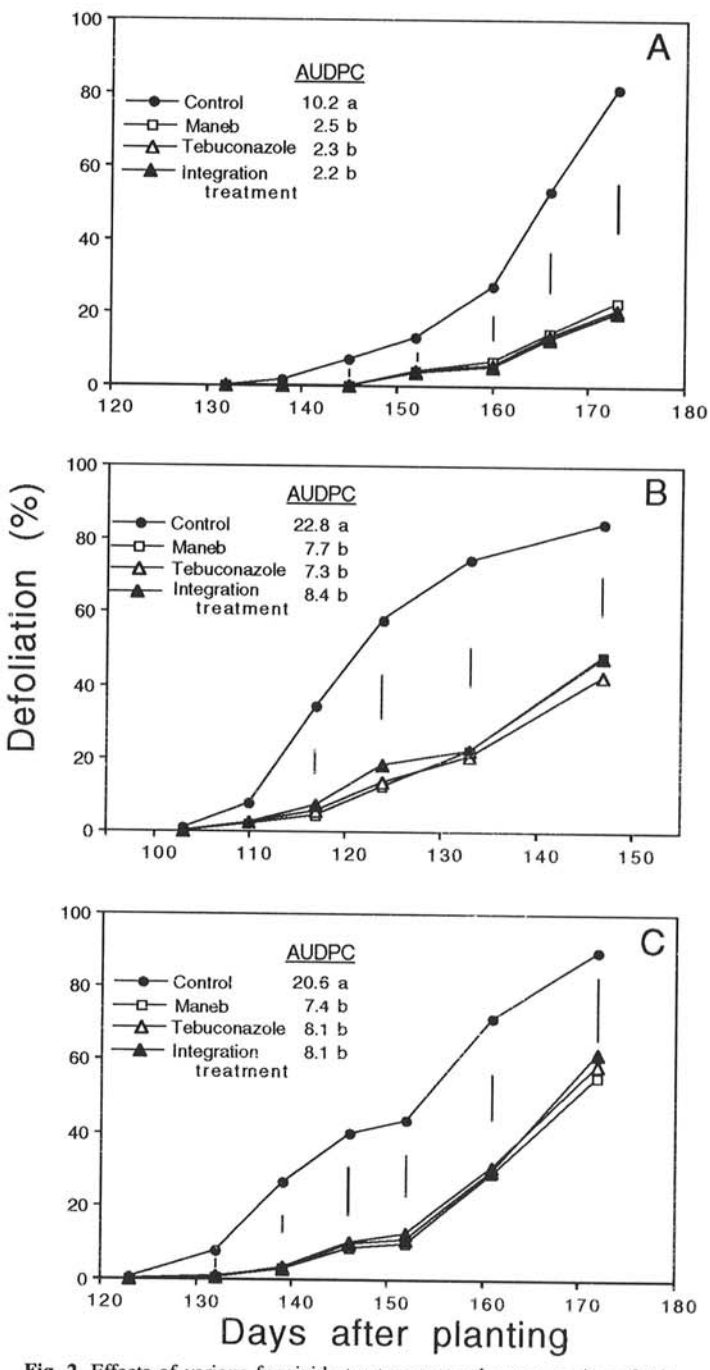

Fig. 2. Effects of various fungicide treatments on the suppression of *Alternaria macrospora* in Pima cotton in three field trials. **A,** Trial 1; **B,** trial 2; and **C,** trial 3. Maneb was applied on a 7-day schedule at a rate of 2.0 kg a.i./ha; tebuconazole was applied on a 14-day schedule at a rate of 0.25 kg a.i./ha; both fungicides were applied in the integration treatment at variable intervals (Table 1). Bars indicate the LSD for each sampling date at $P < 0.05$. Values of AUDPC (area under the disease progress curve) followed by different letters differ significantly ($P < 0.05$) according to Fisher's protected LSD test.

foliation was significantly higher in the difenoconazole-1 treatment than in the difenoconazole-3 treatment (Fig. 4B). The value of the relative control efficacy for the tebuconazole-2 treatment was 3.5% and for the tebuconazole-3 treatment was 24%.

DISCUSSION

The assumption underlying the concepts for integration presented in this study is that the effects of different control measures are complementary and additive. Accordingly, application of one measure may compensate for a decrease in another measure. Integration of three measures was examined in this study—genotype resistance, age-related resistance, and fungicide. Genetic and age-related resistances were considered as measures in which their contributions are more or less predetermined. Fungicides were used as a flexible measure by which it was possible to compensate for a decrease in the efficacy of the other two measures. The magnitude of compensation was determined by adjusting the frequency of sprays and by selecting fungicides with a variable level of efficacy (i.e., protectant or systemic).

The first component of integration was age-related resistance. Increase in host susceptibility with age has been reported in nearly all *Alternaria*-host systems. In his book, Rotem (20) mentions 18 pathosystems in which this phenomenon has been documented. The physiological causes of age-related resistance are not well known (20). Horsfall and Dimond (14) introduced a theory on "high- and low-sugar" diseases and claimed that plant tissue that is low in sugar becomes resistant to biotrophic pathogens (e.g., rusts), which are associated with "high-sugar" diseases. On the other hand, tissue that is low in sugar becomes susceptible to necrotrophic pathogens (e.g., *Alternaria* spp.), which are associated with "low-sugar" diseases. Late in the season the nonreducing sugars disappear from the foliage because they are drained into the ripening fruit. In addition to a decrease in the amount of sugars in the foliage, senescence is associated with other biochemical processes, such as a decrease in the rate of the alkaloid solanin, which inhibits growth of *A. solani* in vitro (33), increased permeability of cell membranes, and changes in nutrient contents (20) .

The results of our study indicate that changes in host resistance with age can be compensated for by application of fungicides. Better disease suppression was achieved in the simulation experiments by a treatment in which the frequency of fungicide applications was increased toward the end of the season compared with a treatment in which the same number of sprays was applied on a fixed 7-day schedule (Table 2). In another experiment, the number of sprays in the integration treatment was reduced, but disease suppression was similar to that of the 7-day treatment (Table 3). The conclusion derived from these trials is that changes in age-related resistance can be complemented by adjusting the frequency of fungicide applications.

The second component of integration was genotype resistance. Sources of genotype resistance to *A. solani* in *S. tuberosum* are relatively rare. Some level of resistance is available in commercial cultivars, but in most cases, resistance is associated with low-yield, late-maturing cultivars (20). Genotype resistance in *G. barbadense* is not yet available in commercial cultivars in Israel. Genotype resistance is not the only factor determining host response to *Alternaria*. In some cases, the condition of growth may be at least as important as genotype resistance. In general, vigorously growing plants are more resistant than poorly developed plants. The yield, or the ratio of yield to foliage, affects susceptibility, with higher yielding crops being more susceptible to the disease. Similarly, retardation of growth and sensitization to

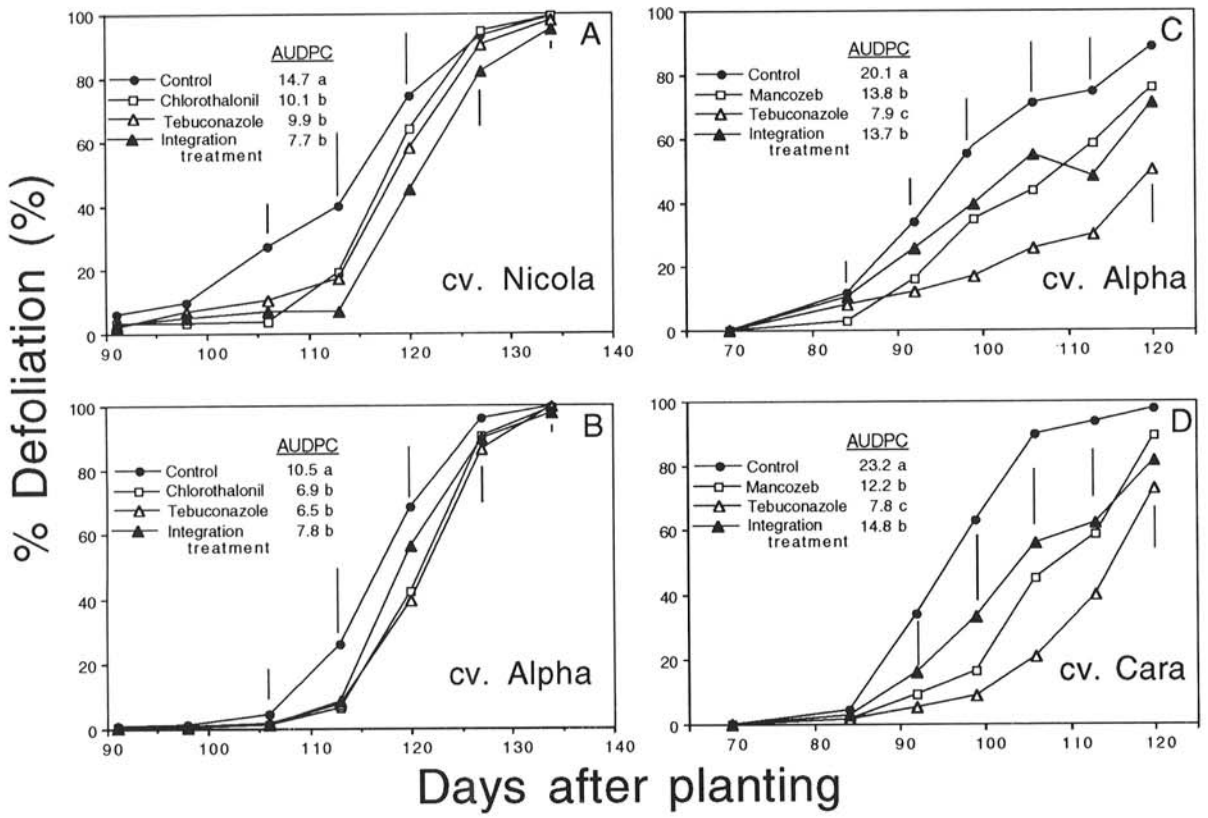

Fig. 3. Effects of various fungicide treatments on the suppression of *Alternaria solani* in potato in two trials. **A and B,** Trial 4 and **C and D,** trial 5. Cultivar Nicola is susceptible, Alpha is moderately susceptible, and Cara is moderately resistant to *A. solani*. Chlorothalonil (at 1.5 kg a.i./ha) and mancozeb (at 2.4 kg a.i./ha) were applied on a 7-day schedule; tebuconazole (at 0.25 kg a.i./ha) was applied on a 14-day schedule; chlorothalonil or mancozeb and tebuconazole were applied in the integration treatment at variable intervals (Table 1). Bars indicate the LSD for each sampling date at $P > 0.05$. Values of AUDPC (area under the disease progress curve) followed by different letters differ significantly ($P < 0.05$) according to Fisher's protected LSD test.

disease are affected by sandstorms, drought, and nutrient deficiencies (20).

Genotype resistance was considered in the integration treatment by reducing the frequency of fungicide applications in the more resistant cultivars. This approach was tested in the simulation experiments and in trials 4 and 5. Since the results obtained in the field trials were not consistent, we concluded that the contribution of genotype resistance should be considered with more caution. In trial 6, spraying intervals did not exceed 14 days, and disease control in the integration treatment was adequate (Fig. 4A).

The third component of integration was fungicide. The availability of the new systemic, highly effective fungicides against *Alternaria* spp. makes it possible to incorporate them into disease management programs and reduce the number of protectant sprays. Difenoconazole and tebuconazole belong to the triazole group of fungicides, which alter the pathway of sterol biosynthesis of the fungal pathogen. The specific mode of action puts these fungicides at risk for development of fungal resistance. Several strategies have been proposed for the use of fungicides with potential resistance problems (7,35). These include using an at-risk fungicide in a mixture with another fungicide, alternating applications of an at-risk fungicide with another fungicide, integrating use of an at-risk fungicide with other methods of disease management, and limiting the number of applications of an at-risk fungicide within a growing season. The last two strategies are implemented in our management approach, i.e., integrating various control measures and limiting the number of systemic sprays.

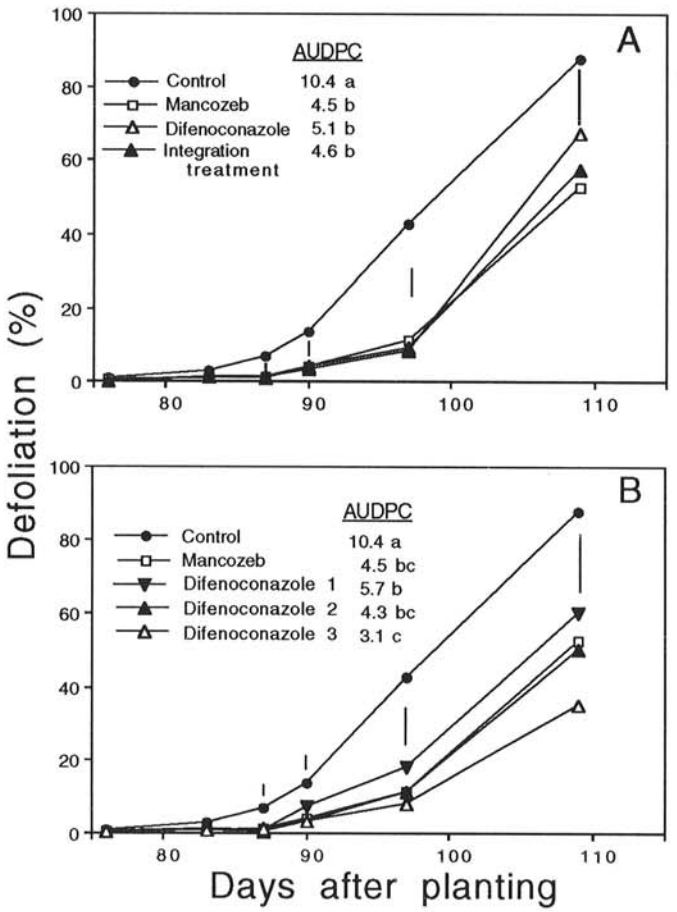

Fig. 4. Effects of various fungicide treatments on the suppression of *Alternaria solani* in potato in trial 5 (cultivar Alpha). **A,** Disease suppression achieved by various fungicide treatments. Mancozeb (at 2.4 kg a.i./ha) was applied on a 7-day schedule; difenoconazole(at 0.25 kg a.i./ha) was applied on a 14-day schedule; mancozeb and difenoconazole were applied in the integration treatment at variable intervals (Table 1). **B,** The contribution of a single application of difenoconazole within a 7-day treatment of mancozeb in suppressing *A. solani*. Difenoconazole was applied once 43, 65, or 86 days after planting (treatments are identified as difenoconazole-1, -2. and -3, respectively). Bars indicate the LSD for each sampling date at $P > 0.05$. Values of AUDPC (area under the disease progress curve) followed by different letters differ significantly ($P < 0.05$) according to Fisher's protected LSD test.

The hypothesis that it is preferable to apply a more effective fungicide control against *Alternaria* at later stages in the season, when host susceptibility increases, was examined by a computer simulation model and in the field. The contribution of the double-protectant treatment (in the model; Fig. 1) or of one difenoconazole spray (trial 6; Fig. 4A) was greater when the treatment was applied 85 days after planting during a season of 105 to 110 days (Figs. 1 and 4B). Accordingly, we concluded that these results corroborated the hypothesis. It is interesting to compare our results and conclusions with those reported in another system—potato late blight (8). Host response to *Phytophthora infestans*, the causal agent of late blight, does not change substantially with age. Experiments performed by means of a computer simulation model and in the field revealed that application of a mixture of systemic (metalaxyl) and protectant (mancozeb) fungicides during the middle of the season (50 to 70 days after planting) resulted in the best late blight suppression and the lowest yield loss. When applications were done later in the season (80 to 90 days after planting), control efficacy was reduced and yield loss increased. When the mixture was applied 2 weeks before vine kill, it had little or no effect on disease control (8).

The effectiveness of the integration treatment was evaluated in the field trials. Based on the disease progress curves and the AUDPC values, this treatment was at least as effective as the routine protectant and systemic treatments (with the exception of trial 5), although fewer sprays were applied (Figs. 2–4). Two crops (cotton and potatoes) and two species of *Alternaria* (*A. macrospora* and *A. solani*) were included in these trials. Based on the results of this study, it may be concluded that the concepts for integration may be applicable to other Alternaria pathosystems where host response changes with age. However, caution should be used in situations in which other pathogens, which are not influenced by the age of the host, threaten the crop as well. A good example is potato late blight. In areas where both early and late blights are important, implementation of the integration treatment described above is not recommended. The relatively long intervals between protectant sprays at the beginning of the season and the application of systemic fungicides toward the end of the season would not provide appropriate protection against late blight. Furthermore, tebuconazole and difenoconazole are not effective against *P. infestans*. In such cases, a revised integration treatment, targeted against both diseases, should be implemented. The development and field testing of such a treatment for potatoes is the subject of another study.

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