Anthracnose Development in Mixtures of Resistant and Susceptible Dry Bean Cultivars

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ABSTRACT

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Field experiments were conducted from 1992 to 1994 to characterize the effect of cultivar mixtures on development of bean anthracnose caused by *Colletotrichum lindemuthianum*. Three light-red kidney bean cultivars were combined in different proportions to achieve seven mixture treatments. Plots were inoculated by transplanting diseased spreader plants in the center of each plot. Disease incidence and severity were consistently lower in the mixtures containing 25 and 50% resistant cultivar, whereas a mixture with 10% resistant cultivar was less effective in controlling bean anthracnose. Disease progress curves for both incidence and severity were fit to four models (exponential, logistic, monomolecular, and Gompertz). The Gompertz model best described disease progress in all treatments. The rates of disease increase (dy/dt) were always lower for mixtures containing the resistant cultivar than for pure stands of the susceptible cultivars. The Gompertz infection rates (r) decreased as the proportion of the resistant cultivar in the mixtures increased and mixture efficacy values increased.

Common bean (*Phaseolus vulgaris* L.) is one of the most important legumes in the world. In some tropical areas, dry beans are produced by small farmers in mixtures of cultivars and in complex associations with other crop species, such as maize, bananas, yams, and sweet potatoes (1). Bean anthracnose, caused by *Colletotrichum lindemuthianum* (Sacc. & Magnus) Lams.-Scrib. (29), is a major disease of common beans in many regions of the world (29,42). Losses from bean anthracnose are attributed to poor germination of infected seeds, reduced yield and quality, and decreased value of the product (29,42). Several physiologic races of *C. lindemuthianum* have been identified (18,29), and cultivars with resistance to one or more races are available commercially. Breeding for anthracnose resistance is costly and time-consuming, and new races continue to develop that overcome the available resistant cultivars (18,35). Therefore, managing host and pathogen resistance is critical in bean-production areas of the world.

Plant disease epidemics are favored by genetically uniform crops grown on large areas of land (6,39). In contrast, a combination of genetically diverse crops grown together in the same field does not provide the uniform substrate needed by a pathogen to multiply to large populations. Diversity can be created by growing different crop species (intercrop) or cultivars of the same species carrying resistance to different races (crop mixtures) on the same land at the same time (38,41). The latter form of diversification is referred to as multilines or cultivar mixtures.

Cultivar mixtures have been recommended for management of many cereal diseases (17,24,25). It has been suggested that cultivar mixtures reduce disease by lowering the population of susceptible host plants, which results in a loss of effective inoculum (7,39). For a given proportion of susceptible plants, the mixture efficacy depends on several factors. Field experiments and computer simulation studies indicate that the size of individual plants, the spatial distribution of susceptible hosts, and the spatial distribution of inoculum have an important effect on mixture efficacy (10,20,23). These factors depend heavily on the steepness of the spore dispersal gradient (26). Steep gradients lead to greater autoinfections that result in disease intensification on susceptible plants and lower mixture efficacy (20). For this reason, it has been suggested that cultivar mixtures would not provide effective disease control for splash-dispersed pathogens (10,39) such as *C. lindemuthianum*. Practically, however, cultivar mixtures have been used extensively to control major bean diseases, such as angular leaf spot (*Phaeoisariopsis griseola*), anthracnose, and bacterial halo blight (*Pseudomonas syringae* pv. *phaseolicola*), in the African Great Lakes Region (1,13,30,33). In this region, local mixtures contain substantial proportions of cultivars with resistance to local races of *C. lindemuthianum* (33). Cultivar mixtures of beans were more stable and better buffered against disease than pure lines (13,14).

The objectives of our research were to quantitatively evaluate the effect of cultivar mixtures on bean anthracnose development and to determine which mathematical model best describes disease progress of bean anthracnose in cultivar mixtures. A preliminary report on portions of this study was published previously (28).

MATERIALS AND METHODS

Experimental design. In 1992, the trial was located on the Fruit and Vegetable Research Farm at the New York State Agricultural Experiment Station (NYSAES) in Geneva. Instruments used to collect weather data were located at the Climatological Reference Station operated by NYSAES in cooperation with the National Weather Service (latitude 42°53′N, longitude 77°02′W, elevation 187 m). The soil type was Lima silt loam with 0 to 3% slopes and 2 to 6% organic matter content. The field was used to produce alfalfa the previous year and was isolated from other bean fields. The treatments were arranged in a randomized complete block design with three replications. The distance between blocks and the distance between plots within a block were 6.1 and 2.7 m, respectively. Each plot was a rectangle 5.4 m wide (six rows) × 6.1 m long. The entire field was fertilized by broadcasting 337 kg of

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15-15-15 (N-P-K) per ha. The field was treated with the preemergence herbicide metolachlor (Dual 8E, 0.845 liters/ha) for weed control. The postemergence herbicide fomesafen (Reflex 2LC, 2.1 liters/ha) was applied without oil on the whole field to reduce the weed population before transplanting the inoculum source. Main plots were hand-weeded. Areas between plots were left unplanted and became weedy (*Ambrosia* spp.) as the season progressed.

Three light-red kidney bean cultivars, Ruddy, Redkloud, and Sacramento, were mixed in different proportions to achieve seven treatments. Cultivar Ruddy was resistant (R) to race β. Cultivars Redkloud and Sacramento, both susceptible to race β, were designated S_1 and S_2, respectively. The different proportions were as follows: 100 R; 50 R + 25 S_1 + 25 S_2; 25 R + 37.5 S_1 + 37.5 S_2; 10 R + 45 S_1 + 45 S_2; 50 S_1 + 50 S_2; 100 S_1; and 100 S_2. To produce mixtures, the relative seed proportion for each cultivar was determined by the weight of 100 seeds. Cultivar mixtures were hand-mixed in the laboratory prior to planting. Certified seed was planted mechanically on 29 June 1992 at a density of 26 to 30 plants per m.

The experiment was repeated in 1993 in another isolated field at the Fruit and Vegetable Research Farm. Both plot size and number of replications were increased compared to the previous year. Plots were increased to 6.3 × 6.3 m. The distance between plots was increased to 8.1 m. Fertilizer rate and plant density were similar to the previous season. The experimental plots were hand-planted on 28 May 1993. The areas between the plots and around the field were mechanically planted on 2 June 1993 to the snap bean cultivar Labrador, which is resistant to race β of *C. lindemuthianum*.

In 1994, the experimental design was similar to that of 1993 and was located in another isolated field. The plots were planted on 5 and 6 June 1994. The area between and around the plots was mechanically planted on 7 June 1994 to cultivar Labrador.

Source of inoculum. All experiments were inoculated with an isolate of *C. lindemuthianum* (race β) obtained in 1990 from cultivar Bush Blue Lake 47 in Geneva, NY. Reisolations were made from susceptible bean plants (cultivar California Dark Red Kidney) that were inoculated in the greenhouse to maintain a viable and pathogenic culture. To produce inoculum, mycelial fragments were grown on improved Mathur's medium (34) and incubated at 22 to 25°C for 14 to 20 days. Two-week-old greenhouse seedlings of susceptible cultivar Redkloud or Sacramento growing in 9 × 9-cm pots were thinned to five plants per pot and inoculated by atomizing a spore suspension (1.2×10^6 conidia per ml) on whole plants to runoff.

In 1992, five diseased plants were transplanted on 27 July in the center of each plot. Two weeks later, a second set of five plants was transplanted in each plot because many of the plants initially transplanted in the field died from the combined effect of high disease severity and environmental shock. In 1993, inoculated seedlings were incubated for 2 days in the greenhouse before transplanting to the center of each plot on 21 June. A second set was transplanted 2 weeks later to compensate for plants that had died. In 1994, to reduce the effect of environmental shock, 6-day-old seedlings were placed in a cold frame for 5 days before inoculation. The inoculated plants were incubated for 2 days in the greenhouse and transplanted on 24 June.

Disease assessment and harvest. In 1992, disease progression was monitored by assessments of all plants for each plot based on a severity scale of 0 to 5, where 0 = healthy, no apparent disease symptoms; 1 = flecks reflecting young lesions not yet sporulating; 2 = a few isolated lesions on the mid and occasionally on secondary veins of the leaf blade or on pods, covering about 3 to 6% of the surface area; 3 = several small lesions scattered on the mid and secondary veins, with collapse of surrounding leaf or pod tissue, covering about 6 to 12% of the surface area; 4 = numerous enlarged lesions scattered over the petiole and leaf blade or on pods, covering up to 25% of the surface area; and 5 = severe necrosis resulting from many large coalesced lesions on more than 25% of the surface area, accompanied by tissue collapse and plant death. Conversion of disease ratings into disease severity was accomplished by a midpoint value attributed to each severity class.

In 1993 and 1994, disease incidence and severity were evaluated every week on leaves and pods of flagged plants located at distances of 0.9, 2.7, 4.5, and 6.3 m from the inoculum source. The disease-assessment scale of 0 to 5 was used. Thirty-six plants, representing about 3% of the plants in each plot, were evaluated.

Data analysis. Each year disease progress curves were determined for each treatment. The area under the disease (incidence and severity) progress curves (AUDPC) were calculated by the midpoint rule between assessment dates (8). Means of disease incidence and severity and their standard errors were calculated from all the replicate plots of pure and mixed stands for each treatment and assessment date. Analysis of variance was performed on incidence, severity, and AUDPCs by SAS packages (31). Comparisons among means were made with Fisher's protected least significant difference.

For each treatment, the mixture efficacy was calculated as the relative difference between disease incidence or severity of the mixture and the susceptible pure stands (19). Measurements of mixture efficacy were based on incidence and severity assessments made at the end of the growing season (I_F and S_F, respectively) and on AUDPCs (I_A and S_A, respectively). Mixture efficacy was calculated as (20): $E_I = 1 - [I \text{ (mixture)}/I \text{ (mean of susceptible pure stands)}]$ and $E_S = 1 - [S \text{ (mixture)}/S \text{ (mean of susceptible pure stands)}]$. Mixture efficacy values were expressed as a percent by multiplying by 100.

Incidence and severity data on bean anthracnose disease progression also were fitted to exponential, logistic, monomolecular, and Gompertz models by linear regressions (8,22). Transformed data fit to these models were tested for goodness-of-fit. In addi-

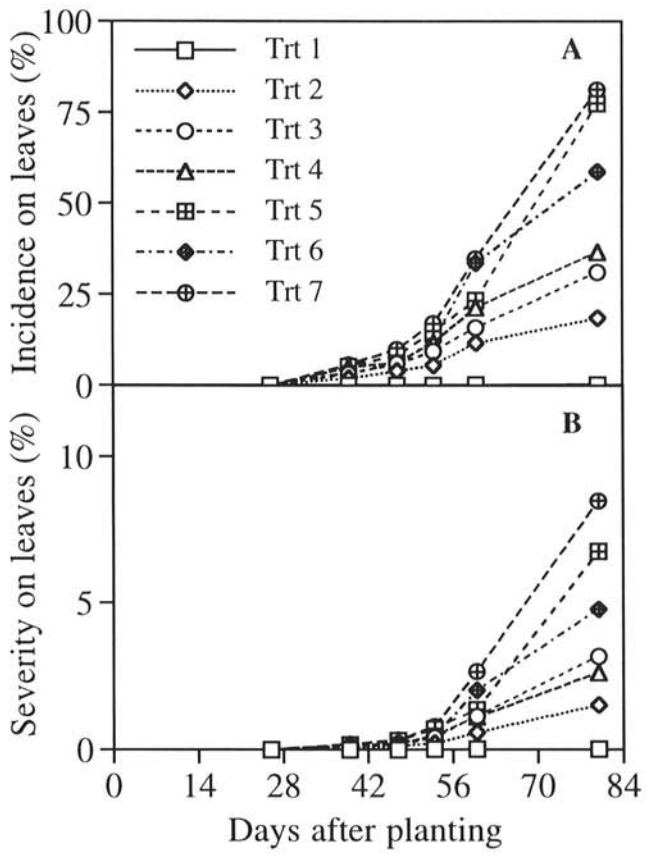

Fig. 1. Disease progress curves for **A,** incidence and **B,** severity of bean anthracnose on leaves in cultivar mixtures in 1992. Treatments (Trt) were 1 = 100 R; 2 = 50 R + 25 S_1 + 25 S_2; 3 = 25 R + 37.5 S_1 + 37.5 S_2; 4 = 10 R + 45 S_1 + 45 S_2; 5 = 50 S_1 + 50 S_2; 6 = 100 S_1; and 7 = 100 S_2. R = resistant cv. Ruddy, S_1 = susceptible cv. Redkloud, and S_2 = susceptible cv. Sacramento.

tion, predicted transformed values of disease were back-transformed to obtain predicted y values. Observed y values were regressed on predicted y values, and coefficients of determination (R^2) and mean square errors (MSE) were compared among models. Higher R^2, lower MSE values, and subjective evaluation of plots of residuals were used to determine the most appropriate model for describing the data.

RESULTS

Anthracnose development. In 1992, anthracnose incidence on leaves was low (0 to 5.7%) in all treatments for the first 3 weeks after transplanting of spreader plants. Disease incidence on leaves increased rapidly near the end of the growing season (Fig. 1A). The resistant cultivar, Ruddy, did not develop disease, as was expected. Final disease incidence in mixtures with 50, 25, and 10% resistant cultivar were significantly ($P = 0.0001$) lower than pure stands of susceptible cultivars Redkloud and Sacramento. Final disease incidence on Redkloud was less than on Sacramento. Disease incidence in the mixture containing equal proportions of Redkloud and Sacramento was intermediate to disease incidence observed in the pure stands of these cultivars. Similar results were observed when the disease progress curves based on anthracnose severity on leaves were used (Fig. 1B). Mixtures with 50, 25, and 10% resistant cultivar had significantly ($P = 0.0001$) lower final disease severities than the pure stands of the susceptible cultivars. Cultivar Sacramento had the highest final disease severity.

In 1993 and 1994, the disease progress curves of bean anthracnose incidence and severity on leaves and pods were similar to those of 1992 (data not shown). The mixtures with 50 and 25% resistant cultivar developed less disease than other treatments, whereas the mixture with 10% resistant cultivar was less effective in reducing anthracnose development. Disease incidence was greater in pure stands of the susceptible cultivars than in mixtures containing the resistant cultivar.

When AUDPCs calculated from disease incidence progress curves were compared in 1992, the 25 and 50% mixtures of susceptible cultivars with the resistant cultivar had significantly ($P = 0.0001$) lower AUDPCs than the pure stands of the susceptible cultivars (Fig. 2A). The results were similar in 1993 and 1994. In general, the AUDPCs of leaves increased as the proportion of susceptible plants increased in the mixtures. The AUDPC of the mixture with 10% resistant cultivar was not significantly less than that of pure stands of the susceptible cultivars. Similar results were obtained for AUDPC of disease incidence on pods (Fig. 2B).

Mixture efficacy in reducing anthracnose development. In 1992, the effectiveness of cultivar mixtures in reducing bean anthracnose development on leaves decreased as the proportion of susceptible cultivar increased in the mixture (Fig. 3A). Based on disease incidence (I_F), mixtures of susceptible cultivars with 50, 25, and 10% resistant cultivar were 74, 56, and 48% less diseased, respectively, than susceptible pure stands. Mixture efficacy values were 77, 52, and 60% when they were calculated based on disease severity (S_F) in the mixtures with 50, 25, and 10% resistant cultivar, respectively. The results were consistent when AUDPCs (I_A and S_A) were used. All mixture efficacy curves were located to the left of the diagonal (from the lower left corner to upper right corner in Figure 3A).

Results were similar in 1993. Mixture efficacy values calculated for disease incidence (I_F) of mixtures of susceptible cultivars with 50, 25, and 10% resistant cultivar were 57, 42, and 29%, respectively (Fig. 3B). A similar trend was observed with the other three variables (S_F, I_A, and S_A). Mixture efficacy curves were located slightly to the left of the diagonal in Figure 3B.

In 1994, mixtures of susceptible cultivars with 50, 25, and 10% resistant cultivar had 52, 29, and 8% less disease incidence (I_F), respectively, than susceptible pure stands (Fig. 3C). Mixture efficacy curves were located on the diagonal, which indicates that overall disease in the mixture was not less than expected based on the frequency of the resistant cultivar. Similar efficacy values were observed when the variables were calculated based on pod data in 1993 and 1994 (data not shown).

Model comparison. During the 3 years of study, the Gompertz model provided the best fit to 54 of 62 (87%) of the disease progress curves (Table 1). The logistic model provided an acceptable fit to 45% of the total curves analyzed, and the monomolecular and the exponential models gave acceptable fits to 35 and 32%, respectively, of the total curves. In 1992, the rates of disease development *(dy/dt)* in susceptible pure stands were always greater than those of the mixtures with the resistant cultivar (Fig. 4A). In susceptible pure stands and the mixture of susceptible cultivars (treatments 5, 6, and 7), *dy/dt* increased until it reached a maxi-

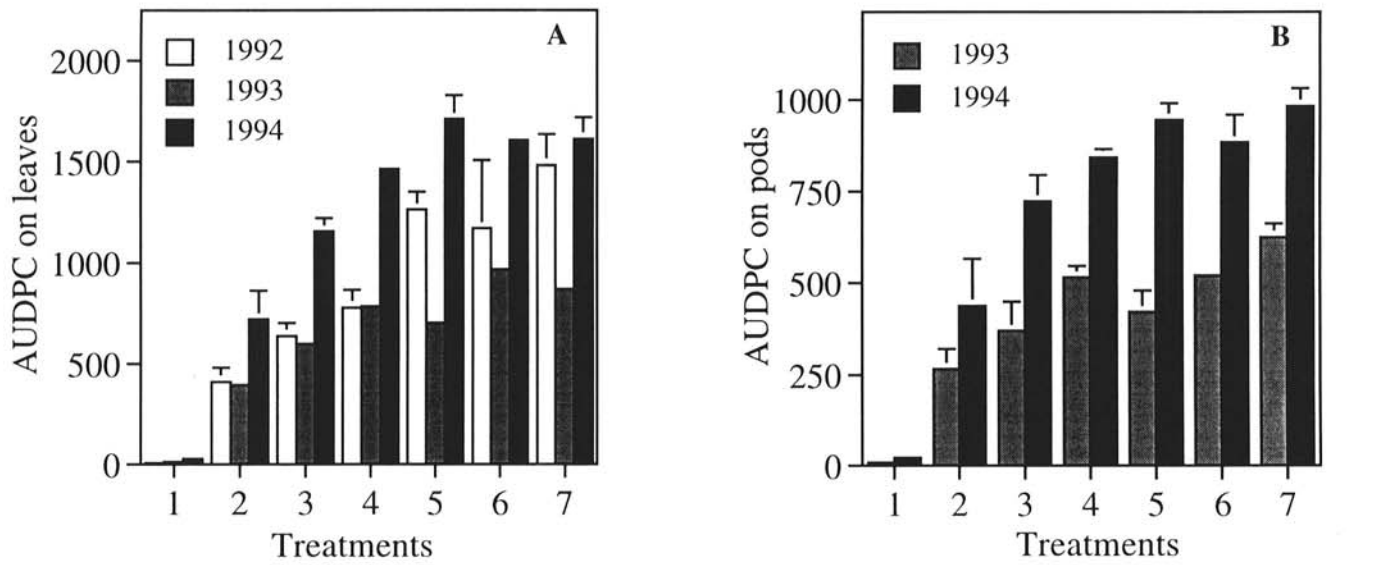

Fig. 2. Area under the anthracnose disease incidence progress curves (AUDPC) on **A,** leaves and **B,** pods for different bean cultivar mixtures grown in the field in 1992, 1993, and 1994. Treatments were 1 = 100 R; 2 = 50 R + 25 S_1 + 25 S_2; 3 = 25 R + 37.5 S_1 + 37.5 S_2; 4 = 10 R + 45 S_1 + 45 S_2; 5 = 50 S_1 + 50 S_2; 6 = 100 S_1; and 7 = 100 S_2. R = resistant cv. Ruddy, S_1 = susceptible cv. Redkloud, and S_2 = susceptible cv. Sacramento. Fisher's protected least significant difference (LSD) values for leaves were 448.13, 150.4, and 339.5 in 1992, 1993, and 1994, respectively. LSD values for pods were 138.2 and 222.1 in 1993 and 1994 ($P = 0.05$), respectively. Bars represent the standard error of the mean.

mum and then decreased later in the season. This trend was not observed in the mixtures containing the resistant cultivar, in which *dy/dt* continually increased until the end of the season. A similar trend was observed in 1993 (Fig. 4B) and 1994 (Fig. 4C). The rates of disease development of the susceptible cultivars grown in pure stands were greater than the mixtures with resistant cultivar Ruddy. However, contrary to what was observed in 1992, *dy/dt* of most treatments reached a maximum and then decreased later in the season.

The resistant cultivar significantly reduced the Gompertz infection rates (r_G) of disease increase relative to the pure stands of the susceptible cultivars (Fig. 5). The Gompertz infection rates, whether based on disease incidence on leaves or pods, consistently increased as the proportion of the susceptible cultivar increased. Pods always exhibited higher infection rates (r_G) than leaves. The infection rates (r_G) of the mixtures with 50 and 25% resistant cultivar were lower than those of the pure stands, but the differences were not significant. Results were similar in 1993 and 1994.

DISCUSSION

Based on the results from these trials, we conclude that cultivar mixtures can reduce development of bean anthracnose in the field. Anthracnose incidence and severity were consistently lower in mixtures containing 25% or more resistant cultivar (Ruddy). The AUDPCs in mixtures with 25% cultivar Ruddy were always lower than those of pure stands of the susceptible cultivars. More importantly, the calculated mixture efficacy values can be interpreted as indicating a favorable effect of mixtures that extends beyond the frequency of resistant individuals in the plant population. This is evidenced by the location of the mixture efficacy values to the left of the diagonal in Figure 3, which is the expected amount of disease based solely on the frequency of resistant plants in the plot (7). These results support previous research on other crops and diseases in which cultivar mixtures restricted the spread of disease (6,15,16,30,39).

The effectiveness of mixtures in restricting epidemic development has been spectacular in small grains infected by rusts and powdery mildews (10,21,25,39). The effect was temporary in controlling *Cercospora apii* in celery (2) or lost after an initial period of controlling swede powdery mildew (*Brassica napus*) (32). It has been stated that cultivar mixtures may have no effect on splash-dispersed pathogens (39) because mixtures do not have an effect on autoinfection. Mixtures gave variable results in controlling anthracnose caused by *C. gloeosporioides* on pasture legume (*Stylosanthes scabra)* (9). Certain mixtures had lower disease severity and AUDPC than the pure stands of the components, whereas others had disease levels similar to the pure stands. The difference between these results and ours may reside in the level of disease severity and the nature of the two crops. Very low disease severity values (<0.4%) were obtained on pasture legumes compared to levels on beans in our trials (1.5 to 8.5%). Furthermore, pasture cropping systems have higher plant populations than bean systems, which are row-cropped. Our results were similar to those obtained on spring wheat infected by *Septoria nodorum* (15,16) and bean angular leaf spot caused by *Phaeoisariopsis griseola* (30). In a splash-dispersed pathogen system such as *Septoria nodorum*, epidemic development can be retarded in a heterogeneous plant population (15,16). Similar results were found in the angular leaf spot system, where the disease was reduced through supplementation of local mixtures with 25% resistant cultivar (30). Halo blight of bean caused by *Pseudomonas syringae* pv. *phaseolicola* also was significantly reduced in mixtures containing 25% or more resistant cultivar (13).

TABLE 1. Frequency of models providing an acceptable fit to disease progress curves for anthracnose incidence and severity in bean cultivar mixtures grown in the field in Geneva, NY (1992 to 1994)

Year	No. of epidemics	Data type	Disease progress model[z]			
			E	L	M	G
1992	14	Incidence on leaves	1	3	1	6
		Severity on leaves	2	2	2	7
1993	24	Incidence on leaves	4	5	2	5
		Severity on leaves	5	5	1	6
		Incidence on pods	0	1	0	6
		Severity on pods	1	1	2	3
1994	24	Incidence on leaves	2	3	5	5
		Severity on leaves	1	1	5	5
		Incidence on pods	0	3	3	5
		Severity on pods	4	4	1	6
Total	62		20	28	22	54
%			32	45	35	87

[z] E = exponential; L = logistic; M = monomolecular; and G = Gompertz.

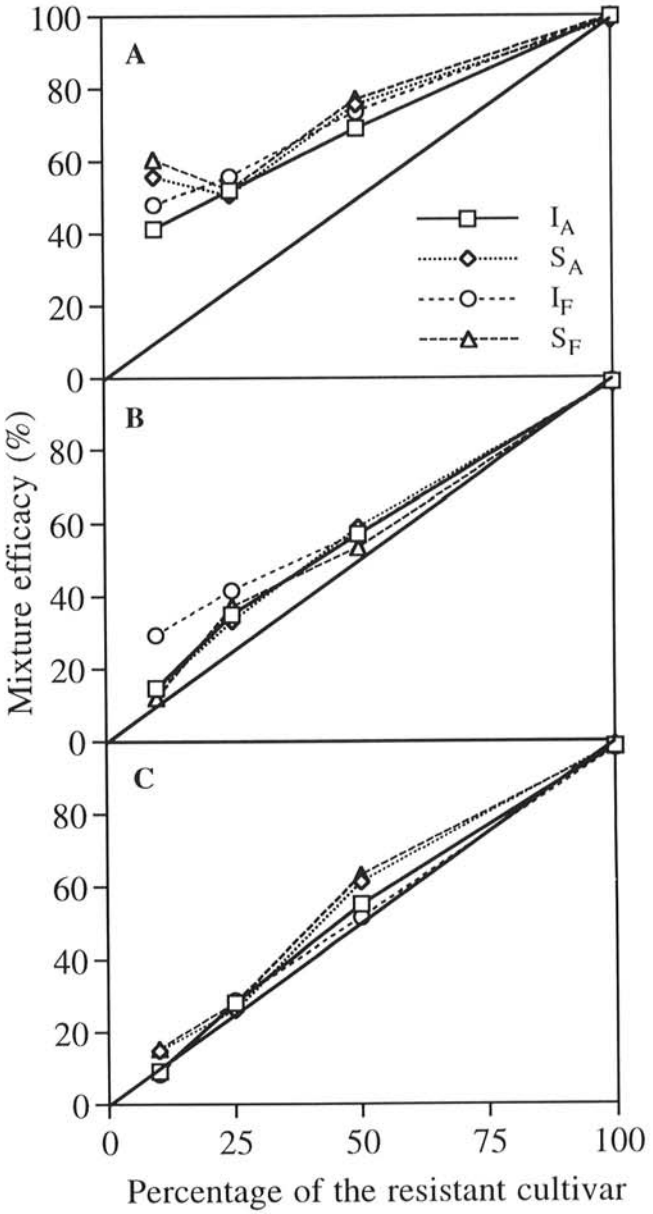

Fig. 3. Effect of the proportion of a resistant bean cultivar grown in mixtures with a susceptible cultivar on anthracnose development. Mixture efficacy is the relative amount of disease on a susceptible cultivar in a mixture when compared to a pure stand of the susceptible cultivar. Data shown are for final leaf disease incidence (I_F), disease severity (S_F), and area under the disease incidence (I_A) and severity (S_A) progress curves in **A,** 1992, **B,** 1993, and **C,** 1994. The solid diagonal line indicates expected values if the mixture effect is a function of the frequency of resistant plants in the mixture.

In our study, the Gompertz model provided the best fit of the disease progress curves constructed for incidence and severity and allowed comparison of epidemic rates between different mixtures and pure stands. When the Gompertz and the logistic models were compared, the logistic model overestimated disease severity early and late in disease progress (3,12). In some situations, the monomolecular model provided a good fit of the disease progress curves. However, the amount of inoculum of *C. lindemuthianum* that is available changes over time during the growing season, and thus, the biological assumptions for a monocyclic disease are violated, because secondary infections are expected with *C. lindemuthianum*. The logistic model assumes that all infection sites are equally susceptible; however, for bean anthracnose, plant tissue becomes more resistant as plants mature. It has been suggested that the Gompertz model is more appropriate for diseases in which infection sites become less susceptible with age (12). For these reasons, we consider the Gompertz model to be more appropriate for bean anthracnose than the logistic model.

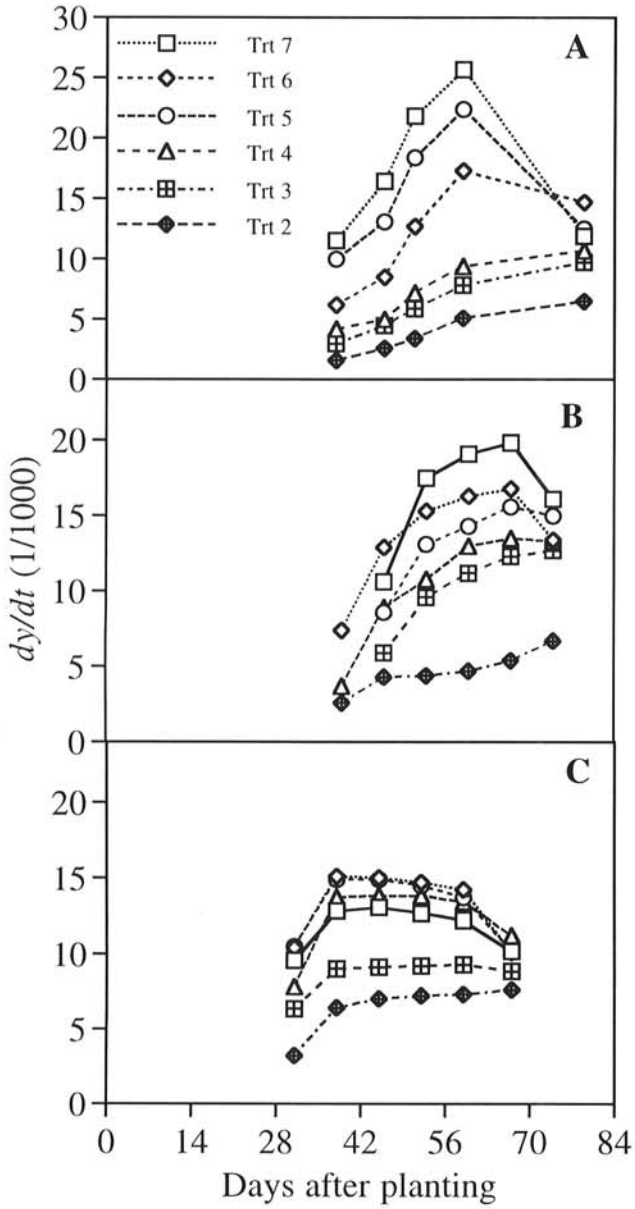

Fig. 4. Rate of bean anthracnose development over time *(dy/dt)* for incidence on leaves in **A,** 1992, **B,** 1993, and **C,** 1994. Treatments (Trt) were 2 = 50 R + 25 S_1 + 25 S_2; 3 = 25 R + 37.5 S_1 + 37.5 S_2; 4 = 10 R + 45 S_1 + 45 S_2; 5 = 50 S_1 + 50 S_2; 6 = 100 S_1; and 7 = 100 S_2. R = resistant cv. Ruddy, S_1 = susceptible cv. Redkloud, and S_2 = susceptible cv. Sacramento.

Infection rate data obtained in this study did not sufficiently discriminate between treatments. However, the general trend was that bean mixtures had lower infection rates than the susceptible cultivars grown in pure stands. Rates of disease increase are very sensitive to environment, host, pathogen, and fungicide applications (37). Every contribution, whatever its source, is pooled into the rate estimate.

Biological mechanisms that reduce disease incidence and severity in cultivar mixtures were reviewed by Browning and Frey (6) and Wolfe (39). It is believed that resistant plants in cultivar mixtures not only reduce the effective initial inoculum, but also impede the dispersal of the pathogen. The susceptible individuals receive some protection from disease buildup because fewer spores are available for infection (2). Because there was no possibility of cross-protection phenomena operating in these single-pathogen experiments, it is likely that the resistant plants were interfering in some way with the transmission of inoculum. The resistant components act as barriers to the spread of the pathogen (6,39,40). In computer-simulated epidemics, the effectiveness of mixtures in controlling disease declined with increasing percentage of susceptible plants in the mixtures (26). Also, the effectiveness of disease control in cultivar mixtures is negatively correlated with the genotype unit size (23,24,27), which is the ground area occupied by an independent, genetically homogeneous unit of plants. The most effective and ideal mixture would be one obtained by random mixing, which is technically and economically more attractive than systematic seed placement (36).

The percentage of resistant cultivar required to protect a mixed population against plant pathogens is not well defined (25). Evidence from indigenous ecosystems indicates that when 30% of the population is resistant, the population will be sufficiently protected if this resistance is back-stopped by general resistance (4). The 30% figure was supported by studies conducted with oat crown rust in agricultural systems (5). Jensen and Kent (17) suggested that 40% resistance is adequate to protect a population of plants. However, a level of resistance that is adequate to protect a plant population in one environment may be inadequate for a different

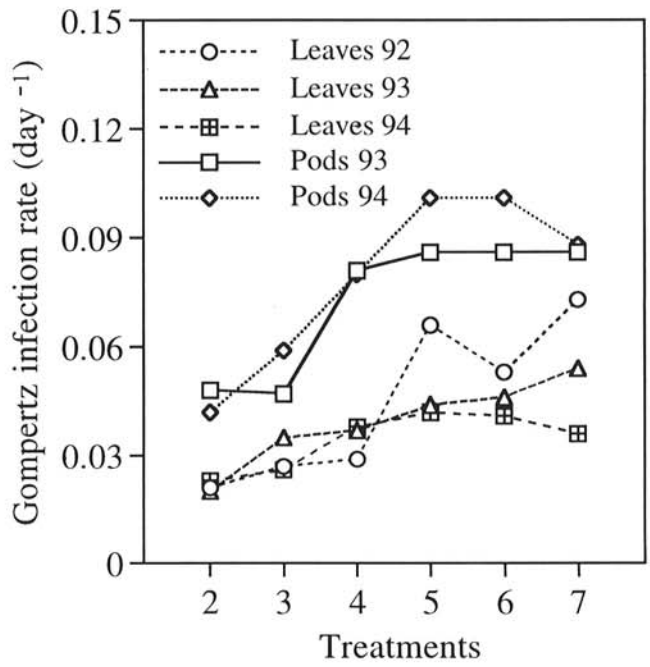

Fig. 5. Values of Gompertz infection rate parameter (r_G) estimated for anthracnose incidence on leaves and pods in bean cultivar mixtures in 1992, 1993, and 1994. Treatments were 2 = 50 R + 25 S_1 + 25 S_2; 3 = 25 R + 37.5 S_1 + 37.5 S_2; 4 = 10 R + 45 S_1 + 45 S_2; 5 = 50 S_1 + 50 S_2; 6 = 100 S_1; and 7 = 100 S_2. R = resistant cv. Ruddy, S_1 = susceptible cv. Redkloud, and S_2 = susceptible cv. Sacramento.

crop or in an environment more conducive for disease development (11). Furthermore, variation would likely occur between different host-pathogen systems. Our results consistently showed that 50% mixtures with a resistant bean cultivar were effective in reducing anthracnose development in the field. Twenty-five percent resistant cultivar gave a satisfactory level of protection, whereas 10% provided variable results.

Cultivar mixtures have been extensively used in tropical agriculture systems to protect crops against arthropod pests, diseases, and many other environmental stresses. In the central African highlands where beans are a critical food source, many farmers prefer cultivar mixtures over a single cultivar (33). In our 3-year study, cultivar mixtures consistently reduced the development of bean anthracnose. Where bean anthracnose is a recurring problem, cultivar mixtures may provide an effective, inexpensive, sustainable, and fungicide-free disease management strategy. Further research is needed to determine the benefits of cultivar mixtures when multiple races or pests are present.

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