Analysis of Factors Affecting Disease Increase and Spread in Mixtures of Immune and Susceptible Plants in Computer-Simulated Epidemics

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ABSTRACT

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A modification of the simulation model EPIMUL was used to study the effects of host genotype unit area (ground area occupied by an independent, genetically homogeneous unit of a host population), spatial distribution of initial disease, percentage of susceptible plants, rate of disease increase, and steepness of pathogen dispersal gradients on epidemic development of polycyclic, foliar disease in mixtures of immune and susceptible plants. The effectiveness of the mixtures for disease control decreased with increasing genotype unit area and with increasing steepness of the dispersal gradient. The effects of genotype unit area and dispersal gradient steepness on mixture efficacy for disease control were greater when initial disease was distributed uniformly than in epidemics with a single focus of initial disease. Genotype unit area had the greatest effect on mixture efficacy for epidemics with intermediate gradient steepnesses. There was usually a smaller effect of genotype unit area on mixture efficacy when the dispersal gradient was steep and there was very little effect of genotype unit area on mixture efficacy when the gradient was very shallow. The effectiveness of the mixtures in controlling disease declined with increasing percentage of susceptible plants in the mixtures. However, the loss of mixture efficacy with increasing genotype unit area was less in mixtures with a high proportion of susceptible plants than in mixtures with a lower proportion. The mixtures were usually less effective in controlling epidemics with high rates of multiplication per lesion than in controlling epidemics with low rates. For epidemics with a very low rate of multiplication per lesion, the mixtures were less effective in controlling disease when the dispersal gradient was very shallow than when the gradients were of intermediate steepness.

Additional key words: genetic diversity, multiline cultivars, spore dispersal.

The effectiveness of host mixtures for controlling plant diseases has been documented for some host-pathogen systems (3,7,10,19,28), most of which are rusts or powdery mildews of small grains. In addition, most studies of epidemic development in host mixtures have been with random mixtures of plants. There is little information concerning the effectiveness of other strategies for attaining diversity such as intercropping and interfield diversification, although interfield diversification is used for small-grain production in the United Kingdom (6,23).

There are several factors that may determine the effectiveness of host mixtures for controlling plant disease, and some of these factors have been studied theoretically. In simulation studies, the effectiveness of host mixtures declined with increasing steepness of the pathogen's dispersal gradient (13,14). Similarly, Barrett (1) and Barrett and Wolfe (2) found that increasing the proportion of autoinfection (infections on a genotype unit resulting from propagules produced on that same genotype unit [24]) in theoretical mixtures resulted in a decrease in the effectiveness of the mixtures. The proportion of autoinfection is a function of the steepness of dispersal gradients.

The effect of genotype unit area (ground area occupied by an independent, genetically homogeneous unit of a host population [18]) on mixture efficacy has been studied in both field and computerized simulation studies. Barrett and Wolfe (2) changed genotype unit area in a mixture of three barley (*Hordeum vulgare* L.) cultivars by altering sowing density. Their results suggested that the effectiveness of the mixture for the control of powdery mildew (induced by *Erysiphe graminis* DC. f. sp. *hordei* Marchal) declined with increasing genotype unit area. By altering the spatial arrangement of near-isogenic oat (*Avena sativa* L.) lines, Mundt and Browning (18) found that increasing the genotype unit area from 0.003 to 0.84 m^2 had no significant effect on the efficacy of oat multilines for the control of crown rust (induced by *Puccinia coronata* Cda. var. *avenae* Fraser & Ledingham) when epidemics were initiated with a single disease focus per plot. However, subsequent field (21) and computerized simulation (22) studies indicated that increasing the genotype unit area greatly reduces the effectiveness of oat mixtures for crown rust control if epidemics are initiated with several to many foci per plot but not if epidemics are initiated from a single focus per plot.

Other factors influencing the efficacy of host mixtures for disease control will probably interact to determine the effectiveness of a mixture. For example, results from computerized simulation studies of epidemic development in mixtures of resistant and susceptible plants suggest that dispersal gradient steepness has a stronger influence on mixture efficacy when the proportion of susceptible plants is low than when it is high (13).

The purpose of our research was to use a computerized simulation model to study the effects of host genotype unit area, spatial distribution of initial disease, steepness of pathogen dispersal gradients, rate of disease increase, and percentage of susceptible plants on epidemic development of foliar disease in mixtures of immune and susceptible plants.

MATERIALS AND METHODS

Simulation model. The computerized simulation model was a modification of EPIMUL (12) and has been described previously (22).

Treatment design. For mixtures of 1:3 and 1:1 susceptible/immune plants, there was a factorial arrangement of treatments that included five degrees of dispersal gradient steepness, five genotype unit areas, and two spatial distributions of initial disease.

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The five degrees of gradient steepness were b (slope) of the modified Gregory model (20) = 0.5, 1.0, 2.0, 3.0, and 4.0. The five genotype unit areas were 0.0025, 0.0225, 0.0900, 0.2500, and 0.5625 m^2 and were obtained by aggregating host compartments into units of 1, 9, 36, 100, and 225 compartments of the same genotype, respectively (Fig. 1). To obtain the first spatial distribution of initial disease, a single spore was placed in each of the 3,600 compartments of each plot (general epidemics), and to obtain the other spatial distribution of initial disease, 900 spores were placed in each of the four central compartments of each plot (focal epidemics). For the 1:3 mixtures, there were four rates of multiplication per lesion for each combination of gradient steepness and genotype unit area (daily multiplication factor (DMFR) = 0.3, 1.5, 7.5, and 37.5 lesions per lesion per day). The DMFR is the number of progeny lesions produced per parent lesion per day of the infectious period, which is equivalent to Vanderplank's (27) corrected basic infection rate. Because of cost limitations, only epidemics with DMFR = 7.5 were simulated for the 1:1 mixtures. For both the 1:3 and 1:1 mixtures and both distributions of initial disease, four additional dispersal gradients (b = 1.50, 2.25, 2.50, and 2.75) were tested using a DMFR = 7.5. Simulations in pure-line susceptible populations were performed for each combination of gradient steepness and rate of multiplication per lesion that was used for the mixtures.

For 1:7 mixtures of susceptible/immune plants, the same simulations were performed as for the 1:3 mixtures, except genotype unit areas of 0.0025, 0.0225, 0.0625, and 0.5625 m^2 were used (Fig. 1); this was necessary because with a 1:7 ratio of susceptible/immune plants, the 0.0900- and 0.2500-m^2 genotype units would not fit in the plots an even number of times. Only 450 spores per initially inoculated compartment were used for the focal inoculations of 1:7 mixtures to give the same number of initial infections per susceptible compartment as in the general epidemics with one initial spore per compartment. This was necessary because when only four compartments at the center of each plot are inoculated, at least one must be susceptible to initiate the epidemics.

For all simulations, epidemics were studied in blocks of 3,600 compartments where each compartment can be considered a plant, or when compartments of the same genotype are aggregated, each compartment can be considered either a plant or a subsection of a larger plant. Variables held constant in all simulations were: length of the side of a compartment = 0.05 m, length of latent period = 7 days, length of infectious period = 14 days, area of a lesion = 4.00 mm^2, leaf area index = 3.00, c (truncation factor) of the modified Gregory model (20) = 0.03 m, length of epidemic = 61 days, and frequency of data output = 1 day.

Values of b used in the simulations were chosen to cover the range of gradients commonly observed with plant pathogens and provided dispersal gradients that predict that the number of spores deposited per unit area declines to 10% of that deposited in the source compartment at distances of 2.97, 0.27, 0.11, 0.082, 0.065, 0.053, 0.045, 0.035, and 0.023 m from the source for b values of 0.50, 1.00, 1.50, 1.75, 2.00, 2.25, 2.50, 3.00, and 4.00, respectively. DMFRs were chosen to give logarithmic infection rates (27) of about 0.12, 0.25, 0.40, and 0.60 per unit per day for DMFRs of 0.3, 1.5, 7.5, and 37.5, respectively, in an infinitely large, pure-line susceptible host population.

Data analysis. Epidemics were analyzed by calculating the area under the disease progress curve (ADPC) for the susceptible genotype in each treatment by the equation

$$\text{ADPC} = \left\{ \sum_{i=1}^{n} y_i - [(y_1 + y_n)/2] \right\} \times f,$$

where y_i = percentage of disease severity of the susceptible genotype at the ith observation, y_1 and y_n = first and last observations of disease severity, respectively, f = number of days between observations of disease severity, and n = the total number of observations. This equation gives the same results as the equation given by Shaner and Finney (25) when all observations are equally spaced in time.

For each simulation, the ADPC was calculated from day 7 (the

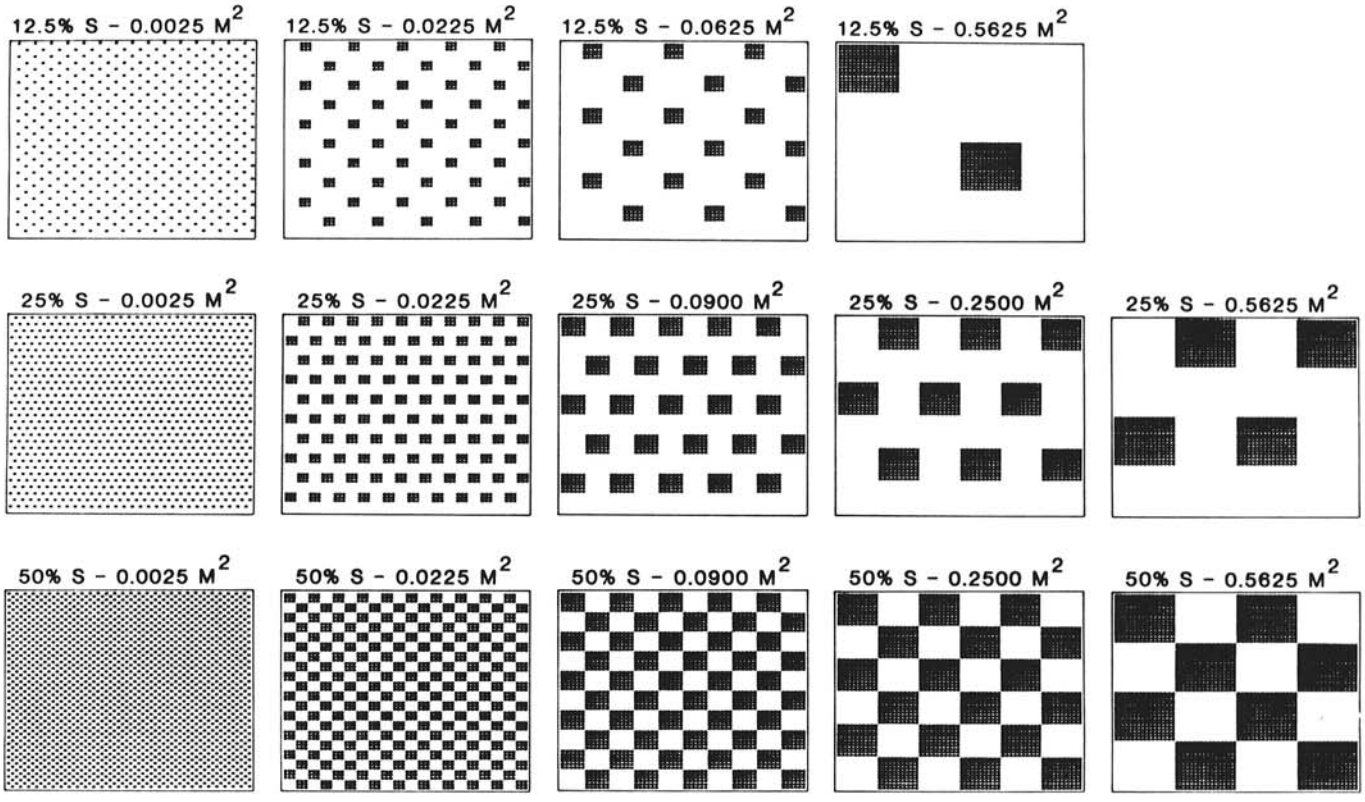

Fig. 1. Arrangements of host genotypes in computer-simulated epidemics in mixtures of immune and susceptible plants with 12.5, 25, or 50% susceptible plants and with genotype unit areas of 0.0025, 0.0225, 0.0625, 0.0900, 0.2500, or 0.5625 m^2. Each large block represents one simulated plot of 3,600 host compartments. Each small block represents one susceptible compartment, and blank spaces represent immune compartments.

first day disease appeared) until the day 90% disease severity was reached in the pure-line susceptible check with the same dispersal gradient steepness and DMFR. For epidemics with DMFRs of 7.5 and 37.5, the ADPC was calculated from daily outputs of disease severity. For epidemics with daily multiplication factors of 0.3 and 1.5, the ADPC was calculated from disease severity ratings at 3-day intervals. For all simulations, the relative ADPC (RADPC) was calculated by dividing the ADPC for susceptible plants by the ADPC for the pure-line susceptible check with the same DMFR and dispersal gradient steepness. In mixtures, the ADPC is for susceptible plants only, so a RADPC of 0.50 for a 1:3 mixture of susceptible/immune plants means that the ADPC for susceptible plants in mixtures was reduced to 50% of that in the pure-line susceptible check; the RADPC calculated over all plants in the mixtures would be $0.25 \times 50\% = 12.5\%$ of that in the pure-line check.

RESULTS

Because the number of spores dispersed out of simulated plots depended on the steepness of the dispersal gradient, rates of disease increase varied among epidemics with different values of b, even when the DMFR was the same. For example, for the pure-line susceptible plots with DMFR = 0.3, apparent infection rates (27) were calculated from disease severity ratings on 7-day intervals from 10 to 59 days after inoculation (the midpoints of the second to the eighth latent periods). Infection rates were 0.043, 0.057, 0.093, 0.11, and 0.12 for simulations with $b = 0.50$, 1.00, 2.00, 3.00, and 4.00, respectively.

In nearly every instance, the effectiveness of the mixtures declined with increasing steepness of the dispersal gradient (Figs. 2–5). The only exceptions were for epidemics with DMFR = 0.3 and with very shallow dispersal gradients (Fig. 2). With DMFR =

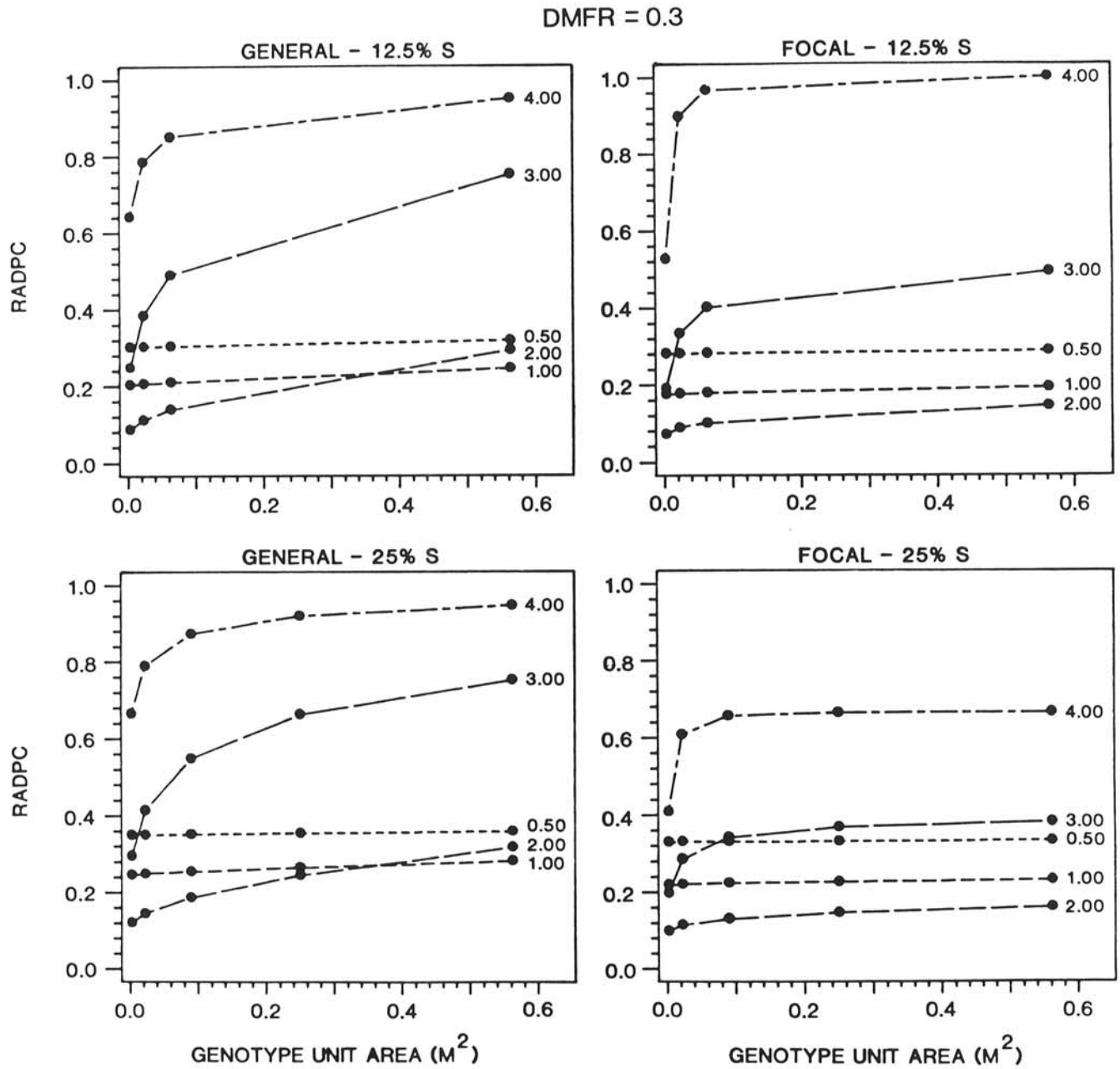

Fig. 2. Effects of host genotype unit area and steepness of pathogen dispersal gradients on the relative area under the disease progress curve (RADPC) for the susceptible genotype in computer-simulated epidemics of polycyclic, foliar disease in 1:7 (12.5% S) and 1:3 (25% S) mixtures of susceptible/immune plants when the daily multiplication factor (number of lesions produced per lesion per day of the infectious period in an infinitely large, susceptible host population) was 0.3. Mixtures with different genotype unit areas were obtained by aggregating host compartments of the same genotype into units (see Fig. 1). General epidemics were initiated with one spore in each of the 3,600 compartments in each plot. Focal epidemics were initiated with 900 spores in each of the four compartments at the center of each plot. Numbers on the right side of the figures are b values of the modified Gregory model (20), which was used to simulate spore dispersal; the larger the value of b the steeper the dispersal gradient.

0.3, the mixtures were less effective with $b = 0.50$ than $b = 1.00$, and for all but the largest genotype unit area in the general epidemics, the mixtures were less effective with $b = 1.00$ than with $b = 2.00$. The general effect of decreasing mixture efficacy with increasing gradient steepness can be seen most clearly in Figure 6. The loss of mixture efficacy was greatest at values of b between 1.5 and 3.0 and was greater for larger than for smaller genotype unit areas.

In all instances, the effectiveness of the mixtures declined with increasing genotype unit area, but this decline was greatest for epidemics with values of b between 1.50 and 3.00 (Figs. 2–6). There was a lesser effect of genotype unit area on mixture efficacy with the steepest gradient ($b = 4.00$) and very little effect with the most shallow gradients ($b = 0.50$ and 1.00). The decline in mixture efficacy with increasing genotype unit area was more pronounced for general than for focal epidemics (except with $b = 4.00$ and 12.5% susceptible plants), for epidemics with a low percentage of susceptible plants than for mixtures with a higher percentage, and for epidemics with a small DMFR than for epidemics with a large DMFR.

Mixtures with 12.5% susceptible plants were always more effective than mixtures with 25% susceptible plants for general epidemics and were usually more effective for focal epidemics (Figs. 2–5). Exceptions for focal epidemics were those with steep infection gradients of $b = 4.00$ or 3.00 and with the largest genotype unit area. For focal epidemics with DMFR = 0.3, mixtures with 12.5% susceptible plants were less effective than those with 25% susceptible plants at all genotype unit areas tested when $b = 4.0$ and for most genotype unit areas when $b = 3.0$. These exceptions to the general rule of greater mixture effectiveness with lower proportions of susceptible plants may be artifacts of the simulations and will be discussed later.

For epidemics with DMFR = 7.5, for which we also simulated mixtures with 50% susceptible plants, increasing the percentage of susceptible plants from 25 to 50% had a greater effect on mixture efficacy than did increasing the percentage from 12.5 to 25%. For general epidemics with steep gradients and large genotype unit areas, there was very little difference in the efficacy of mixtures with 12.5, 25, or 50% susceptible plants.

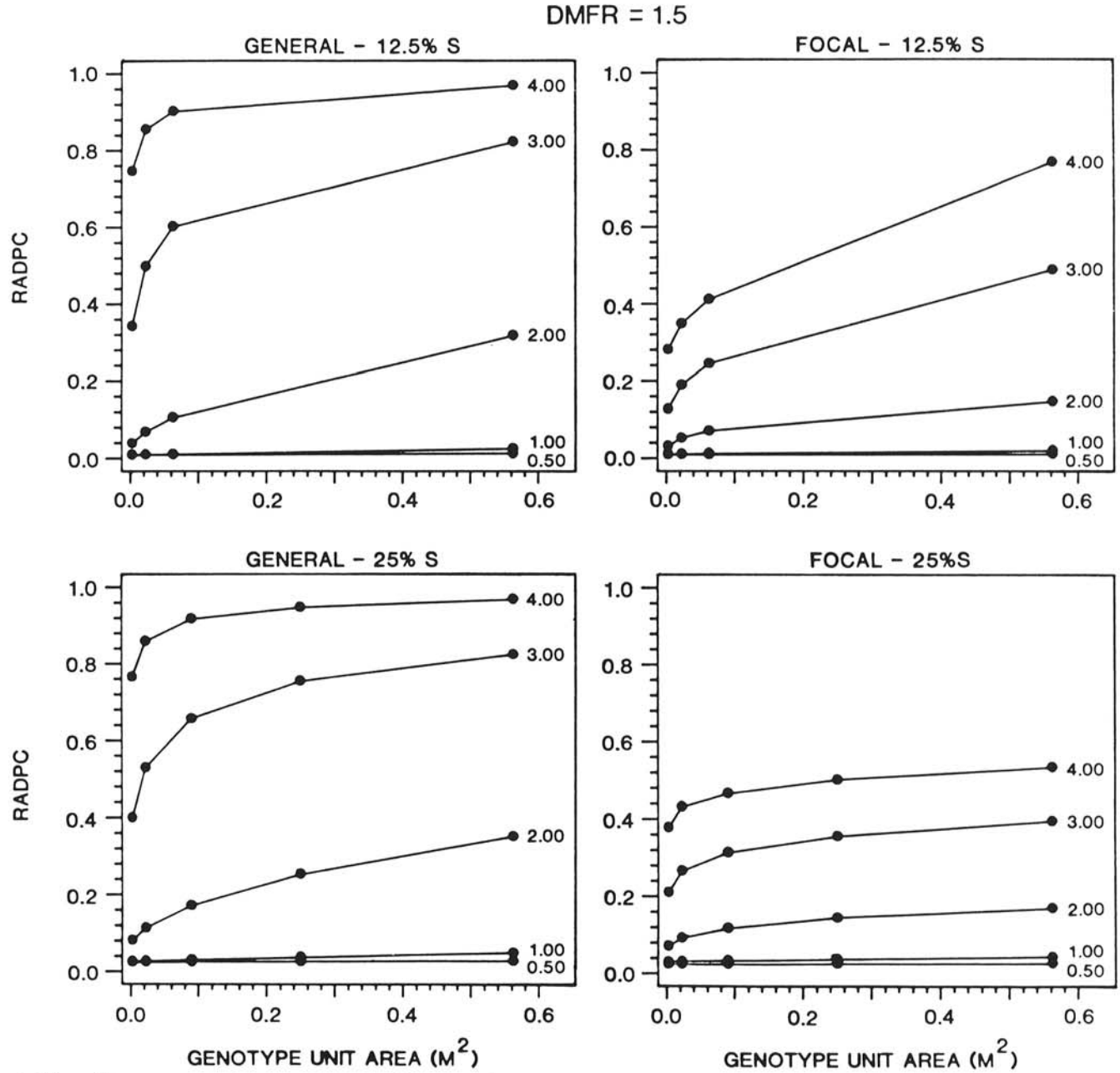

Fig. 3. Effects of host genotype unit area and steepness of pathogen dispersal gradients on the relative area under the disease progress curve (RADPC) for the susceptible genotype in mixtures of immune and susceptible plants. Treatments were the same as in Fig. 2, except the daily multiplication factor was 1.5 lesions per lesion per day.

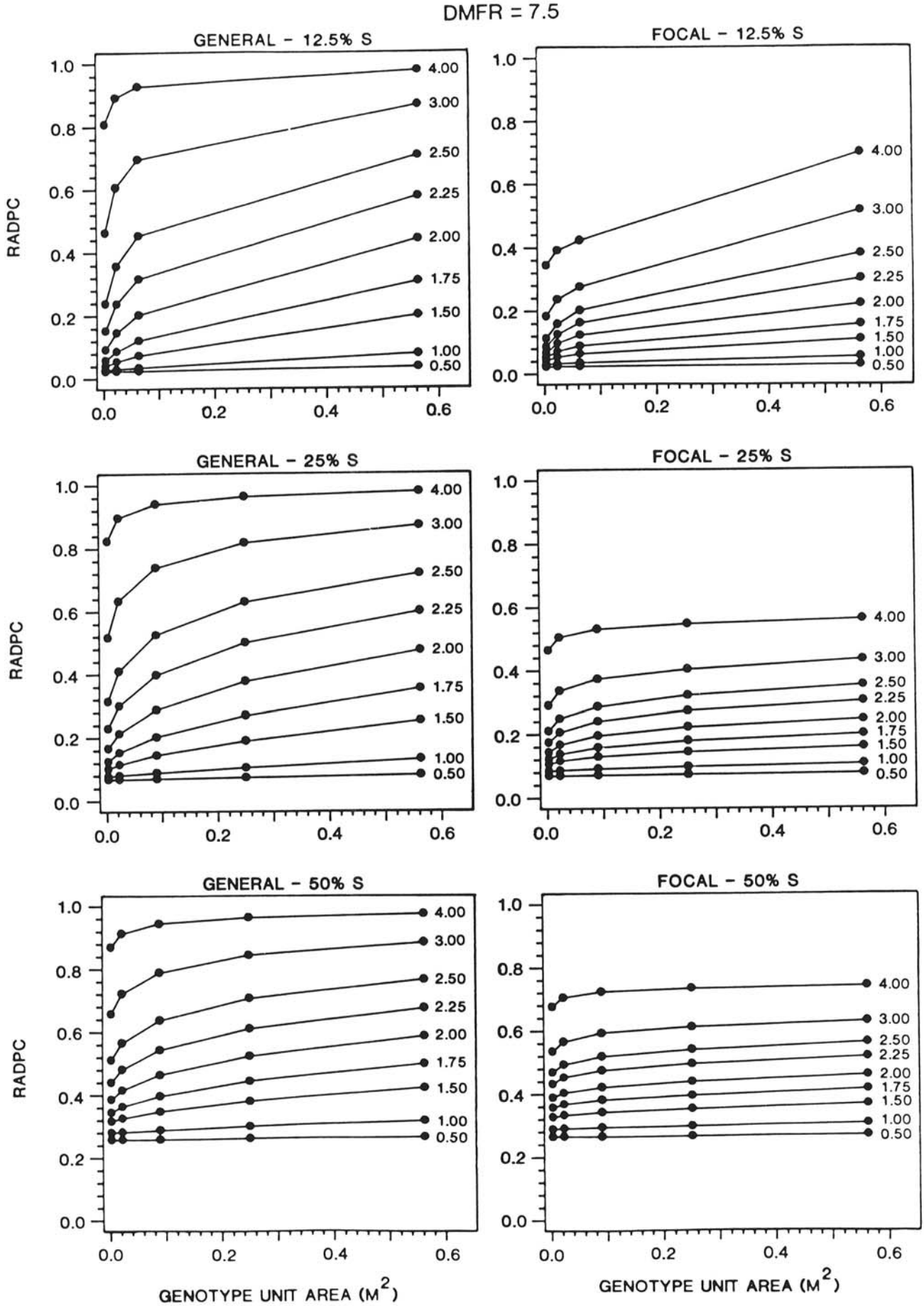

Fig. 4. Effects of host genotype unit area and steepness of pathogen dispersal gradients on the relative area under the disease progress curve (RADPC) for the susceptible genotype in mixtures of susceptible and immune plants with different genotype unit areas. Treatments were the same as in Fig. 2, except the daily multiplication factor was 7.5 lesions per lesion per day and 1:1 (50% S) mixtures of immune/susceptible plants were also simulated.

For DMFR ≥ 1.5, the effectiveness of the mixtures decreased with increasing DMFR for both general and focal epidemics. For DMFR = 0.3, there were several anomalous results. For both general and focal epidemics with $b = 0.50$ and $b = 1.00$, the mixtures were less effective for DMFR = 0.3 than for DMFR = 1.5. This was also true for $b = 2.00$ with all but the largest genotype unit areas.

DISCUSSION

Appropriateness of parameters. Dispersal gradients used in the simulations were chosen to cover the range of b values reported for plant pathogens when gradient data were fit to the original Gregory model. The value of b in the modified Gregory model depends on the value of c, and thus, b values cannot be directly compared for the original and modified models. Nevertheless, when the value of c is small (as in our simulations with $c = 0.03$ m), it has only a relatively small effect on the value of b. For example, we calculated b values of 2.01 and 2.24 for primary disease gradients of oat crown rust with the original and modified Gregory models, respectively, when c of the modified Gregory model was 0.038 m (20).

Values of b calculated from the original Gregory model for spore dispersal or primary disease gradients from point sources of inoculum have been reported to be 1.3 to 1.9 for wheat (*Triticum aestivum* L.) powdery mildew (induced by *E. graminis* DC. f. sp. *tritici* E. Marchal) (8), 2.0 for oat crown rust (20), 2.0–2.6 for southern maize (*Zea mays* L.) rust (induced by *Puccinia polysora* Underw.) (9), 2.0 for common maize rust (induced by *Puccinia sorghi* Schw.) (20), and 2.3–3.3 for potato (*Solanum tuberosum* L.) late blight (induced by *Phytophthora infestans* (Mont.) de Bary) (9). Gregory (9) presented b values for disease gradients of potato viruses X and Y between 1 and 2; it is unclear, however, if secondary spread resulted in flattening of these gradients. MacKenzie (16) reported a very wide range of b values (2.3–4.5) for primary disease gradients of wheat stem rust (induced by *Puccinia*

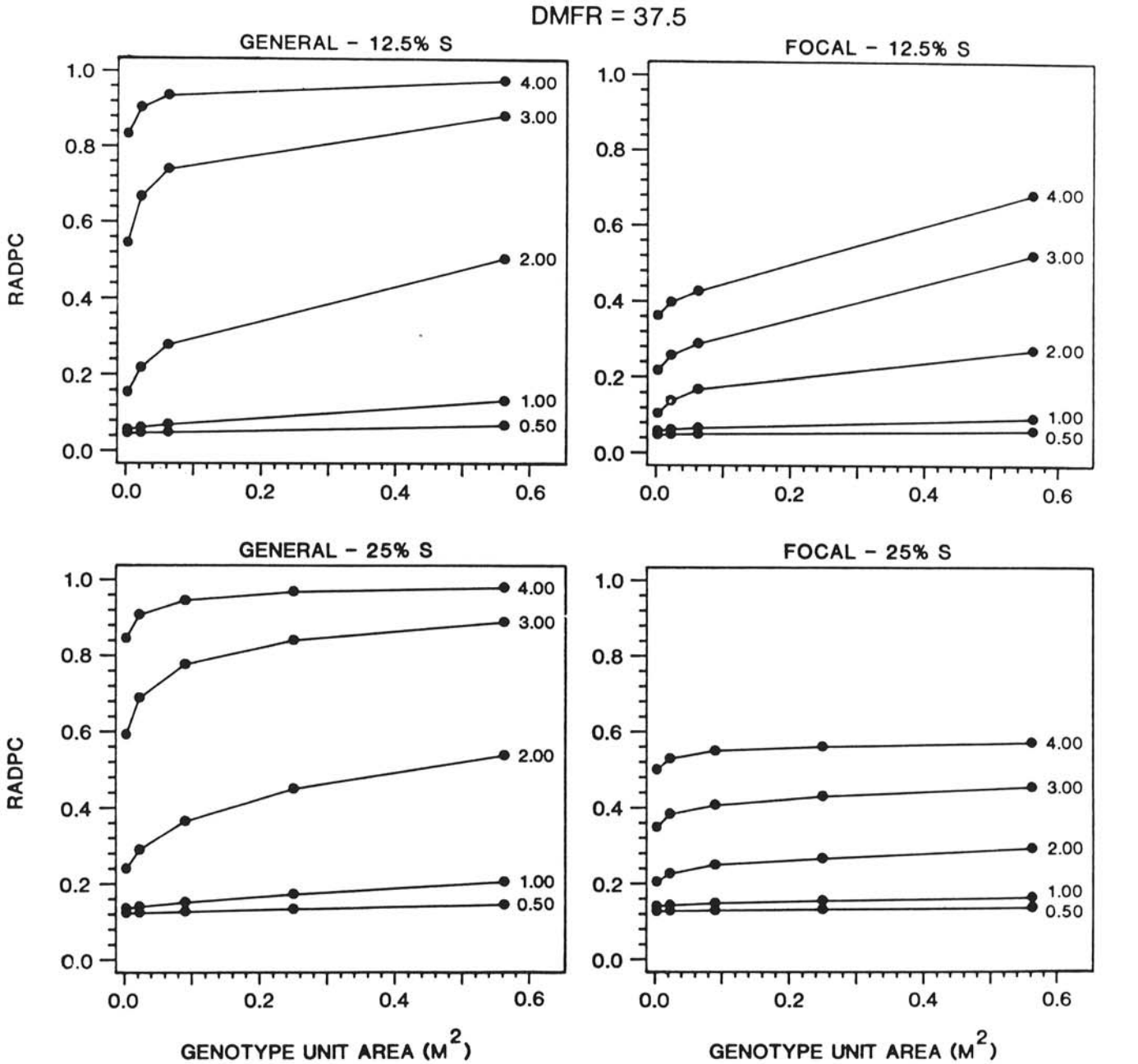

Fig. 5. Effects of host genotype unit area and steepness of pathogen dispersal gradients on the relative area under the disease progress curve (RADPC) for the susceptible genotype in mixtures of susceptible and immune plants. Treatments were the same as in Fig. 2, except the daily multiplication factor was 37.5 lesions per lesion per day.

graminis Pers. f. sp. *tritici* Eriks. & E. Henn). Gradients for *Botrytis cinerea* Pers. ex Fr. recorded at the time of 50% bloom of snap beans (*Phaseolus vulgaris* L.) ranged from 2.5 to 3.3 (11). Stedman (26) reported *b* values for the dispersal of *Lycopodium* spores in a field bean (*Vicia faba* L.) canopy as 1.6–2.0 for wind dispersal and 3.6–3.9 for splash dispersal.

We used the RADPC as a parameter to make treatment comparisons so that epidemic development in mixtures would be expressed relative to the pure-line susceptible check with the same DMFR and dispersal gradient. We calculated ADPCs from the time first pustules appeared until 90% disease severity was attained in the appropriate pure-line check, so that epidemics with different DMFRs would be treated more equally than if we used a common cutoff date for all epidemics. If, for example, we had calculated ADPCs for the entire length of the epidemics, mixtures would probably have been much less effective in epidemics with a high DMFR than in epidemics with a lower DMFR. This is because in a fast epidemic, 100% disease severity is attained early in the epidemic for both the pure-line check and the mixtures. In a slower epidemic, 100% disease severity might not be attained for any treatment.

Effects of epidemic variables on mixture efficacy. The decline in the effectiveness of mixtures with increasing values of *b* was probably due to an increase in the amount of autoinfection as *b* increased. In the simulations, the proportion of autoinfection per single source compartment was 0.00057, 0.0041, 0.025, 0.056, 0.11, 0.20, 0.31, 0.55, and 0.85 for epidemics with *b* values of 0.50, 1.00, 1.50, 1.75, 2.00, 2.25, 2.50, 3.00, and 4.00, respectively. In simulating the increase of disease along single rows of mixtures of resistant and susceptible plants, Kiyosawa and Shiyomi (14) found a decrease in mixture efficacy with increasing steepness of the dispersal gradient, and they also attributed this decline to increased autoinfection.

The decrease in the effectiveness of mixtures with increasing genotype unit area was probably also caused by an increase in the amount of autoinfection, as was indicated in an earlier study (22). There was little effect of genotype unit area on mixture efficacy at small values of *b* because when *b* is small, inoculum is distributed more uniformly and there is less autoinfection. With the steepest gradient ($b = 4.00$), there was usually a lesser effect of genotype unit area than at intermediate values of *b*. This could be because when the gradient was steep, the proportion of autoinfection per compartment was high, and therefore, there was little potential for increasing autoinfection by increasing genotype unit area.

The simulations showed a smaller effect of genotype unit area on mixture efficacy for focal than for general epidemics. Similar results were reported for field (21) and computer-simulated (22) epidemics of oat crown rust and for experiments investigating the effects of host aggregation on damping-off disease (4). It has been suggested that the interaction between genotype unit area and the spatial distribution of initial disease is due to differences in the rate of disease intensification within and between genotype units (4,21). Computerized simulation studies of oat crown rust epidemics suggest that focus saturation may also help explain the interaction between genotype unit area and the spatial distribution of initial disease (22).

The positioning of initial inoculum for focal epidemics caused a bias in the amount of autoinfection in the mixtures. Initial inoculum was placed in the centers of the plots at the junction of susceptible and immune genotype units, and thus, only the corners of susceptible genotype units were initially infected. When these initial infections produced spores, a smaller number of spores landed on susceptible genotype units than if initial inoculum had been placed at a more central location within genotype units. In an earlier study (22), we found that the position of an initial focus significantly influenced epidemic development only in the early stages of an epidemic.

Results from focal epidemics with 12.5% susceptible plants should be viewed with caution because a different inoculation procedure was used than for the other focal epidemics. To begin all epidemics with the same number of initial infections per susceptible plant, it was necessary to use half as many spores per inoculated plant for the 12.5% susceptible plots than for the other focal epidemics. Thus, there was a smaller amount of spore deposition on previously infected tissue for focal epidemics with 12.5% susceptible plants than for focal epidemics with 25–50% susceptible plants; this could be an important factor, especially for steeper gradients that result in a large proportion of autoinfection. In addition, with the 12.5% susceptible mixtures, there were only two susceptible genotype units in the mixture with the largest

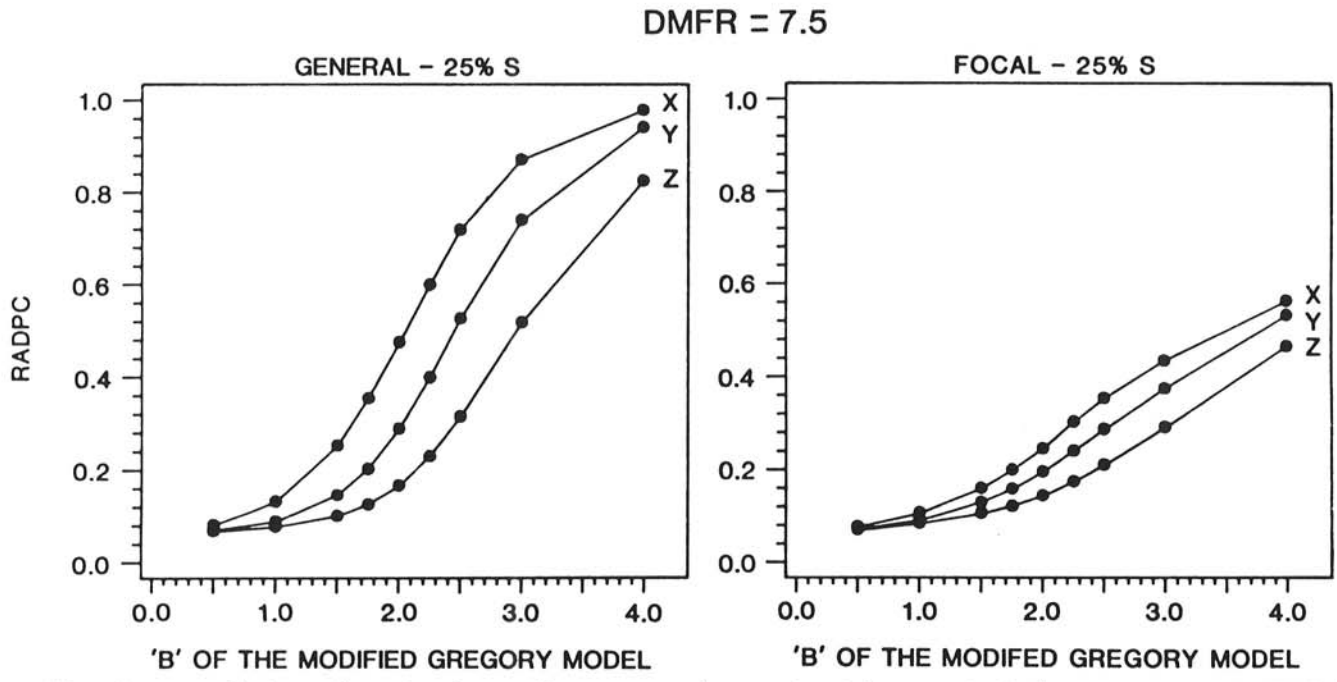

Fig. 6. Effects of steepness of pathogen dispersal gradients and host genotype unit area on the relative area under the disease progress curve (RADPC) for susceptible plants in computer-simulated epidemics of polycyclic, foliar disease in 1:3 mixtures of susceptible/immune plants. X, genotype unit area = 0.5625 m^2; Y, genotype unit area = 0.0900 m^2; Z, genotype unit area = 0.0025 m^2. General epidemics were initiated with one spore in each of the 3,600 compartments of each plot. Focal epidemics were initiated with 900 spores in each of the four compartments at the center of each plot. Data are the same as in Fig. 4 but were plotted to better illustrate the effect of dispersal gradient steepness on epidemic development in mixtures.

genotype unit area. Therefore, one-half of the susceptible genotype units were initially infected. This may explain why there was a larger increase in the RADPC between the mixtures with the two largest genotype unit areas for focal than for general epidemics with 12.5% susceptible plants and $b = 4.00$.

As expected, the effectiveness of the mixtures relative to pure-line susceptible populations decreased with increasing percentage of susceptible plants. There was usually a much larger decrease in mixture efficacy when the percentage of susceptible plants was doubled from 25 to 50% than when the percentage of susceptible plants was doubled from 12.5 to 25%. Similar results have been reported for rusts and powdery mildews of small grains (5,15), and MacKenzie (17) hypothesized that the benefit in disease control gained from increasing the number of lines in a multiline cultivar would follow a diminishing-returns function.

We found that the effects of genotype unit area and dispersal gradient steepness on mixture efficacy decreased as the proportion of susceptible plants increased. Similarly, Kiyosawa (13) found a much stronger effect of dispersal gradient steepness on mixture efficacy when the percentage of susceptible plants was low than when the percentage of susceptible plants was high.

The interaction between the percentage of susceptible plants and effects of gradient steepness and genotype unit area on the effectiveness of host mixtures might be explained by the ratio of autoinfections to alloinfections (infections on a genotype unit resulting from propagules produced on other genotype units [24]). The amount of autoinfection per genotype unit depends on the steepness of the dispersal gradient and the size of the genotype unit but is independent of the proportion of susceptible genotype units in the mixture. On the other hand, as the proportion of susceptible genotype units increases, the amount of alloinfection per genotype unit will increase because there are more susceptible genotype units contributing to alloinfections and because the average distance between susceptible genotype units is decreased. Therefore, autoinfections will contribute a lesser proportion of the total infections in a mixture as the proportion of susceptible genotype units increases. Consequently, factors that determine the amount of autoinfection become less important as the proportion of susceptible units increases.

For general epidemics with steep gradients and large genotype units, there was very little difference among mixtures with 12.5, 25, or 50% susceptible plants. This is probably because there are relatively few alloinfections in these epidemics. Therefore, disease development within susceptible genotype units is nearly independent of other susceptible units in the population, as was indicated in an earlier computerized simulation study (22).

The effectiveness of mixtures for disease control declined with increasing values of DMFR, probably because the epidemics approached completion sooner in the faster epidemics and therefore there were fewer generations of disease increase. For a polycyclic disease, one would expect differences among epidemics with different rates to increase over time. For example, the difference in the amount of disease between a pure-line susceptible population and mixtures of resistant and susceptible plants should be greater with increasing numbers of pathogen generations (1,13–15). We did not vary the latent period in our simulations, but for diseases with equivalent infection rates but different latent periods, mixtures should be most effective against those with the shortest latent periods because there would be more generations of disease increase.

The general trend of increasing RADPC (decreasing mixture efficacy) with increasing gradient steepness held true for epidemics with DMFR ≥ 1.5 but not for those with DMFR = 0.3. In epidemics with DMFR = 0.3, there was a quadratic response with the lowest RADPC at $b = 1.00$ or 2.00. Kiyosawa (13) reported a similar quadratic relationship between mixture efficacy and steepness of dispersal gradient. He suggested that the decrease in mixture efficacy with the shallower gradients might be related to the large proportion of spores that are dispersed out of plots with shallow gradients. Our results indicate that this quadratic response is related to both dispersal gradient steepness and the rate of disease increase because the same response was not seen at DMFR ≥ 1.5. If this quadratic response is real and not an artifact of simulation models, it is probably biologically unimportant for many plant diseases, because the response was seen only at rates of disease increase that would not cause significant economic loss. For example, with DMFR = 0.3 and $b = 0.50$, the percentage of disease increased only 8.5 times over a 61-day general epidemic in a pure-line susceptible population. Nevertheless, very slow epidemics can be important when the amount of initial disease is high and/or when epidemics progress over long periods of time.

Implications of results. Our results suggest that growing mixtures of plants with different race-specific resistance genes will always result in less disease than growing the same host genotypes separately. However, mixtures will be less effective for the control of pathogens with steep dispersal gradients (e.g., pathogens that are splash-dispersed) than for rusts and powdery mildews, which have shallower gradients. Diseases such as potato late blight and Botrytis blight of snap beans might represent intermediate cases.

Our results also indicate that the use of host mixtures for disease control will be less effective for crops with large plants (e.g., potatoes with genotype unit areas for individual plants of about 0.2 m^2) than for crops with small plants (e.g., small grains with genotype unit areas for individual plants of about 0.003 m^2). However, for pathogens with shallow gradients (e.g., powdery mildews), plant size may not be as limiting to the effectiveness of mixtures for disease control as for pathogens with steeper gradients (e.g., *P. infestans*). Also, for epidemics initiated from widely separated disease foci, plant size should be less of a limiting factor than for epidemics in which inital disease is more uniformly distributed. Similarly, growing alternate rows or strips of different host genotypes rather than random mixtures of plants should be more effective for focal epidemics and for pathogens with shallow gradients.

Decreasing the proportion of susceptible plants in a mixture of immune and susceptible plants is analogous to increasing the number of host genotypes with different race-specific resistance genes in a multiline or cultivar mixture (assuming the presence of pathogen genotypes virulent on a small number of host genotypes). As expected, our results indicate that increasing the number of host genotypes in a mixture will increase the disease-reducing effectiveness of that mixture. However, a large genotype unit area or a steep dispersal gradient will result in a greater decrease in mixture efficacy for a mixture with a large number of host genotypes than for a mixture with a smaller number of host genotypes. Therefore, increasing the number of host genotypes in a mixture may not compensate completely for reductions in mixture efficacy caused by steep dispersal gradients or large genotype unit areas.

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