Computerized Simulation of Crown Rust Epidemics in Mixtures of Immune and Susceptible Oat Plants with Different Genotype Unit Areas and Spatial Distributions of Initial Disease

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ABSTRACT

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A modified version of the computerized model EPIMUL was used to study effects of host genotype unit area (the ground area occupied by an independent, genetically homogeneous unit of a host population) and spatial distribution of initial disease on crown rust epidemics in oat populations consisting of 25% susceptible and 75% immune plants. Mixtures with different genotype unit areas were represented by considering each compartment in an N × N matrix to be a 0.0025 m^2 plant and aggregating plants into units of plants of the same genotype. The effects of genotype unit area and the spatial distribution of initial disease on the effectiveness of the mixtures for oat crown rust control were similar to those found in previous studies conducted in the field. The proportion of rust infection in the mixtures usually increased as genotype unit area was increased from 0.0025 to 0.56 m^2. The magnitude of this increase, however, was much larger when initial disease was distributed uniformly than when initial disease was concentrated in a central focus. The position and size of the initial disease focus relative to the position and size of host genotype units were found to influence the effect of genotype unit area on the effectiveness of mixtures for disease control. Spores produced on the first day of spore production were categorized into five groups: those resulting in autoinfections, those resulting in alloinfections, spores dispersed to immune plants in the mixtures, spores deposited on previously infected tissue, and spores dispersed outside of plots. The ratio of autoinfection to alloinfection and focus saturation were found to contribute to the interaction between genotype unit area and the spatial distibution of initial disease in host mixtures.

Additional key words: *Avena sativa*, cultivar mixtures, disease gradients, genetic diversity, multilines, *Puccinia coronata*, spore dispersal.

Biological studies indicate that the effect of host genotype unit area (the ground area occupied by an independent, genetically homogeneous unit of a host population [9]) on the effectiveness of host mixtures for disease control depends on whether the initial disease is concentrated in a single focus or distributed more uniformly. Altering the spatial arrangement of near-isogenic oat (*Avena sativa* L.) lines to increase the area of genotype units from 0.003 to 0.84 m^2 had no significant effect on the efficacy of oat multilines in controlling crown rust (induced by *Puccinia coronata* Cda. var. *avenae* Fraser and Ledingham) when epidemics were initiated with one disease focus per plot (9). Barrett and Wolfe (2) found that in the United Kingdom, increasing the area of spring barley (*Hordeum vulgare* L.) plants in a cultivar mixture by altering sowing density resulted in a decrease in the effectiveness of the mixture in controlling powdery mildew (induced by *Erysiphe graminis* DC. f. sp. *hordei* Marchal). In the United Kingdom, initial infections of powdery mildew on spring barley are distributed nearly randomly (18). Mundt and Leonard (11) reported that increasing genotype unit area greatly reduces the efficacy of oat mixtures for crown rust control when initial disease is distributed uniformly or randomly but not when it is concentrated in a single focus. Therefore, they hypothesized that the difference between the results of Barrett and Wolfe (2) and those of Mundt and Browning (9) may have been due to differences in the spatial distribution of initial disease. In controlled-environment studies, Burdon and Chilvers (5) found that increasing the area occupied by clumps of cress (*Lepidium sativum* L.) seedlings (while keeping constant the overall area occupied by seedlings) did not affect the rate of linear advance of damping-off (induced by *Pythium irregulare* Buisman) when epidemics were initiated from a single focus of initial inoculum. When initial inoculum was distributed randomly in the soil, however, the apparent infection rate (sensu Vanderplank [17]) of damping-off was significantly higher in the treatment with the largest clumps.

The purpose of our study was to use a computerized model to study the interaction between genotype unit area and the spatial distribution of initial disease in host mixtures and to investigate possible mechanisms for this interaction.

MATERIALS AND METHODS

Description of the model. The model was a modified version of EPIMUL (8,20). EPIMUL divides a simulated host population into an N × N matrix of compartments in which each compartment is considered to be a host unit such as a plant, experimental plot, or field. The proportion of disease in each compartment is updated by simulating equation 8.3 of Vanderplank (17), which can be expressed as

$$dx_i/dt = R_c(x_i)\,(1 - x_i) \qquad (1)$$

in which x_i is the proportion of diseased tissue at time t, R_c is the corrected basic infection rate (the number of lesions produced per lesion per day of the infectious period), and x_i is the proportion of infectious tissue at time t.

The EPIMUL model uses arrays and delay functions to keep track of the number of latent infections (infections that are not yet producing spores), the number of infectious lesions (lesions that are producing spores), and the number of removals (lesions that are no longer producing spores). This information is updated at the end of

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each day for every compartment in the block. At the end of each day, the number of potentially effective spores (spores that would produce new infections on susceptible plants in an infinitely large host population) is calculated for each compartment in the block. The number of potentially effective spores (hereafter called "spores") is calculated as DMFR × NIL in which DMFR is the daily multiplication factor (the number of lesions produced per lesion per day of the infectious period in an infinitely large, susceptible host population = Vanderplank's [17] corrected basic infection rate) and NIL is the number of infectious lesions in the compartment on that day. Therefore, spores that would be deposited on the ground and spores that would fail to infect susceptible tissue due to unfavorable environmental conditions are not modeled. In EPIMUL, all spores that are deposited in susceptible compartments will produce lesions after passing a period of latency if the number of available infection sites is not limiting. The number of infection sites per compartment is determined by the area of the compartment, the leaf area index, and the area of a lesion. At the end of each day, the spores produced from each compartment are distributed over all compartments in the block by using a dispersal model based on the equations of Pasquill (12). This model produces a Gaussian distribution for the spores produced from each compartment.

With EPIMUL, epidemic development in mixtures of immune and susceptible plants is simulated by randomly assigning a compatibility value to each compartment. Each different compatibility value represents a different host genotype and the number of different compatibility values is an input of the model. There is an equal probability for each of the compatibility values to be assigned to each compartment. Simulations are begun by assigning a single spore to one compartment of the block. Only compartments with the same compatibility value as that receiving the initial spore are considered to be susceptible. No provision is made for simultaneously modeling the increase of more than one race of the pathogen. The proportion of susceptible compartments is determined by the number of genotypes studied and is approximately equal to $1/n$, in which n is the number of host genotypes. The actual proportion of susceptible compartments varies among simulation runs because compatibility values are assigned randomly with replacement.

Given a constant compartment size, EPIMUL allows one to alter the genotype unit area for a mixture by setting up "units" (Fig. 1). Units are $n \times n$ aggregates of compartments that are assigned the same compatibility value (i.e., they are of the same resistance genotype). Units with different compatibility values are positioned randomly (with replacement) within the block.

Input variables for EPIMUL are the length of the side of a compartment, the distance from a source compartment at which the number of spores deposited is one-half that at the source (a measure of dispersal gradient steepness), the number of host genotypes (number of compatibility values), the number of compartments per unit, a number for initializing randomization of compatibility values, the length of the epidemic, the frequency (in days) at which output will be generated, the position of the compartment receiving the initial spore, the daily multiplication factor, the leaf area index of the host, the area of a lesion, the number of compartments in the block (maximum of 400), and the lengths of the latent and infectious periods. Output from the model includes plots and listings of several epidemic variables including the proportion of diseased tissue and graphical representations of the spatial distribution of disease over time.

Modifications to EPIMUL included reformatting for compatibility with IBM computers and making the following changes in the structure and logic of the program: maximum block size was expanded from a 20 × 20 to a 60 × 60 matrix of compartments; provisions were made to allow ordered positioning of genotypes rather than using the randomization scheme; the program was changed so host genotypes are randomly positioned without replacement, thus producing an equal number of each genotype in the population (this procedure guarantees that the proportion of susceptible compartments will not vary among runs of the model;) the program was modified to allow for more than one compartment to be initially inoculated and more than one initial spore per inoculated compartment to be used; and alterations were made in the spore-sweeping routine (a routine that cumulates fractions of spores into whole spores and assigns them to compartments) to enable it to function more realistically with our program and to make the sweep routine optional.

The most significant change made in Kampmeijer and Zadoks' (8) program was to replace their spore dispersal model with the modified Gregory model, which has been shown to describe primary disease gradients of cereal rusts well (10). Because EPIMUL models the dispersal of potentially effective spores only, it is desirable that the dispersal model produce primary disease gradients similar to those observed in the field. The dispersal model that Kampmeijer and Zadoks (8) used in EPIMUL describes a Gaussian distribution and provides gradient shapes very different from primary disease gradients we have calculated from field experiments and disease gradients we have found in the literature.

The modified Gregory model describes spore or infection density in a single direction only, so it was necessary to alter the model to describe dispersal in all directions by using the equation:

$$z = a[(x^2 + y^2)^{0.5} + c]^{-b} \quad (2)$$

in which z is the number of spores deposited per compartment, a is the number of spores deposited per compartment at $1 - c$ units of distance from the center of the source compartment, x and y are the abscissa and ordinate distances in the matrix of compartments, c is a truncation factor that allows for a finite number of infections in the source compartment (i.e., distance = 0), and b is a measure of the steepness of the gradient.

The parameter a is a measure of the amount of inoculum produced from a source compartment. In the simulation of epidemics, however, the amount of inoculum produced per compartment will vary among compartments and over time because disease is not uniformly distributed in space or time. Therefore, equation 1 must be modified to describe probability density, i.e., the relative number of spores produced from a compartment that are deposited in each compartment of the block.

To describe probability density in our modification of EPIMUL, a matrix is set up that is four times the area to be simulated (i.e., a 2N × 2N matrix). Inoculum is dispersed from a single, central compartment to all compartments in the 2N × 2N matrix by using

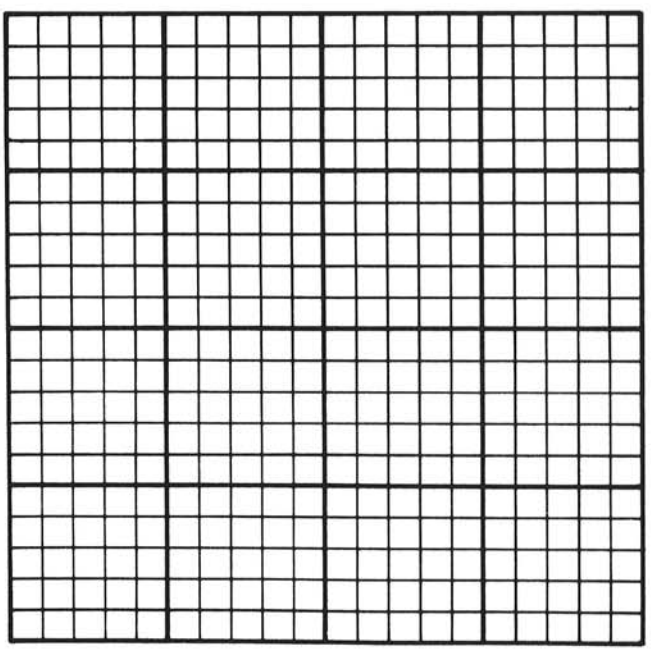

Fig. 1. Compartmentalization of space in the EPIMUL simulator showing a block of 400 compartments. The groups of 25 compartments enclosed by bold lines represent units of compartments containing the same compatibility value.

equation 1 and a value of a that will allow the proportion of spores deposited in all compartments of the 2N × 2N matrix to sum to one, thus giving the proportions of spores produced from the source compartment that are deposited in each compartment of the 2N × 2N matrix. Therefore, the model assumes that all inoculum produced from the center of the 2N × 2N matrix is maintained within that matrix. The proportions are calculated for an octant of the 2N × 2N matrix and are stored in a matrix along with their corresponding distances from the source compartment. Because of symmetry in the 2N × 2N matrix, this octant of probabilities can be used to determine the proportion of inoculum dispersed between any two compartments in the N × N block.

Comparison of simulations with field data. The model was used to simulate Mundt and Leonard's (11) 1984 field study in which the effect of genotype unit area on epidemic development of oat crown rust was studied in both focal and general epidemics (i.e., epidemics initiated with a single disease focus per plot and epidemics in which initial disease was uniformly distributed within plots). Our goal was not to duplicate results from the field study precisely, but to see if simulated epidemics showed a qualitatively similar response to genotype unit area and spatial distribution of initial disease as was found in the field.

Input values were lesion size = 4.00 mm^4; length of the side of a compartment (length of the side of the area occupied by a plant) = 0.05 m, leaf area index = 3.00, c of the modified Gregory model = 0.038 m, b of the modified Gregory model = 2.236, daily multiplication factor = 10.0 pustules·pustule^{-1}·day^{-1}, latent period = 9 days, infectious period = 17 days, block size = 60 × 60 compartments, output frequency = 1 day, and length of epidemic = 61 days. The spore-sweeping routine was not used and simulations were performed using fractional spores.

Input values of the preceding paragraph were chosen to represent conditions present in the field experiment (11). The values of b and c were those reported for a field experiment with oat crown rust (10) that was performed in the same field and at the same time as Mundt and Leonard's (11) oat mixture experiment. The lesion size and leaf area index values chosen provided a carrying capacity of 1,875 pustules per plant and a plant was considered to consist of a single culm. This is similar to the carrying capacity of 1,800 pustules per culm used by Burleigh et al (7) for wheat (*Triticum aestivum* L.) leaf rust (induced by *Puccinia recondita* Rob. ex Desm. f. sp. *tritici*). Because such data were not available for oat crown rust, we assumed that carrying capacities for oat crown rust and wheat leaf rust would be similar.

There were six treatments consisting of a factorial arrangement of three simulated oat populations and two spatial distributions of simulated initial inoculum. Each compartment was considered to be a single oat plant and a block of 3,600 compartments represented a field plot. The first simulated oat population was a pure stand of susceptible plants. The other two simulated populations each consisted of 25% susceptible and 75% immune plants. In one mixture, positions of plants within plots were random without replacements. In the other mixture, plants (compartments) were aggregated into units of 225 plants of the same genotype. Positions of the units of 225 plants were also randomized without replacement, but the center four genotype units in each plot were randomized separately so that one would be susceptible and the other three immune (the same randomization scheme used in Mundt and Leonard's [11] field experiments). Four replications using different randomizations were performed for each of the mixture treatments.

For all three simulated oat populations, initial inoculum consisted of 1,152 spores positioned to give a similar pattern and amount of initial disease as in the field experiment (11). For focal inoculations, 32 spores were assigned to the central 36 compartments of each plot. For general inoculations, 36 spores were assigned to two evenly spaced compartments in each of the 16 groups of 225 contiguous compartments in each plot.

Mechanisms of disease control in host mixtures. Input variables were the same as in the preceding section except for the

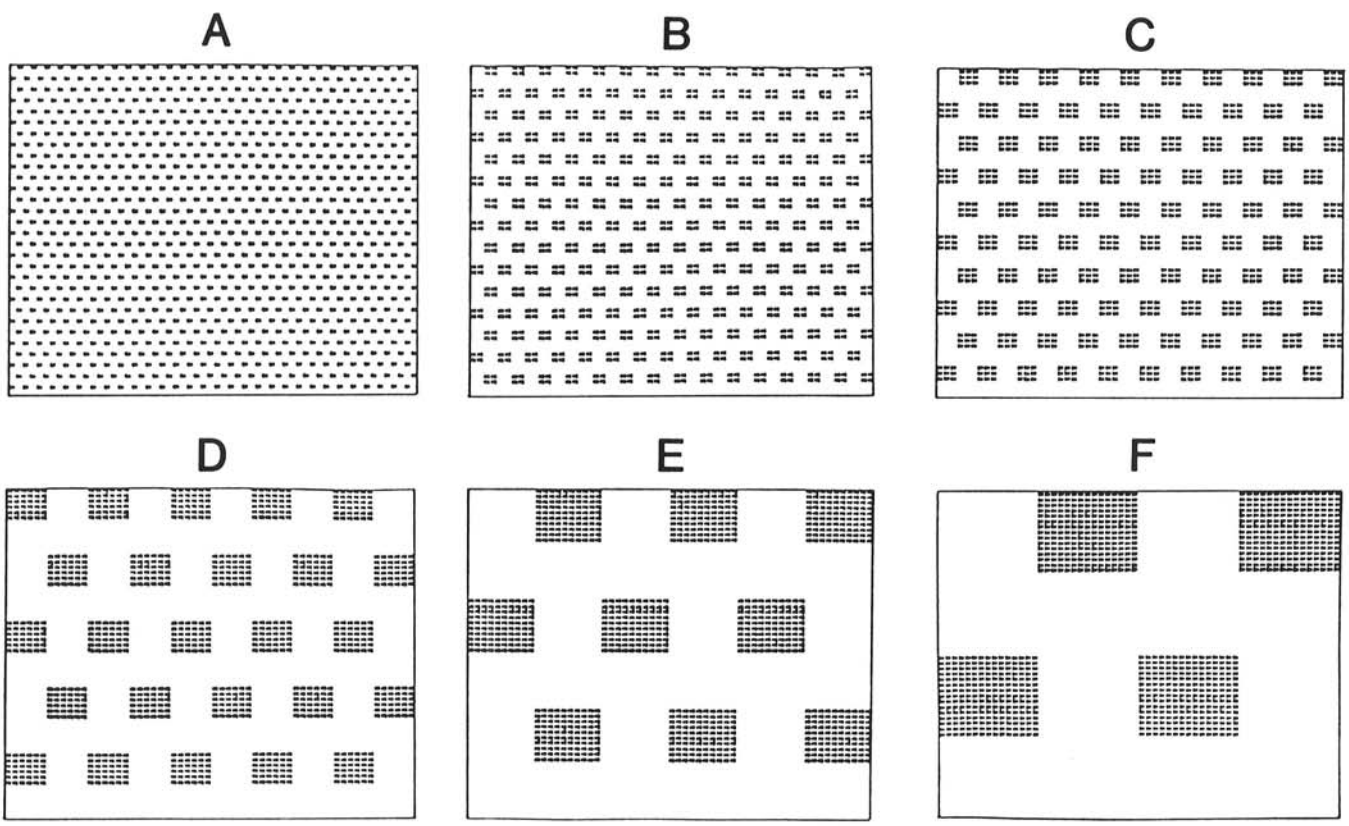

Fig. 2. Arrangement of host genotypes in computer-simulated oat crown rust epidemics. Each of the six large blocks **(A-F)** represent one simulated plot of 3,600 oat plants. Each small rectangle represents one susceptible oat plant. Blank spaces represent immune plants. **A,** Genotype unit area = 0.0025 m^2; **B,** genotype unit area = 0.0100 m^2; **C,** genotype unit area = 0.0225 m^2; **D,** genotype unit area = 0.0900 m^2; **E,** genotype unit area = 0.2500 m^2; **F,** genotype unit area = 0.5625 m^2. Plots and plants appear rectangular rather than square due to limitations of the printer used to produce the figure.

arrangements of host genotypes and inoculation methods. A factorial arrangement of seven oat populations and two spatial distributions of initial inoculum were studied. One oat population was a pure stand of susceptible plants. The other six populations each consisted of 25% susceptible and 75% immune plants in which plants were aggregated into units of 1, 4, 9, 36, 100, and 225 plants of the same genotype to provide mixtures with genotype unit areas of 0.0025, 0.010, 0.022, 0.090, 0.25, and 0.56 m^2, respectively. The positions of genotype units within plots were ordered as shown in Figure 2.

For all simulated oat populations, initial inoculum consisted of 3,600 spores per plot. General inoculations consisted of one spore assigned to each of the 3,600 compartments of each plot. Focal inoculations consisted of 900 spores assigned to each of the center four compartments of each plot (one compartment in the corner of each of the four central genotype units in each plot). A treatment was added to the factorial as a check on the effect of the positioning of initial inoculum in the mixtures. In this treatment, 900 spores were assigned to the center compartment of the central, susceptible genotype unit for a mixture with a genotype unit area of 0.56 m^2 and 25% susceptible plants. Inoculations resulted in a mean of one initial infection per susceptible plant for all 15 treatments.

As a check on the effect of focus saturation on epidemic development in mixtures, we performed the same simulations described above for focal epidemics, but we changed the leaf area index and lesion size parameters to give a carrying capacity of 2.50×10^9 pustules per plant. The purpose of the higher carrying capacity was to prevent saturating the initially inoculated genotype units with infections during the course of the epidemics.

We recorded the number of spores produced per plot on the ninth day after inoculation (the first day of spore production) for the factorial set of simulations with a carrying capacity of 1,875 pustules per plant. We determined the proportion of these spores that were deposited on immune plants in the mixtures, were dispersed out of the plots, that resulted in autoinfections (sensu Robinson [16]), and that resulted in alloinfections (sensu Robinson [16]). Autoinfections were considered to be infections on a susceptible genotype unit that resulted from spores produced on that same genotype unit. Alloinfections were considered to be infections on a susceptible genotype unit that resulted from spores produced on other genotype units in the population. For focal inoculations, the proportions of spores deposited on previously infected tissue also were calculated; it was unnecessary to make this calculation for the general epidemics because there were no plants with a sufficiently high level of infection at the beginning of the epidemics to account for a significant amount of deposition on previously infected tissue.

For each simulated plot, the number of spores accounted for by each of the categories described above was calculated as follows: (i) For general inoculations, autoinfection = S × PS, in which S is the number of susceptible genotype units in a plot and PS is the number of new pustules on day 17 (the day that pustules resulting from spores produced on the first day of spore production first appeared) for a separate simulation with a single, susceptible genotype unit that was surrounded by immune plants in a population of 3,600 plants and that had a single infection per compartment on day 0. For focal inoculations, autoinfection = the number of new pustules occurring on the initially inoculated genotype unit on day 17. (ii) For both general and focal epidemics, alloinfection = TP − autoinfections, in which TP is the total number of new pustules in the plot on day 17. (iii) For both general and focal epidemics, the number of spores deposited on immune plants = TPS − (autoinfections + alloinfections), in which TPS is the total number of new pustules per plot occurring on day 17 when spores were introduced into a pure-line susceptible population in the same positions at which initial pustules were present in the mixtures. (iv) For focal epidemics, the number of spores deposited on previously infected tissue = TPA − TP, in which TPA is the total number of pustules per plot on day 17 when the simulations were run with a single spore per initially inoculated compartment and a daily multiplication factor of 9,000.0 pustules·pustule^{-1}·day^{-1} to produce the same number of spores on day 9 as in the original simulations with 900 spores per initially inoculated compartment and a daily multiplication factor of 10.0 pustules·pustule^{-1}·day^{-1}. Inoculation with only one spore per compartment prevented a significant amount of spore deposition on previously infected tissue. (v) For general epidemics, the number of spores dispersed out of a plot = TS − (autoinfections + alloinfections + spores deposited on immune plants), in which TS is the total number of spores produced on day 9. For focal epidemics, the number of spores dispersed out of a plot = TS − (autoinfections + alloinfections + spores deposited on immune plants + spores deposited on previously infected tissue). For both general and focal epidemics, the number of spores accounted for by these categories were converted to proportions by dividing each by TS.

Effect of size of initial focus. A set of simulations was performed using the same input variables and factorial arrangement of treatments as described for focal epidemics in the previous section (including the two carrying capacities) except that initial inoculum was distributed over the central 900 compartments of the plots, four spores per compartment.

RESULTS

Comparison of simulations with field data. The simulated epidemics reached a given level of disease sooner than the field epidemics (Fig. 3). For data of the field study, 31% disease was calculated for the last assessment date for the pure-line susceptible plots inoculated generally, whereas there was about 96% disease for the same treatment at the same time after inoculation for the simulated epidemic. Also, there was a lesser effect of the spatial distribution of initial disease on epidemics in the pure-line susceptible populations in the simulated epidemics than in the field epidemics. Nevertheless, the qualitative effects of host genotype unit area and spatial distribution of initial disease were similar in the simulated and field epidemics. In both field and simulated epidemics, the completely random mixture greatly reduced epidemic development relative to the pure-line susceptible treatment for both focal and general epidemics. The mixture of large genotype units, however, was much less effective in the general than in the focal epidemics.

Mechanisms of disease control in mixtures. The effect of genotype unit area and spatial distribution of initial disease were similar in simulated epidemics using an ordered arrangement of genotypes (Figs. 4 and 5) as they were in the simulations used to compare simulations with field studies (Fig. 3). The effectiveness of the mixtures decreased with increasing genotype unit area, but this decline was more pronounced with the general than with the focal epidemics. With focal epidemics, the treatment in which initial inoculum was placed in the center of the 0.56-m^2 genotype unit closest to plot center had much more disease than the other mixtures in the early stages of the epidemics. Later in the epidemics, however, the disease progress curves for the two mixtures with 0.56-m^2 genotype units were nearly identical (Fig. 5). When the carrying capacity was raised to prevent focus saturation, the difference between these two treatments was maintained throughout the epidemic (Table 1). Disease progress curves for mixtures with genotype unit areas of 0.010 and 0.25 m^2 were omitted from Figures 4 and 5 for clarity of presentation; the curve for the mixture with a genotype unit area of 0.010 m^2 was always intermediate between curves for mixtures with genotype unit areas of 0.0025 and 0.022 m^2 and the curve for the mixture of 0.25-m^2 genotype units was always intermediate between curves for mixtures with 0.090- and 0.56-m^2 genotype units.

For general epidemics, the proportion of spores produced on day 9 that induced autoinfections increased and the proportion of spores accounted for by alloinfections decreased with increasing genotype unit area (Table 2). The increase in the number of autoinfections was greater than the decline in the number of alloinfections, with the result that the total number of infections on susceptible plants (autoinfections + alloinfections) increased with increasing genotype unit area. The total number of infections was nearly twice as great in the mixture with the largest than in the mixture with the smallest genotype units.

The ratios of autoinfection to alloinfection for the general epidemics suggest that disease increase in susceptible genotype units becomes nearly independent of other susceptible genotype units in the population as the genotype unit area becomes large. For example, with genotype units of 0.0025 m^2, 52% of the total infections in the population were alloinfections. In the mixture of 0.56-m^2 genotype units, however, only 3.6% of the total infections were alloinfections. This is illustrated in Figure 6, which shows that epidemic development in a single, isolated 0.0025-m^2 genotype unit is much slower than epidemic development in a population of 0.0025-m^2 genotype units. There was very little difference, however, in epidemic development in a single, isolated 0.56-m^2 genotype unit as compared with a population of 0.56-m^2 genotype units.

With focal epidemics, the proportion of spores accounted for by autoinfection increased and the proportion of spores accounted for by alloinfections decreased with increasing genotype unit area (Table 3). The total number of infections for the mixtures inoculated focally increased with increasing genotype unit area, but much less so than for the general epidemics. There was a correspondingly smaller decline in the number of spores deposited on immune plants in the mixtures as gentoype unit area increased for focal than for general epidemics. The proportion of spores deposited on previously infected tissue was 0.063 for all mixture treatments and represented 48% of the total potential autoinfections for the mixture of 0.0025-m^2 genotype units but only 20% of the total potential autoinfections for the mixture of 0.56-m^2

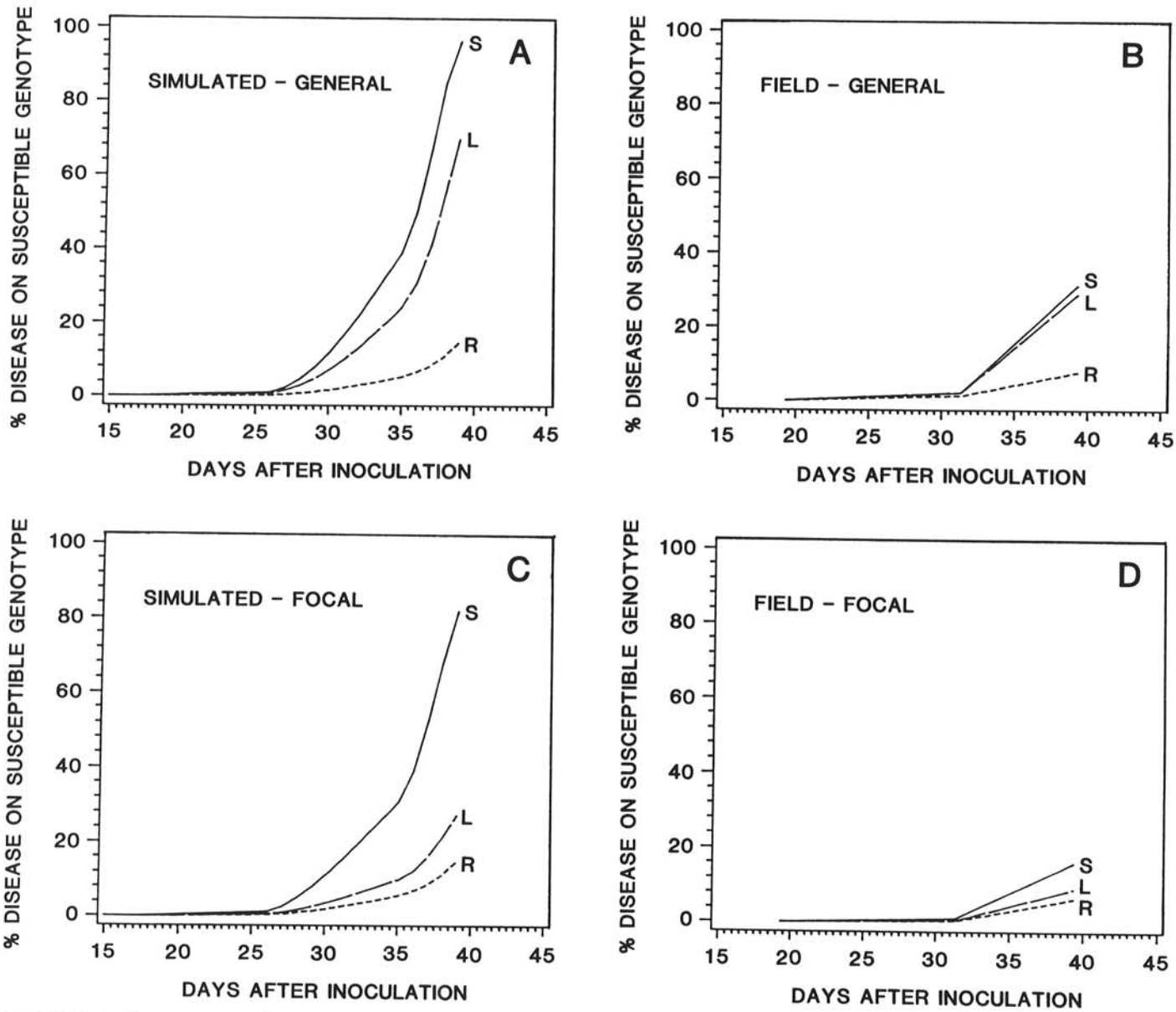

Fig. 3. Effects of host genotype unit area and spatial distribution of initial disease on the increase of crown rust in pure-line susceptible oat populations and two mixtures of 25% susceptible and 75% immune oat plants for field- and computer-simulated epidemics. **A and C,** Computer-simulated increase of crown rust on susceptible plants in 9.0-m^2 plots of 3,600 simulated oat plants. S = pure-line susceptible population; L = mixture in which 16 units of 225 plants of the same genotype were randomly positioned within plots (genotype unit area = 0.5625 m^2); R = mixture in which each plant was randomly positioned within plots (genotype unit area = 0.0025 m^2). Disease progress curves for the mixtures are means of four different randomizations of host units. The disease progress curve for the pure-line susceptible treatment was not replicated. In **A,** all epidemics were initiated with 36 spores per plant on each of 32 plants spaced uniformly within each plot (total of 1,152 spores per plot). In **C,** epidemics were initiated with 32 spores per plant on each of the 36 plants at the center of each plot (total of 1,152 spores per plot). **B and D,** Increase of crown rust on susceptible oat plants in 37.2 m^2 field plots. S = pure-line susceptible population; L = mixture in which seeds were aggregated into 64 randomly positioned blocks of 200 seeds of the same genotype; R = completely random mixture. Each curve is the mean of three replications. Data were adapted from Mundt and Leonard (11) by assuming that the carrying capacity of a culm was 1,875 pustules. For **B,** epidemics were initiated by inoculating 128 culms at the center of each plot. For **D,** epidemics were initiated by inoculating two culms in each of 64 quadrats of each plot.

genotype units. There was a larger proportion of spores deposited on previously infected tissue in the pure-line susceptible population than in the mixtures because there were four initially infected plants adjacent to each other in the pure-line susceptible population, but only one initially infected plant in the mixtures.

Effect of size of initial focus. The effect of the size of the initial disease focus on epidemic development can be seen by comparing epidemics in which initial inoculum was restricted to the central 900 compartments (large focus) (Fig. 7) with epidemics in which initial inoculum was restricted to the central four compartments (small focus) (Fig. 5) and the general epidemics (Fig. 4). Disease progress curves for the epidemics with a large initial disease focus were more similar to those with a small initial focus than to the general epidemics. However, there were a few differences in disease progress curves between mixtures in the small-focus versus large-focus epidemics. Early in the epidemics there were larger differences among mixture treatments in the epidemics initiated with a large focus than in epidemics initiated with a small focus. Later in the epidemics, however, there was less variation among mixture treatments with the larger than with the smaller initial foci. In addition, midway through the epidemics the disease progress curve for the mixture of 0.56-m^2 genotype units dropped below that of the mixtures with genotype unit areas of 0.25 and 0.090 m^2 for epidemics initiated with a large focus. When the carrying capacity was raised to prevent focus saturation, however, the mixture of 0.56-m^2 genotype units was the most severely diseased treatment throughout the epidemic (Table 4). For epidemics initiated with a large focus, the disease progress curve for the mixture with a genotype unit area of 0.010 m^2 was always intermediate between the curves for the mixtures with 0.0025- and 0.022-m^2 genotype units and was omitted from Figure 7 for clarity of presentation.

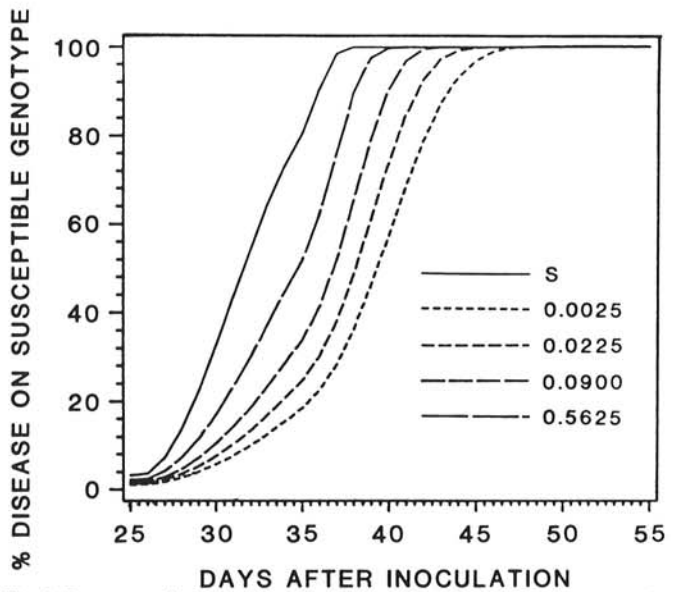

Fig. 4. Computer-simulated increase of crown rust on susceptible plants for a pure-line susceptible oat population and mixtures of 25% susceptible and 75% immune plants in plots of 3,600 oat plants. Mixtures with genotype unit areas of 0.0025, 0.0225, 0.0900, and 0.5625 m^2 were obtained by aggregating plants of the same genotype into units (Fig. 2). Epidemics were initiated with one spore on each of the 3,600 plants in each plot.

DISCUSSION

Performance of the model. There are several artificialities associated with the model used in this study. The effects of weather variability on epidemic development were not accounted for and the model assumes that the latent period, the infectious period, and leaf area index are constant over time. Although infections do not result from fractional spores in nature, we included fractional spores in our simulations because we viewed rates of dispersal among plants as population averages that can be represented by fractional spores and because cumulating spore fractions into whole spores can result in errors when calculating autoinfection rates for single genotype units. With our spore dispersal subroutine, we assumed that all spores produced from a

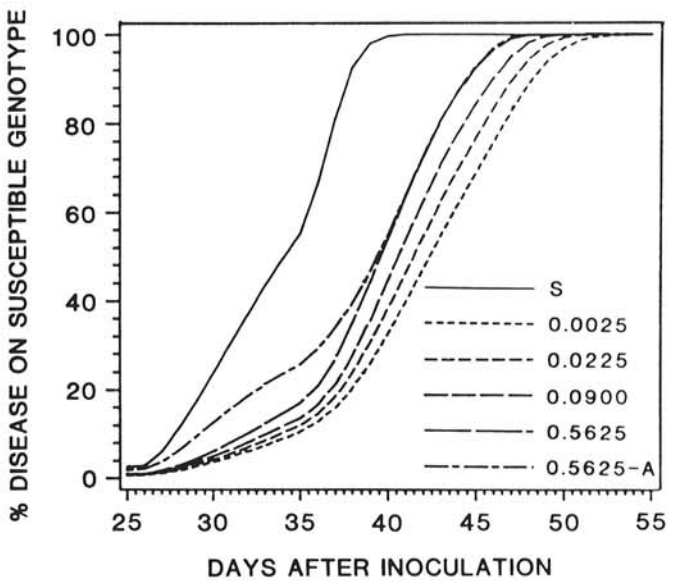

Fig. 5. Computer-simulated increase of crown rust on susceptible plants for a pure-line susceptible oat population and mixtures of 25% susceptible and 75% immune plants in plots of 3,600 oat plants. Host populations were the same as in Figure 4. For the treatment designated "Mixture 0.5625-A," the epidemic was initiated with 900 spores on the plant at the center of the susceptible 0.5625-m^2 genotype unit nearest plot center (see Fig. 2). For all other treatments, epidemics were initiated with 900 spores on each of the four plants at the center of each plot.

TABLE 1. Percent disease on susceptible plants at six times after inoculation for crown rust in a pure-line susceptible oat population and in mixtures of 25% susceptible and 75% immune plants for computer-simulated epidemics initiated with 900 spores on each of the central four plants in each plot of 3,600 oat plants; the carrying capacity (maximum number of pustules per plant) was set at 2.50×10^9 to avoid saturating the initially inoculated areas with infections

Population	Genotype unit area (m^2)	Days after inoculation[a]					
		13	22	31	40	49	58
Pure-line	9.0000	4.00 −10[c]	1.80 −8	5.05 −7	1.23 −5	3.02 −4	7.89 −3
Mixture	0.0025	4.00 −10	6.35 −9	6.96 −8	7.45 −7	8.61 −6	1.03 −4
Mixture	0.0100	4.00 −10	6.83 −9	7.98 −8	9.04 −7	1.10 −5	1.41 −4
Mixture	0.0225	4.00 −10	7.05 −9	8.67 −8	1.03 −6	1.33 −5	1.80 −4
Mixture	0.0900	4.00 −10	7.32 −9	9.94 −8	1.32 −6	1.91 −5	2.95 −4
Mixture	0.2500	4.00 −10	7.44 −9	1.08 −7	1.57 −6	2.49 −5	4.26 −4
Mixture	0.5625	4.00 −10	7.49 −9	1.14 −7	1.75 −6	2.98 −5	5.50 −4
Mixture[b]	0.5625	4.00 −10	1.27 −8	2.50 −7	4.39 −6	8.04 −5	1.56 −3

[a] Days after inoculation are midpoints of 9-day latent periods during the epidemics.
[b] Mixture inoculated with 900 spores on the center plant in the susceptible genotype unit nearest to the center of the plot.
[c] Negative values are exponents of 10, e.g., $1.80 -8 = 1.80 \times 10^{-8}$.

compartment would be contained within an area four times the size of the block under study. Others have used the integral (or a numerical approximation of the integral) of dispersal equations to describe probability density (8,13). We found that the numerical approximation of the integral of equation 1 inadequately described primary disease gradients obtained from the field, perhaps for reasons that were discussed previously (10).

The choice of an initial $2N \times 2N$ matrix to calculate dispersal probabilities was arbitrary and the $2N \times 2N$ matrix was the minimum size that could be used to account for dispersal between the two most widely separated compartments of the $N \times N$ matrix (i.e., between compartments at opposite ends of the diagonals of simulated plots). Choosing a different-sized initial matrix would change the proportion of spores dispersed out of a plot but would not affect the relative numbers of spores deposited in different compartments within the plot.

Due to limitations of the model, we were unable to duplicate variables of the field epidemics accurately in some cases. For example, EPIMUL uses a constant latent period whereas, in the field, the latent period was longer earlier than later in the epidemic due to changing temperatures as the season progressed. In addition, cost limitations restricted us to using a simulated plot size about one-fourth the area of plots used in the field experiment.

Comparison of the simulations with field data indicates that simulated oat crown rust epidemics responded to genotype unit area and spatial distribution of initial disease in a manner similar to that observed in Mundt and Leonard's field epidemics (11), i.e., the decline in mixture efficacy with increasing genotype unit area was much stronger for general than for focal epidemics. Results of the simulations are also consistent with other biological studies on the effect of genotype unit area on epidemic development (2,5,9). A given level of disease was attained sooner in the simulated than in

TABLE 2. Fate of spores produced on the first day of spore production for crown rust in a pure-line susceptible oat population and in mixtures of 25% susceptible and 75% immune plants in which computer-simulated epidemics were initiated with one spore on each of 3,600 oat plants in each plot

Population	Genotype unit area (m^2)	Proportion of spores accounted for by[a]			
		Auto[b]	Allo[c]	Total[d]	Immune[e]
Pure-line	9.0000	0.773	···	0.773	···
Mixture	0.0025	0.131	0.140	0.271	0.502
Mixture	0.0100	0.184	0.112	0.296	0.477
Mixture	0.0225	0.229	0.092	0.322	0.451
Mixture	0.0900	0.332	0.058	0.390	0.383
Mixture	0.2500	0.423	0.035	0.458	0.316
Mixture	0.5625	0.501	0.019	0.520	0.253

[a] For all treatments, the proportion of spores dispersed out of the plot was 0.227.
[b] Proportion of spores accounted for by autoinfections on susceptible genotype units.
[c] Proportion of spores accounted for by alloinfections on susceptible genotype units.
[d] Total proportion of spores accounted for by infections on susceptible genotype units (sum of autoinfections + alloinfections).
[e] Proportion of spores deposited on immune genotype units.

TABLE 3. Fate of spores produced on the first day of spore production for crown rust in a pure-line susceptible oat population and in mixtures of 25% susceptible and 75% immune plants in which computer-simulated epidemics were initiated with 900 spores on each of the central four plants in each plot of 3,600 oat plants

Population	Genotype unit area (m^2)	Proportion of spores accounted for by[a]				
		Auto[b]	Allo[c]	Total[d]	Immune[e]	PIT[f]
Pure-line	9.0000	0.793	···	0.793	···	0.088
Mixture	0.0025	0.068	0.166	0.235	0.583	0.063
Mixture	0.0100	0.121	0.138	0.258	0.559	0.063
Mixture	0.0225	0.152	0.118	0.270	0.548	0.063
Mixture	0.0900	0.202	0.082	0.283	0.535	0.063
Mixture	0.2500	0.235	0.054	0.289	0.529	0.063
Mixture	0.5625	0.258	0.033	0.291	0.526	0.063

[a] For all treatments, the proportion of spores dispersed out of the plot was 0.119.
[b] Proportion of spores accounted for by autoinfections on susceptible genotype units.
[c] Proportion of spores accounted for by alloinfections on susceptible genotype units.
[d] Total proportion of spores accounted for by infections on susceptible genotype units (sum of autoinfections + alloinfections).
[e] Proportion of spores deposited on immune genotype units.
[f] Proportion of spores deposited on previously infected tissue.

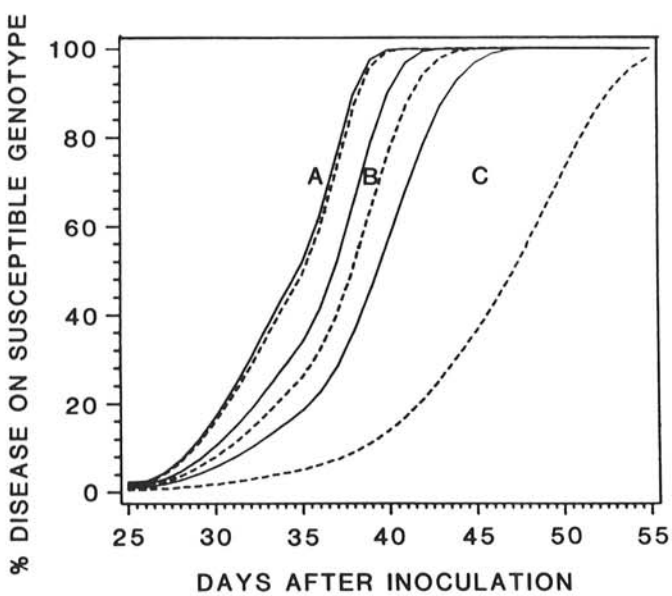

Fig. 6. Computer-simulated increase of crown rust on susceptible oat plants in mixtures of 25% susceptible and 75% immune plants (solid lines) in plots of 3,600 oat plants and for single, susceptible genotype units surrounded by immune plants in plots of 3,600 oat plants (dashed lines). A, Genotype unit area = 0.5625 m^2; B, genotype unit area = 0.0900 m^2; C, genotype unit area = 0.0025 m^2. Epidemics were initiated with one spore on each plant.

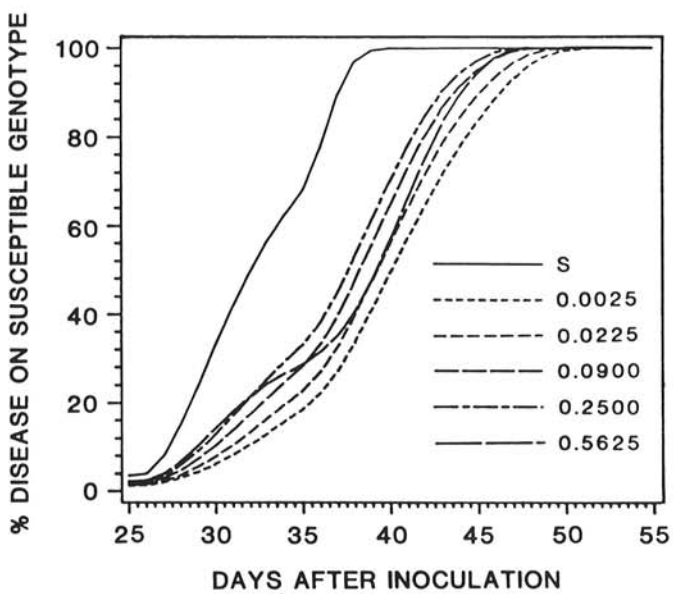

Fig. 7. Computer-simulated increase of crown rust on susceptible plants for a pure-line susceptible oat population and mixtures of 25% susceptible and 75% immune plants in plots of 3,600 oat plants. Mixtures with different genotype unit areas were simulated by aggregating plants of the same genotype into units (Fig. 2). Epidemics were initiated with four spores on each of the central 900 plants of each plot.

the field epidemics probably because cool temperatures significantly lengthened the latent period during the beginning of the field epidemics. We chose a latent period for the simulations that we felt would be representative of the latent period in the later stages of the field epidemics when most of the disease increase occurred.

The difference between disease progress curves for focal and general epidemics in the simulations was smaller than that observed with the field epidemics. This discrepancy may be due to interactions between the spatial distribution of initial disease and plot size. When initial disease is highly aggregated and infected plants are adjacent to each other, a larger proportion of inoculum will be deposited on previously infected tissue than if the initial disease is more widely dispersed and infected plants are adjacent to uninfected or lightly infected ones. If the focus of initial disease is at the center of a plot, however, a smaller proportion of inoculum will be dispersed out of the plot than if disease were more widely dispersed and there were more infected plants closer to plot edges.

Interactions between deposition on previously infected tissue and dispersal out of the plot will determine the difference in the rate of epidemic development between focal and general epidemics and this difference may change as the severity and spatial distribution of infections changes during the course of epidemics. For example, in the pure-line susceptible treatments of the simulations shown in Figure 3A and C, there was more disease in the focal than in the general epidemics until 30 days after inoculation, after which there was less disease in plots receiving a focal inoculation than in plots receiving a general inoculation. The plots used in the field study (11) were about four times the area of our simulated plots, and field experiments have shown that there is a larger amount of spore loss from small plots than from larger ones (3,6,13). Thus, differences in the amount of spore loss from plots between focal and general epidemics may have counteracted the effect of focus saturation more in the simulated epidemics than in the field studies.

We used an ordered rather than a random arrangement of genotype units to study mechanisms of disease control in host mixtures to eliminate the need for replicating the mixture simulations (the only stochastic feature of the simulation model is the randomization of genotype units) and so that dispersal among genotype units would be spatially symmetrical and, therefore, easier to study. Because responses to genotype unit area and spatial distribution of initial disease were similar in the ordered and random populations, we felt justified in using data from the ordered populations to study mechanisms of disease control in mixtures.

Mechanisms of disease control in host mixtures. Differences among disease progress curves for mixtures in the general epidemics can be easily explained from Table 2. As the size of genotype units increased, the increase in the number of autoinfections was greater than the decline in the number of alloinfections. We chose to evaluate the epidemics on the first day that second generation pustules appeared because later in the epidemic it would be much more difficult to determine sources of infection and to account for spore loss on previously infected tissue. The proportions given in Table 2 will change during the course of epidemics due to spore deposition on previously infected tissue. Because disease is distributed relatively uniformly in a general epidemic, however, we expected that the ratio of autoinfection to alloinfection would remain relatively constant during the epidemic.

Our results suggest that, with small grain rusts, disease development within genotype units becomes increasingly independent of disease development in other genotype units as the size of genotype units increases (Table 2 and Fig. 6). The rate of inoculum exchange among genotype units can be of great importance when initial disease is confined to a focus (see following paragraphs) and may also influence the degree of selection for simple versus complex pathogen races in host mixtures (1,2).

Dividing the spore population into categories is less useful for focal epidemics (Table 3) than for general epidemics because the spatial distribution of disease and, therefore, the relative distribution of spores into categories may vary greatly as epidemics progress. In addition, the amount of initial disease will affect the number of spores deposited on previously infected tissue in the initially inoculated genotype unit, thereby influencing the proportion of autoinfections. Nevertheless, the data of Table 3 illustrate mechanisms that may account for the relatively small effect of genotype unit area on the efficacy of the simulated mixtures, at least for the early stages of epidemic development. The decline in the proportion of spores accounted for by alloinfections was not drastically different for the focal as compared with the general epidemics. For focal epidemics, however, there was a much smaller increase in the proportion of autoinfection as genotype unit area increased than was found with general epidemics.

In the simulated focal epidemics in ordered host populations, initial inoculum was introduced at the center of each plot at the junction of one susceptible and three immune genotype units. This inoculation procedure guaranteed that 25% of the initial inoculum would be deposited on susceptible plants in every mixture treatment, but it caused a bias that reduced the amount of autoinfection in the second generation of disease increase for mixtures with large genotype units. For example, in the mixture of 0.56-m^2 genotype units, the initially inoculated compartment was at the corner of a susceptible genotype unit and spores produced from pustules resulting from the initial inoculation dispersed fewer spores to susceptible plants than they did when the initial inoculum was placed in the center of a susceptible 0.56-m^2 genotype unit.

We found that epidemics initiated with inoculum placed at the center of a 0.56-m^2 susceptible genotype unit initially had much more disease than epidemics initiated with inoculum placed at the corner of the susceptible 0.56-m^2 genotype unit because a greater amount of inoculum was maintained within the genotype unit that was inoculated centrally. Later in the epidemic, however, disease progress curves for the two treatments became nearly identical. Presumably, this was because the initially inoculated genotype unit

TABLE 4. Percent disease on susceptible plants at six times after inoculation for crown rust in a pure-line susceptible oat population and in mixtures of 25% susceptible and 75% immune plants for computer-simulated epidemics initiated with four spores on each of the 900 plants at the center of each plot of 3,600 plants; the carrying capacity (maximum number of pustules per plant) was set at 2.50×10^9 to avoid saturating the initially inoculated genotype units with infections

Population	Genotype unit area (m^2)	Days after inoculation[a]					
		13	22	31	40	49	58
Pure-line	9.0000	4.00 −10[b]	1.77 −8	4.89 −7	1.17 −5	2.86 −4	7.42 −3
Mixture	0.0025	4.00 −10	6.28 −9	6.80 −8	7.23 −7	8.29 −6	9.89 −5
Mixture	0.0100	4.00 −10	6.75 −9	7.82 −8	8.79 −7	1.07 −5	1.35 −4
Mixture	0.0225	4.00 −10	7.25 −9	8.95 −8	1.07 −6	1.37 −5	1.84 −4
Mixture	0.0900	4.00 −10	8.54 −9	1.23 −7	1.67 −6	2.45 −5	3.79 −4
Mixture	0.2500	4.00 −10	9.79 −9	1.60 −7	2.45 −6	4.00 −5	6.94 −4
Mixture	0.5625	4.00 −10	1.09 −8	1.96 −7	3.29 −6	5.84 −5	1.11 −3

[a] Days after inoculation are midpoints of 9-day latent periods during the epidemic.
[b] Negative values are exponents of 10, e.g., 1.77 −8 = 1.77×10^{-8}.

in both treatments became "saturated" with infections and the majority of new infections were occurring from spores dispersed from the initially infected genotype unit to other susceptible units in the population. This hypothesis is supported by data of Table 1, which show that, when the carrying capacity was raised to prevent saturation of the focus, there is a large difference in the amount of disease for the two mixtures of 0.56-m^2 genotype units throughout the epidemic.

Mundt and Leonard (11) hypothesized that the interaction between the size of host genotype units and the spatial distribution of initial disease could be caused by differences in rates of epidemic development within and between host genotype units. They suggested that, in a focal epidemic, the increased rate of epidemic development within large genotype units may be offset by a decreased amount of spread between genotype units. This is because, if the total area occupied by a host genotype is constant, the distance between host units will increase as the size of host units increases. On the other hand, when the initial disease is more uniformly distributed, most or all genotype units may be initially infected and the increased rate of epidemic development within large genotype units will be the dominant force. Burdon and Chilvers (5) came to a similar conclusion in studying the effect of host aggregation on the epidemic progression of damping-off disease. Results from the simulations confirm that, as the area of host genotype units increases, epidemic development within genotype units increases and the amount of inoculum exchange among host units is strongly diminished. The simulations also indicate that "saturation" of the initially infected host unit(s) with infections plays an important role in determining the effect of genotype unit area on development of focal epidemics. If the initial focus never becomes saturated with infections, disease can continue to increase rapidly in the large genotype units at the focus.

The size of the initial disease focus may also influence the effect of genotype unit area on mixture efficacy. In the simulations, the disease progress curve for the mixture of 0.56-m^2 genotype units dropped below the curves for mixtures with 0.25- and 0.090-m^2 genotype units midway during the epidemic when initial inoculum was dispersed over the central 900 plants in the simulated plots (Fig. 7). This may have been because all initially inoculated susceptible plants in the mixture of 0.56-m^2 genotype units were contained within a single genotype unit that occupied only one-fourth of the area initially inoculated. In all other mixture treatments, there were initially infected genotype units (or parts of initially infected genotype units) spread out over a larger proportion of the initially inoculated area. Therefore, we hypothesized that the epidemic initially progressed faster in the mixture of 0.56-m^2 genotype units because of increased autoinfection in the larger genotype unit. Once the initially inoculated genotype unit became saturated with infections, however, the epidemic in the mixture of 0.56-m^2 genotype units may have been slower than in other treatments because infections were confined to a smaller area. When we ran the same simulations as in Figure 7 but with the carrying capacity raised to 2.5×10^9 pustules per plant, the mixture of 0.56-m^2 genotype units was the most heavily rusted mixture treatment throughout the epidemic (Table 4).

Practical implications. Results of the simulations suggest that host mixtures may provide considerable protection against rust epidemics in crops with large plants and in cropping systems where plants of the same genotype are highly aggregated, if the epidemic begins from a restricted area and spreads through the crop. Observations suggest that diversification programs using very large genotype units can provide disease control in practice. For example, Browning et al (4) discussed evidence indicating that the deployment of resistance genes in different regions of North America can provide considerable protection against small grain rusts. In North America, rust epidemics often progress from the southern to the northern part of the continent (4). In the United Kingdom, interfield diversification of cultivars is used to reduce the spread of stripe rust (induced by *Puccinia striiformis* West.) on winter wheat and powdery mildew on spring barley (14,15). It is difficult to determine what role the spatial distribution of initial disease plays in determining the effectiveness of interfield diversification. In Europe, initial infections of powdery mildew on spring barley tend to be generally distributed (18), whereas stripe rust epidemics on winter wheat often begin focally (19).

There are variables that we held constant in our simulations that may influence the effects of genotype unit area and spatial distribution of initial disease on mixture efficacy. We have used the model described in this paper to investigate the effects of variables such as the steepness of the dispersal gradient, the proportion of susceptible host tissue, and the rate of multiplication per lesion on the interaction between genotype unit area and the spatial distribution of initial disease. Results of these studies will be reported in a forthcoming paper.

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