Influence of Number of Host Genotype Units on the Effectiveness of Host Mixtures for Disease Control: A Modeling Approach

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ABSTRACT

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Analytical and simulation models were used to study the influence of the number of host genotype units (individual, independent units of host tissue that are genetically homogeneous) on the development of epidemics in pure and mixed (diverse) stands of crops. Analytical models indicated that the alloinfection/autoinfection ratio and, therefore, disease control derived from genetic diversification is greater for populations with a large number of host genotype units than for populations with a smaller number of host genotype units. For computer simulations of wheat stem rust over large geographic areas, the effectiveness of interfield diversification for disease control increased with increasing number of fields planted to wheat. The absolute severity of disease, however, increased for both diversified and nondiversified regions when the number of fields was increased. Simulation studies of oat crown rust for small plots showed that the number of host genotype units in a population is a more important determinant of the effectiveness of mixtures for disease control than is host genotype unit area (the ground area occupied by a host genotype unit). Results suggest that intraspecific or interspecific mixtures of large plants and the culture of alternating rows, swaths, or fields of different host genotypes may provide greater disease control than previously anticipated.

Additional keywords: *Avena sativa*, cultivar mixtures, epidemiology, genetic diversity, multilines, oats, *Puccinia coronata*, *Puccinia graminis*, *Triticum aestivum*.

An important variable that influences the effectiveness of host mixtures for disease control is host genotype unit area (16). A host genotype unit (genotype unit) is an individual, independent unit of host tissue that is genetically homogeneous, and host genotype unit area (genotype unit area) is the ground area occupied by a genotype unit. For example, in a random mixture of plants of different genotypes the individual, independent unit that is genetically homogeneous is a single plant, and the genotype unit area is the ground area occupied by that plant. When individual fields are genetically homogeneous but different resistance genes or plant species are deployed among fields, the genotype unit is a field and the genotype unit area is the area of the field.

The greater the ratio of alloinfections to autoinfections (22) in a genetically homogeneous host population, the greater will be the potential for host mixtures to control that disease. In this paper, autoinfections are considered to be infections resulting from spores produced on the same genotype unit, whereas alloinfections are defined as infections resulting from spores produced on other genotype units in the host population. Host mixtures reduce the number of alloinfections, because inoculum produced on one host genotype will be avirulent on other host genotypes. Autoinfections induced by asexually reproducing pathogens are not reduced by mixtures, however, because inoculum produced on a genotype unit will be virulent on that host genotype. Therefore, mixtures with large genotype units (e.g., genotypic mixtures of a crop with large plants or the culture of alternating rows, swaths, or fields of different genotypes) may not provide very effective disease control because a large proportion of inoculum may be retained on the same genotype to produce autoinfections (1,19,24).

Both field (18) and computer simulation (20) studies with oat (*Avena sativa*) crown rust (induced by *Puccinia coronata*) showed that mixtures of immune and susceptible plants became less effective for disease control as genotype unit area was increased from 0.003 m^2 (the ground area occupied by a single oat plant) to 0.6 m^2 (the ground area occupied by about 200 oat plants), if the initial disease was distributed uniformly. In these studies, mixtures with different genotype unit areas were obtained by altering the degree of aggregation of plants of the same resistance genotype within field or computer-simulated plots, while keeping constant the overall proportion of immune and susceptible plants and the overall area of the plots. These results suggest that, for rust diseases, mixtures may not provide effective disease control for crops with large plants or for strip-cropping or interfield diversification. However, results from these studies might have been different had the authors used larger experimental plots that contained a larger number of genotype units, because the number of genotype units in a host population will affect the alloinfection/autoinfection ratio, as will be described below.

The number of autoinfections per genotype unit per unit of disease depends on the area of the genotype unit and the steepness of the pathogen's dispersal gradient; it is independent of the number of other genotype units in the host population. The number of alloinfections per genotype unit, on the other hand, depends on both gradient steepness and the number of genotype units in the population. For small host populations with large genotype units, there will be a small number of units available to contribute alloinfections. Keeping constant the proportion and size of susceptible genotype units, the total number of genotype units available to contribute alloinfections will increase with increasing overall area of the host population.

There are two opposing factors influencing inoculum exchange among genotype units as the overall area of a host population increases, which can be demonstrated by considering the number of alloinfections on a susceptible genotype unit at the center of a host population. As the radius of the overall population increases, the total number of genotype units available to contribute alloinfections will increase with the square of the radius. On the other hand, new units added to the population by increasing its radius will be farther away and, therefore, each new genotype unit added to the perimeter of the host population will contribute fewer alloinfections to the central genotype unit than will a genotype unit closer to the center of the population.

This paper is divided into three distinct, but related, sections. In the first section, a generalized, analytical model is used to show

that the alloinfection/autoinfection ratio increases with increasing number of host units in a population. A hypothesis that is suggested by this model is that mixtures of large host units can be very effective for the control of plant disease when the number of host units is large. This hypothesis is tested in the second section of the paper by simulating the effects of interfield diversification on development of wheat (*Triticum aestivum*) stem rust (induced by *Puccinia graminis* f. sp. *tritici*). Results from the first two sections of the paper imply that both the area and number of host units influence the effectiveness of host mixtures for disease control. The relative importance of these two variables on disease control is quantified in the third section of the paper by using computer simulations of oat crown rust.

MATERIALS AND METHODS

Modeling the alloinfection/autoinfection ratio. Gregory (5) proposed that disease and spore dispersal gradients can be described by the equation

$$y = ax^{-b} \qquad (\#1)$$

in which y is the number of spores or infections per unit area at distance x from the edge of a spore source, b is a measure of the steepness of the gradient, and a, a measure of the source strength, is the number of spores or infections at one unit of distance from the source.

Equation 1 is inappropriate for modeling disease increase in host mixtures because y approaches infinity as x approaches 0 and, thus, the equation predicts an infinite number of autoinfections on the source. To counter this problem, Mundt and Leonard (17) suggested the use of the modified Gregory model

$$y = a(x' + c)^{-b} \qquad (\#2)$$

in which y and b are as in equation 1, x' is the distance from the center of the source to the center of a receptor of spores, a is the number of spores or infections at $1 - c$ units of distance from the source, and c is a positive constant that provides for prediction of a finite number of spores or infections at the source. Thus, at the source, $x' = 0$ and $y = ac^{-b}$. In fitting equation 2 to primary disease gradients around single maize (*Zea mays*), oat, and bean (*Phaseolus vulgaris*) plants infected with rust, Mundt and Leonard (17) found that c approximated the radius of the source plant. Data presented later in this paper suggest that equation 2 can also be used to describe disease gradients from an entire field of source plants by letting c equal the distance from the center to the edge of the field.

For both equations 1 and 2, distance can be defined in any desired unit (e.g., meters, inches, or rods) without affecting the value of b. However, equation 2 can be made more biologically meaningful by describing distance in units defined as the distance from the center to the edge of the spore source (the radius of the source if it is circular). To distinguish this normalized unit of measurement, equation 2 can be rewritten as

$$y = a(r + c)^{-b} \qquad (\#3)$$

in which r is distance expressed as the number of source radii from the center of the source to the center of a receptor of spores. If we let c equal the radius of the source, then c is equal to 1 and, therefore,

$$y = a(r + 1)^{-b} \qquad (\#4).$$

Because a represents the number of spores or infections at $1 - c$ units of distance from the source, then a in equation 4 is the number of spores per unit area at $1 - 1 = 0$ units of distance, which is the number of autoinfections on the source. Equation 4 is the model used to describe spore dispersal in this section of the paper.

The total number of genotype units in a host population (N_t) can be calculated by the equation

$$N_t = (r + 1)^{-b} \qquad (\#5)$$

in which r is the distance from the center of the central genotype unit to the center of a genotype unit on the perimeter of the host population, and r is measured in units equal to the distance from the center to the edge of a genotype unit (hereafter referred to as "radii" of the genotype unit). Equation 5 is valid for both circular and square host populations, but its validity can be most easily demonstrated by assuming square genotype units (Fig. 1). At the center of the block in Figure 1, $r = 0$ and $N_t = (0 + 1)^2 = 1$ genotype unit. If we move along a line perpendicular to a side of the central genotype unit to the center of a genotype unit in the first set of genotype units surrounding the central one, $N_t = (2 + 1)^2 = 9$ genotype units. For a block encompassing two sets of genotype units around the central one, $N_t = (4 + 1)^2 = 25$ genotype units.

The number of genotype units in a surrounding set of units that is r radii from the central genotype unit (N_s) can be determined by first calculating the total number of genotype units in a solid square of the same size and subtracting the number of genotype units in a solid square that is two radii smaller, that is,

$$N_s = (r + 1)^2 - (r - 1)^2 \qquad (\#6)$$

which simplifies to:

$$N_s = 4r \qquad (\#7).$$

Equation 4 gives the number of spores dispersed between genotype units that are r radii apart. Equation 7 gives the number of genotype units present in a surrounding set that is r radii from a central genotype unit. Therefore, multiplying equation 4 by equation 7 gives the total number of alloinfections on the central genotype unit that are contributed by all genotype units in a surrounding set that is r radii of from a genotype unit at the center of a population ($ALLO_s$). The product of this multiplication is:

$$ALLO_s = yN = 4ra(r + 1)^{-b} \qquad (\#8).$$

The total number of alloinfections contributed by all genotype units in the population to the genotype unit at the center ($ALLO_t$) can be calculated by summing over all surrounding sets with the equation

$$ALLO_t = \sum_{r=2}^{2 \rightarrow R} 4ra(r + 1)^{-b} \qquad (\#9)$$

in which R represents the distance to the outermost surrounding set of genotype units. Summations are done by intervals of two radii because the surrounding sets of genotype units are two radii apart.

To summarize, there are four important biological components of equation 9. The first is a summation that cumulates alloinfections to the central genotype unit for all sets of genotype units around it. The second is the term $4r$, which accounts for the number of genotype units in a surrounding set that is r radii from the source. The third is a, the number of autoinfections per genotype unit. Finally, the term $(r + 1)^{-b}$ defines the number of spores dispersed between any two genotype units that are r radii from each other.

Equation 9 was used to calculate the ratio of alloinfection to autoinfection on the central genotype unit for all combinations of host populations with radii ranging from $r = 0$ to 20 in increments of two and pathogens with b ranging from 1.0 to 3.0 in increments of 0.25. For these calculations, the number of autoinfections (a) was held constant at 1.0 per genotype unit.

Simulating effects of interfield diversification. A critical component in modeling the epidemiological effects of interfield diversification is the description of spore dispersal among fields. To describe dispersal among fields, we used data of Kingsolver et al (8), who studied the spread of wheat stem rust away from a single,

inoculated field of wheat on the island of St. Croix, U.S. Virgin Islands, where background infection was not a significant factor. We used data from Figure 20 of that publication, in which the number of pustules per culm is given for an inoculated source field of 2.4 ha and for fields at varying distances from the source. We analyzed pustule counts reported for the 26th day after the first sporulation was observed in the source field. On this date, pustule counts per culm were reported for the source field and for fields 2.7, 3.5, 6.9, and 10.5 km from the source field. Because counts from this date incorporated pustules resulting from pathogen generations after the primary one, the counts were adjusted for deposition of spores on previously infected tissue. This was done by multiplying each count by the quantity $1{,}000/(1{,}000 - y)$ in which y is the observed pustule count and 1,000 represents the maximum number of rust pustules expected per culm. The use of 1,000 as the maximum number of stem rust pustules per culm is based on the assumption of Kingsolver et al (9) that 10 pustules represents 1% disease severity.

To calculate b of the modified Gregory model (equation 2), $\log_{10}$ (adjusted pustule counts) was regressed on $\log_{10}$ (distance from the source + 0.0775 km), in which 0.0775 km represents the distance of a line measured perpendicularly from the center to the edge of a square, 2.4-ha field. The slope of this regression line was used to describe the steepness of stem rust dispersal.

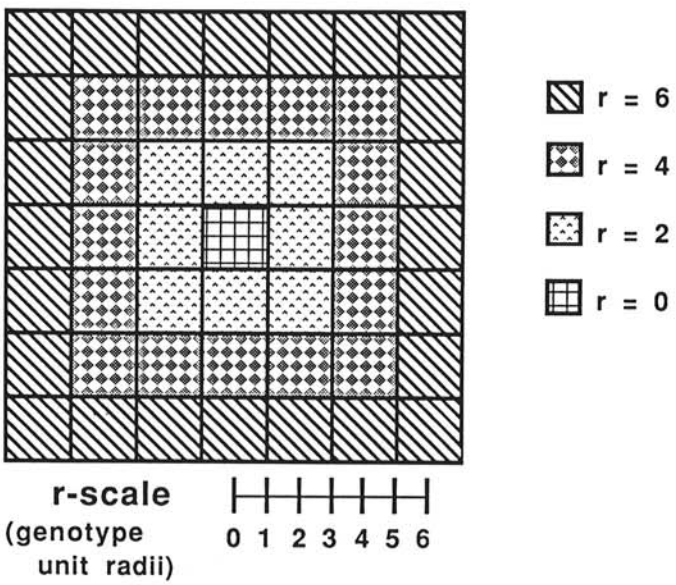

Fig. 1. Representation of the increase in the number of host genotype units (small squares) in surrounding sets of host genotype units (indicated by different fill-patterns) at varying genotype unit radii from a central host genotype unit.

To model the development of wheat stem rust, the modification by Mundt et al (20) of the simulation model EPIMUL (6) was adapted for use on the CYBER mainframe computer at Oregon State University. Briefly, EPIMUL divides a host population into a block of square compartments in which each compartment is a single host unit (in the present case, a single 2.4-ha wheat field). Compatibility values are assigned to make each compartment either immune or susceptible to the pathogen. The amount of disease in each compartment is updated on a daily basis and, at the end of each day, the simulator uses the modified Gregory model to disperse inoculum produced from each compartment over all compartments in the block.

Input values that were held constant for all simulations were: pustule size = 7.50 mm^2, length of the side of a field (compartment) = 155 m, leaf area index = 3.0, c of the modified Gregory model = 77.5 m, daily multiplication factor (number of pustules produced per pustule per day of the infectious period) = 10.0 pustules per pustule per day, latent period = 9 days, infectious period = 24 days, total population size (block size) = 32 × 32 fields, length of epidemic = 60 days. The spore-sweeping option, which cumulates spore fractions and disperses them as whole spores, was not used and simulations utilized noninteger values for spore numbers.

Epidemics were simulated for regions consisting of 4, 16, 64, 256, and 1,024 fields of 2.4 ha each. For each region size, epidemics were run where all fields were susceptible and also for genetically diversified regions where one-fourth of the fields was susceptible and the remaining three-fourths was immune (Table 1). For the diversified regions, the positions of the immune and susceptible fields were alternated in the same pattern as used in the study by Mundt et al (20) (Fig. 2). All epidemics were initiated with 960,000 effective spores (spores that will produce infections on a susceptible host genotype) per field on day 0 of the epidemic, giving an initial disease severity of 0.01% for each susceptible field.

In the modification by Mundt et al (20) of EPIMUL, the number of spores dispersed among compartments depends on the total number of compartments in the block. Therefore, for all simulations described in this section, the block size was 1,024 (32× 32 fields). For simulated regions of less than 1,024 fields, the appropriate number of fields was located at the center of the 32×32 block and all other compartments were assigned compatibility values to make them immune.

The value of b (1.745) calculated from the data of Kingsolver et al (8) was based on pustule counts downwind from the source, along prevailing tradewinds. The gradient averaged over all directions from the source would be steeper. Therefore, we repeated all of the simulations described above with $b = 2.5$, which is our best estimate of the steepness of the average gradient based on knowledge of rust dispersal and the modified Gregory model.

Two statistics were chosen to compare epidemic development among treatments. The first was the disease severity in susceptible fields on day 38 of the epidemic (the day before disease severity exceeded 90% in the most severely rusted treatment). The second

TABLE 1. Effect of crop area and interfield diversification on development of computer-simulated wheat stem rust epidemics in regions with different numbers of 2.4-ha fields, assuming two different steepnesses of dispersal gradient

Number of fields	Overall area (ha)	$b = 1.745$[a] ADPC[b] Nondiversified[d]	$b = 1.745$[a] ADPC[b] Diversified[e]	$b = 1.745$[a] % severity[c] Nondiversified	$b = 1.745$[a] % severity[c] Diversified	$b = 2.5$ ADPC Nondiversified	$b = 2.5$ ADPC Diversified	$b = 2.5$ % severity Nondiversified	$b = 2.5$ % severity Diversified
4	9.6	8.9	5.3 (0.60)[f]	1.82	0.97 (0.53)	92.2	69.5 (0.75)	26.85	19.63 (0.73)
16	38.4	17.6	6.6 (0.38)	4.08	1.26 (0.31)	127.3	75.1 (0.59)	38.08	21.39 (0.56)
64	153.6	37.3	9.0 (0.24)	9.72	1.86 (0.19)	171.6	82.5 (0.48)	51.89	23.75 (0.46)
256	614.4	77.2	13.3 (0.17)	22.13	2.95 (0.13)	218.3	90.5 (0.41)	65.45	26.32 (0.40)
1,024	2,457.6	148.6	20.4 (0.14)	44.83	4.86 (0.11)	261.2	98.1 (0.38)	76.62	28.74 (0.38)

[a] b represents the slope-value of the modified Gregory model (17) and measures steepness of the pathogen's dispersal gradient.
[b] Area under the disease progress curve for susceptible fields calculated from day 0 to day 38 after inoculation. Units are percent-days.
[c] Percent of maximum attainable disease severity at 38 days after inoculation for susceptible fields.
[d] All fields susceptible.
[e] One-fourth of fields susceptible and three-fourths immune.
[f] Numbers in parentheses represent the ADPC or % severity for the diversified region divided by the same statistic for the nondiversified region with the same number of fields and gradient steepness.

was the area under the disease progress curve (ADPC) for susceptible fields, using the equation of Mundt and Leonard (19). The ADPCs were calculated from day 9 (the first day that rust pustules appeared) until day 38. As an aid in making treatment comparisons, relative disease severities and relative ADPCs were calculated by dividing disease severity or ADPC for a diversified region by the disease severity or ADPC for the nondiversified region of the same overall area and with the same dispersal gradient steepness. Thus, for example, a relative ADPC of 0.60 would indicate that interfield diversification reduced the ADPC for susceptible fields to 60% of the ADPC for the nondiversified region of the same overall area.

Comparing effects of genotype unit area versus number of genotype units. To compare the effect of altering genotype unit area and the number of genotype units on the efficacy of mixtures for disease control, the results of Mundt et al (20) were re-evaluated and expanded. Mundt et al (20) simulated the development of oat crown rust in mixtures of 25% susceptible and 75% immune plants with different genotype unit areas. The various genotype unit areas were obtained by altering the degree of spatial aggregation of the two host genotypes in the EPIMUL model, while keeping the overall area of the simulated host populations constant at a 60 × 60 block of 3,600 compartments, in which each compartment represented a single oat plant (Fig. 2, F-K). We used data from that study and additional simulations using the same model and the same input parameters. Populations with a genotype unit area of 0.0025 m^2 were used, but the overall areas of the host populations were altered to obtain mixtures with differing numbers of genotype units (Fig. 2, A-F). These mixtures were always compared with pure-line susceptible populations of the same overall area. Combinations of genotype unit area, overall host area, and number of genotype units that were simulated are given in Table 2. In the most heavily diseased treatment, disease severity reached 90% on day 36. Disease severity on day 35 was thus used to calculate relative disease severity ratings, as described for the stem rust study.

Disease will increase more rapidly in host populations of large area than in populations of smaller area. Previous simulation studies indicated that mixtures are less effective in controlling fast epidemics as compared with slower ones (19), and analytical models have shown that this is caused, at least in part, by differences in the rate at which the host's carrying capacity for disease is approached (15). Two methods were used to eliminate the effect of different epidemic rates on mixture efficacy for disease

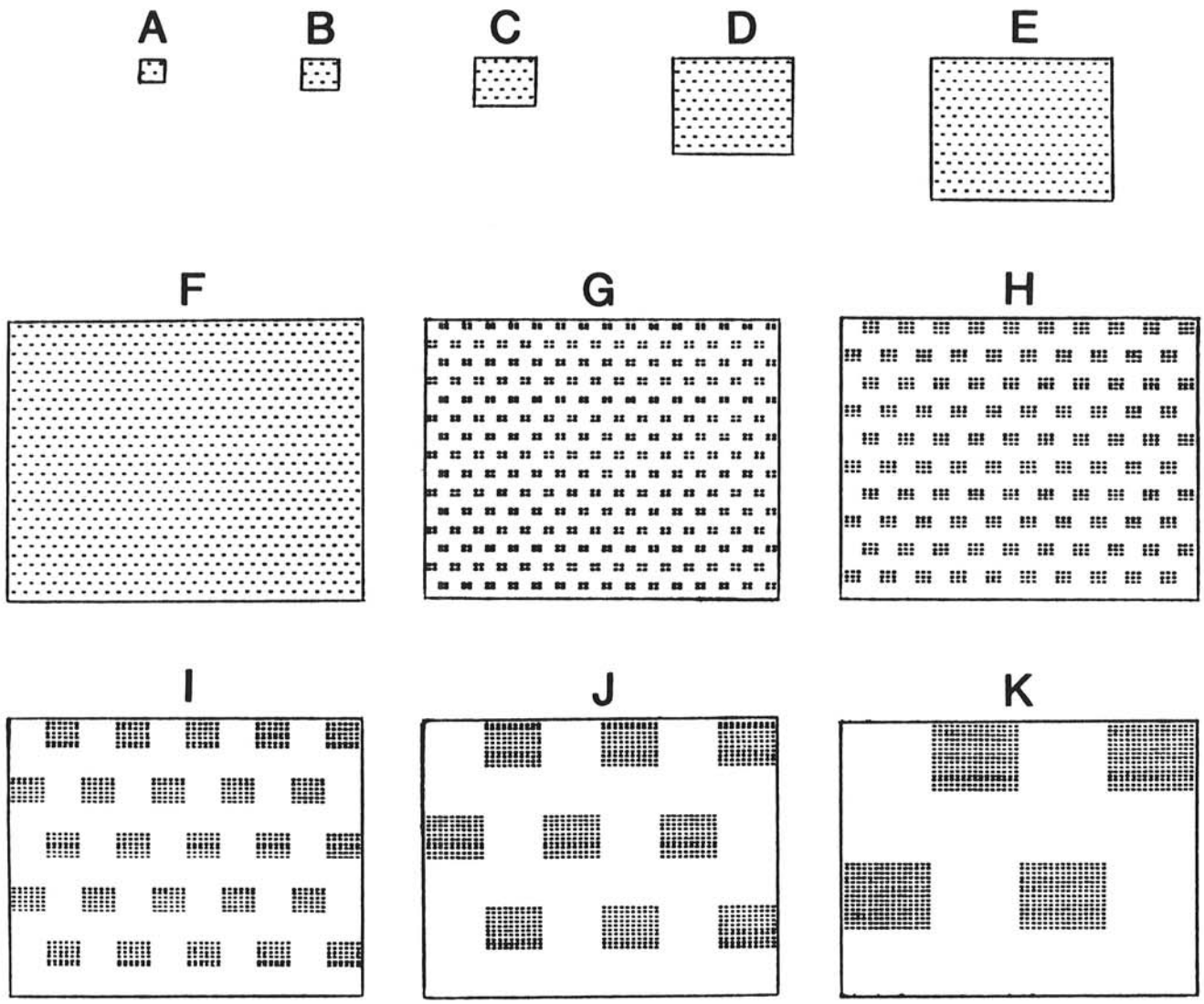

Fig. 2. Arrangement of host genotypes used to study the effect of host genotype unit area and number of host genotype units on development of computer-simulated oat crown rust epidemics. Each of the 11 blocks (A-K) represents a population of oat plants with 25% of plants susceptible and 75% immune to crown rust. Black dots represent susceptible plants, and blank spaces represent immune ones. **A-F,** host genotype unit area = 0.0025 m^2 and overall host area is 0.04 m^2 (**A**), 0.09 m^2 (**B**), 0.25 m^2 (**C**), 1.0 m^2 (**D**), 2.25 m^2 (**E**), and 9.0 m^2 (**F**), providing 16, 36, 100, 400, 900, and 3,600 genotype units per population, respectively. **F-K,** overall host area is 9.0 m^2 and genotype unit area is 0.0025 m^2 (**F**), 0.01 m^2 (**G**), 0.0225 m^2 (**H**), 0.09 m^2 (**I**), 0.25 m^2 (**J**), 0.5625 m^2 (**K**), providing 3,600, 900, 400, 100, 36, and 16 genotype units per population, respectively. Populations and plants appear rectangular rather than square because of limitations of the plotter used to produce the figure.

TABLE 2. Effect of varying the number of host genotype units in an oat population by altering genotype unit area with a constant overall host area and by altering overall host area with a constant genotype unit area[a]

Mixture pattern[e]	Overall area (m^2)	Genotype unit area (m^2)	Number of units	% severity[b]					
				Host tissue limiting[c]			Host tissue unlimiting[d]		
				PL[f]	MIXT[g]	REL[h]	PL	MIXT	REL
A	0.04	0.0025	16	18.2	6.5	0.36	1.50–7[i]	5.06–8	0.34
B	0.09	0.0025	36	26.0	7.7	0.29	2.24–7	5.98–8	0.27
C	0.25	0.0025	100	38.4	9.5	0.25	3.57–7	7.50–8	0.21
D	1.00	0.0025	400	56.7	12.7	0.22	6.10–7	1.10–7	0.18
E	2.25	0.0025	900	66.7	14.8	0.22	7.93–7	1.19–7	0.15
F	9.00	0.0025	3,600	80.4	18.5	0.23	1.15–6	1.52–7	0.13
G	9.00	0.0100	900	80.4	21.4	0.27	1.15–6	1.79–7	0.16
H	9.00	0.0225	400	80.4	24.6	0.31	1.15–6	2.10–7	0.18
I	9.00	0.0900	100	80.4	33.7	0.42	1.15–6	3.04–7	0.26
J	9.00	0.2500	36	80.4	43.2	0.54	1.15–6	4.15–7	0.36
K	9.00	0.5625	16	80.4	51.7	0.64	1.15–6	5.32–7	0.46

[a] Data are from computer-simulated oat crown rust epidemics in which healthy host tissue was and was not limiting to epidemic development.
[b] Percent of maximum attainable number of pustules for susceptible plants at 35 days after inoculation.
[c] Maximum attainable number of pustules per plant was 1,875.
[d] Maximum attainable number of pustules per plant was 2.5×10^9.
[e] Pattern of susceptible and immune plants as shown in Figure 2.
[f] Disease severity for pure-line susceptible population.
[g] Disease severity of susceptible plants for mixtures consisting of 25% susceptible and 75% immune plants.
[h] Relative disease severity, i.e., disease severity for a mixture divided by disease severity of the pure-line population of same overall area.
[i] Numbers following minus signs indicate negative exponents of 10, e.g., 1.5–7 is 1.5×10^{-7}.

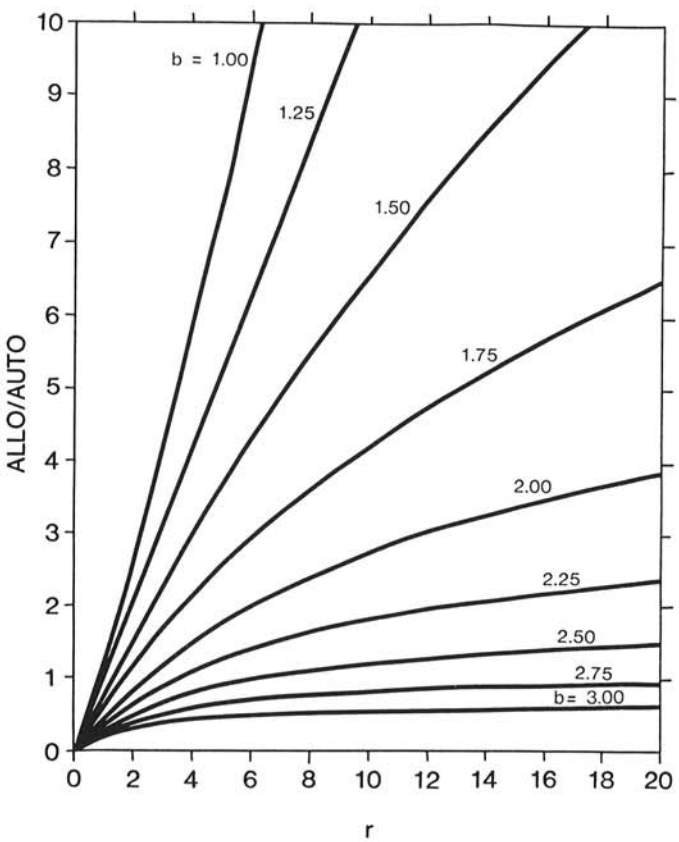

Fig. 3. Calculated alloinfection/autoinfection ratios versus radius of the host population for diseases caused by pathogens with different steepnesses of dispersal gradient, as measured by the slope value (b) of the modified Gregory model (17). The radius of the host population is measured in radii of an individual host genotype unit (r). Data were generated from equation 9 of the text.

control. The first was to alter the leaf area index and pustule size parameters to raise the carrying capacity to 2.50×10^9 pustules per plant, thus making healthy host tissue virtually unlimiting for infection. All of the treatments described in the previous paragraph were repeated with this altered carrying capacity. The second method was to make iterative simulations for a pure-line susceptible population with an overall area of 0.04 m^2 to find a value for the daily multiplication factor (the number of new lesions produced per infectious lesion per day of the infectious period = Vanderplank's [23] corrected basic infection rate) that would give the same rate of disease increase as for a pure-line susceptible population with an overall area of 9.0 m^2. Thus, we could compare epidemics with equal rates of disease increase in pure stands and their respective mixtures for two types of populations with a total of 16 genotype units, one with an overall area of 9.0 m^2 and a genotype unit area of 0.5625 m^2 and the second with an overall area of 0.04 m^2 and a genotype unit area of 0.0025 m^2.

RESULTS

Calculating the alloinfection/autoinfection ratio. The ratio of alloinfection to autoinfection for the central genotype unit increased when the number of genotype units was increased by expanding the overall area of the host population (Fig. 3). This increase was larger for shallow dispersal gradients than for steeper ones. Curves for the steeper dispersal gradients (e.g., $b > 2.0$) decreased in slope very rapidly as overall host area increased. It is important to note, however, that these curves never attain a slope of zero or less when the radius of the host population is expanded. Therefore, biologically significant increases in the alloinfection/autoinfection ratio can occur over large increases in overall host area, even when the pathogen's dispersal gradient is rather steep.

Simulating effect of interfield diversification. The modified Gregory model proved adequate for describing the disease gradient derived from the study by Kingsolver et al (8). Regression of $\log_{10}$ (corrected pustule counts) on $\log_{10}$ (distance from the source field + 0.0775 km) produced the equation $\log(y) = 1.465 + \log(x + 0.0775)(-1.745)$, with a coefficient of determination (r^2) of 0.95. The predicted, adjusted pustule count per field was 127, 0.25, 0.16, 0.049, and 0.024 for fields at 0, 2.7, 3.5, 6.9, and 10.5 km from the source, respectively. These predictions compare favorably with the observed, adjusted pustule counts of 140, 0.21, 0.07, 0.18, and 0.15 at the same respective distances from the source.

For both values of b (1.745 and 2.5), interfield diversification reduced disease development in susceptible fields relative to nondiversified regions of the same overall area (Table 1). As the number of fields per region increased, disease reduction attributed to diversification also increased. This effect was greater for the shallower dispersal gradient ($b = 1.745$) than for the steeper one ($b = 2.5$). Disease severity on day 38 and the ADPC gave similar results when used to calculate disease reduction due to

diversification.

For both diversified and nondiversified regions, disease severity and ADPC increased with increasing number of fields in the region. This increase was greater for the shallow than for the steep dispersal gradient and also was greater for the nondiversified than for the diversified regions.

Comparing effect of genotype unit area versus number of genotype units. Relative disease severity for the mixtures decreased with increasing number of genotype units. This was true whether the number of genotype units was increased by reducing genotype unit area or by increasing overall population area (Table 2). The only exception to this generalization was when the number of genotype units was increased from 900 to 3,600 by increasing the overall host area from 2.25 to 9.0 m^2 for epidemics with the normal carrying capacity of 1,875 pustules per plant. In this case, the small increase in relative disease severity (0.22 to 0.23) resulted from the more rapid approach of disease to the host's carrying capacity for the host population with the larger overall area. When healthy host tissue was made virtually unlimiting for infection by raising the carrying capacity to 2.5×10^9 pustules per plant, the disease-reducing effect of mixtures always increased with increasing number of genotype units. When the carrying capacity was raised to 2.5×10^9, the effect of the number of genotype units on relative disease severity was quantitatively more similar for the two methods of altering the number of genotype units than when the normal carrying capacity was used (Table 2).

The number of genotype units per population had a larger effect on relative disease severity than did genotype unit area (Table 2). Using the "host tissue unlimiting" simulations as examples, there was a 1.4-fold increase in relative disease severity (from 0.34 to 0.46) when genotype unit area was increased 225-fold (from 0.0025 to 0.5625 m^2), and the number of genotype units per population was held constant at 16 (genotype arrangement A vs. K of Fig. 2). In contrast, there was a 2.6-fold increase in relative disease severity (from 0.13 to 0.46) when the number of genotype units was decreased 225-fold (from 3,600 to 16) and genotype unit area was held constant at 0.0025 m^2 (arrangement F vs. A of Fig. 2).

Iterative simulations showed that nearly identical disease progress curves could be attained by using a daily multiplication factor of 28.6 pustules per pustule per day for a pure-line susceptible population of 0.04 m^2 and a daily multiplication factor of 10.0 for a 9.0-m^2, pure-line susceptible population. The disease severity on day 35 for the 0.04-m^2 population was 80.3%, and the severity for the corresponding 25% susceptible mixture (arrangement A of Fig. 2) was 38.6%, giving a relative severity rating of 0.48. Combining these data with those of Table 2 allows for a comparison of altering genotype unit area versus number of genotype units for epidemics that increased at the same rate in pure-line populations of different areas. There was a 1.3-fold increase in relative disease severity (from 0.48 to 0.64) when genotype unit area was increased 225-fold (from 0.0025 to 0.5625 m^2) and the number of genotype units per population was held constant at 16 (arrangement A vs. K of Fig. 2). In contrast, when the number of genotype units was decreased 225-fold (from 3,600 to 16) and genotype unit area was constant at 0.0025 m^2 (arrangement F vs. A of Fig. 2), there was a 2.1-fold increase in relative disease severity (from 0.23 to 0.48).

DISCUSSION

Results from the modeling studies indicate that the effect of genotype unit area on the efficacy of host mixtures for disease control depends strongly on the number of genotype units in the host population. Thus, the effect of genotype unit area on disease control in mixtures cannot be considered independently from the effect of the number of genotype units. Because mixtures control disease by reducing alloinfections, a high alloinfection/autoinfection ratio allows for effective disease control. Therefore, the increase in the alloinfection/autoinfection ratio with increasing number of genotype units shown in the first section of the paper means that the effectiveness of disease control should also increase. The simulations of wheat stem rust and oat crown rust confirm this hypothesis. Further, the oat crown rust simulations suggest that, when designing strategies for resistance gene deployment, the number of manipulable genotype units may be a more important variable to consider than genotype unit area. Therefore, suggestions that mixtures will be relatively ineffective for controlling diseases of crops with large genotype units (19,24) may not be accurate if mixtures contain a large number of genotype units. Similarly, strategies such as interfield diversification, as used in the U.K. (21), may be very effective for disease control if a large number of fields are involved.

The modeling procedures presented in this paper all assumed general epidemics (25) in which initial disease was uniformly distributed. It is also important to recognize that mixtures with large genotype units can be effective for controlling focal epidemics even when the number of genotype units is relatively low (16,18).

Results of the stem rust simulations suggest that the rate of disease increase is considerably less for small host regions than for larger ones. This confirms the view of Browning and Frey (3) that the decrease of oat crown rust severity during the 1970s in the midwestern U.S.A. was caused, in part, by a large decline in the number of hectares planted to oats. The suggestion is also supported by the observation of Lindemann et al (12) that the incidence of brown spot of bean, which is induced by *Pseudomonas syringae*, was greater in plots in the main bean-growing area of Wisconsin than in plots planted outside of the main bean-growing area.

The steepness of a pathogen's dispersal gradient has a critical influence on epidemic development over large areas and in determining the efficacy of host diversification to attain disease control. In the simulations with wheat stem rust, epidemics progressed faster with the steep as compared with the shallow dispersal gradient. This was probably because a greater proportion of spores was retained within the simulated regions when the gradient was steeper. This relationship between increasing steepness of gradient and increasing speed of epidemic will almost certainly reach a limit, however, when the gradient becomes so steep that a large amount of inoculum becomes ineffective due to deposition on previously infected host tissue (Mundt, unpublished). The calculated alloinfection/autoinfection ratio increased more rapidly with increasing radius of the host population for shallow than for steep gradients. This result suggests that host mixtures should have a stronger influence on epidemic development of diseases induced by pathogens with more shallow gradients, a view that is supported by earlier simulation studies (4,10,11,19).

Because of the steepness of many spore dispersal gradients, it is common for plant pathologists to disregard distant plants as a significant source of inoculum. Our models, however, demonstrate that distant genotype units can be a significant source of inoculum when all genotype units in a surrounding set at that distance are considered. For example, assume that the number of autoinfections per genotype unit is 1,000 and b of the modified Gregory model is 2.25. Under these conditions, equation 2 predicts that the central genotype unit in a population will receive only 1.01 spores, on the average, from a genotype unit that is 20 radii away. However, equation 8 predicts that the central genotype unit will receive 84.7 spores when all genotype units that are 20 radii from the source are considered.

Several factors contribute to a loss of realism in models used in this paper. For example, with the generalized model (equation 9), we calculated alloinfection/autoinfection ratios for the central genotype unit only. The average ratio for all host units in a population would be smaller, because a larger proportion of spores will be lost due to dispersal outside of the host population for host units near the edge as compared with those near the center of the host population. The average ratio could have been calculated by using computer simulation. However, we agree with the view of Jeger (7) that analytic approaches are generally better suited for the development of theory than are simulation approaches, and our equations are probably adequate to demonstrate the general effect of overall host area and steepness of pathogen dispersal gradients on the alloinfection/autoinfection ratio. In addition, it would have

entailed considerable cost to produce data shown in Figure 3 by using a computer simulation model.

Although the modified Gregory model quite accurately predicted pustule counts reported by Kingsolver et al (8), there are problems in using these data to predict the effects of interfield diversification on wheat stem rust control. Ideally, only primary disease gradient data resulting from the first generation of inoculum production should be used to estimate the dispersal of inoculum. Data from gradients influenced by later generations of inoculum production are less useful, because secondary spread may flatten gradients (5), and it is impossible to determine if spores spread over the entire observed range in a single movement or by shorter, step-wise "jumps." We accounted for the effect of gradient flattening by transforming pustule counts and plotting the logarithm of these adjusted counts versus the logarithm of distance from the source. This procedure is equivalent to plotting $\log(y/(1-y))$ when y is the proportion of disease severity and 1 represents the maximum disease severity that can occur. Other workers have found that the $\log(y/(1-y))$ transformation results in the calculation of a constant gradient steepness over several generations of plant disease increase (2,14).

The question of whether rust spores were dispersed over the 10.5 km distance in a single jump is less easy to answer. It was not until about 25 days after first sporulation in the source field that Kingsolver et al (8) reported pustules in the field closest to the source. Assuming a latent period of 8 days for wheat stem rust would suggest that these pustules could represent as many as three or four generations of disease increase after the primary infection. However, pustules may have been present in the fields earlier, but in undetectable numbers. For example, the disease gradient we calculated for wheat stem rust predicts that, on the average, about 550 wheat culms would have needed to be sampled in the field closest to the source field in order to detect a single rust pustule for the primary gradient when rust severity was still very low. For the field 10.5 km from the source, about 6,000 culms would have needed to be sampled. In fact, Kingsolver et al (8) reported that old pustules were observed later in the development of the epidemics, which indicated that stem rust was present in some fields earlier than was detected by their sampling procedure.

The fact that Kingsolver et al (8) measured disease only downwind from the source and that trade winds resulted in a very consistent wind direction made interpretation of gradient data more difficult. Because the current EPIMUL model assumes an equal gradient in all directions from the source, we used b 2.5 as an estimate of an average gradient based on our knowledge of stem rust and of the modified Gregory model. Although it is impossible to determine how accurate this estimate is, it at least provides an opportunity to demonstrate the importance of gradient steepness in influencing the effectiveness of genetic diversification for disease control. The purpose of the simulations was to demonstrate the potential importance of the number of fields in determining the effectiveness of interfield diversification for disease control, not to provide a precise, quantitative estimate of the effects of such a practice.

In using the modified Gregory model to describe primary gradients of rust away from single, infected plants of beans, oats, and maize, Mundt and Leonard (17) found that c of that equation approximated the radius of the source plant. In the present study, we found that the modified Gregory model adequately described a stem rust gradient away from a single, infected wheat field when c was assumed to be the distance measured perpendicularly from the center to the edge of a square field. Therefore, c of the modified Gregory model might generally be considered to be the radius of the source of spores.

The dispersal model used in our studies, an inverse power function, will not be appropriate for all plant pathogens. McCartney and Bainbridge (13) noted that an inverse power function is appropriate when the decrease of spores with distance is due entirely to diffusion and that a logarithmic function is appropriate when the decrease with distance is due only to the effect of spore deposition. A logarithmic function was found to fit spore dispersal and disease gradient data better than an inverse power function for spores of splash-dispersed pathogens (4). This might be expected, because such spores are dispersed in rather large water droplets that would have a high impaction efficiency on foliage. Therefore, our results may be less relevant to pathogens that are splash-dispersed.

Previous studies (1,4,10,11,19,20) have indicated that host genotype unit area, the spatial distribution of initial disease, the proportion of susceptible plants, the rate of disease increase, and the steepness of pathogen dispersal gradients are all factors influencing the efficacy of host mixtures for disease control. Results in this paper suggest that the overall host area and its influence on the number of host genotype units in a population must also be considered in determining the potential of host mixtures to control disease.

LITERATURE CITED

1. Barrett, J. A., and Wolfe, M. S. 1980. Pathogen response to host resistance and its implication in breeding programmes. OEPP (Eur. Mediterr. Plant Prot. Organ.) Bull. 10:341-347.
2. Berger, R. D., and Luke, H. H. 1979. Spatial and temporal spread of oat crown rust. Phytopathology 69:1199-1201.
3. Browning, J. A., and Frey, K. J. 1981. The multiline concept in theory and practice. Pages 37-46 in: Strategies for the Control of Cereal Disease. J. F. Jenkyn and R. T. Plumb, eds. Blackwell, Oxford. 219 pp.
4. Fitt, B. D. L., and McCartney, H. A. 1986. Spore dispersal in relation to epidemic models. Pages 311-345 in: Plant Disease Epidemiology, Vol. 1. K. J. Leonard and W. E. Fry, eds. MacMillan, New York. 372 pp.
5. Gregory, P. H. 1968. Interpreting plant disease gradients. Annu. Rev. Phytopathol. 6:189-212.
6. Kampmeijer, P., and Zadoks, J. C. 1977. EPIMUL, a Simulator of Foci and Epidemics in Mixtures of Resistant and Susceptible Plants, Mosaics and Multilines. Centre for Agricultural Publishing and Documentation, Wageningen, Netherlands. 50 pp.
7. Jeger, M. J. 1986. The potential of analytic compared with simulation approaches to modeling in plant disease epidemiology. Pages 265-281 in: Plant Disease Epidemiology, Vol. 1. K. J. Leonard and W. E. Fry, eds. Macmillan, New York. 372 pp.
8. Kingsolver, C. H., Peet, C. E., and Underwood, J. F. 1984. Measurement of the epidemiologic potential of wheat stem rust: St. Croix, U.S. Virgin Islands, 1954-57. Pa. Agric. Exp. Stn. Bull. 854. 20 pp.
9. Kingsolver, C. H., Schmitt, C. G., Peet, C. E., and Bromfield, K. R. 1959. Epidemiology of stem rust: II. (Relation of quantity of inoculum and growth stage of wheat and rye at infection to yield reduction by stem rust). Plant Dis. Rep. 43:855-862.
10. Kiyosawa, S. 1976. A comparison by simulation of disease dispersal in pure and mixed stands of susceptible and resistant plants. Jpn. J. Breed. 26:137-145.
11. Kiyosawa, S., and Shiyomi, M. 1972. A theoretical evaluation of the effect of mixing resistant variety with susceptible variety for controlling plant diseases. Ann. Phytopathol. Soc. Jpn. 38:41-51.
12. Lindemann, J., Arny, D. C., and Upper, C. D. 1984. Epiphytic populations of *Pseudomonas syringae* pv. *syringae* on snapbeans and nonhost plants and incidence of bacterial brown spot in relation to cropping patterns. Phytopathology 74:1329-1333.
13. McCartney, H. A., and Bainbridge, A. 1984. Deposition gradients near to a point source in a barley crop. Phytopathol. Z. 109:219-236.
14. Minogue, K. P., and Fry, W. E. 1983. Models for the spread of disease: Some experimental results. Phytopathology 73:1173-1176.
15. Mundt, C. C. 1988. Disease dynamics in agroecosystems. In: Ecology of Agricultural Systems. C. R. Carroll, J. H. Vandermeer, and P. M. Rossett, eds. Macmillan, New York. In press.
16. Mundt, C. C., and Browning, J. A. 1985. Development of crown rust epidemics in genetically diverse oat populations: Effect of genotype unit area. Phytopathology 75:607-610.
17. Mundt, C. C., and Leonard, K. J. 1985. A modification of Gregory's model for describing plant disease gradients. Phytopathology 75:930-935.
18. Mundt, C. C., and Leonard, K. J. 1985. Effect of host genotype unit area on epidemic development of crown rust following focal and general inoculations of mixtures of immune and susceptible oat plants. Phytopathology 75:1141-1145.
19. Mundt, C. C., and Leonard, K. J. 1986. Analysis of factors affecting disease increase and spread in mixtures of immune and susceptible plants in computer-simulated epidemics. Phytopathology 76:832-840.
20. Mundt, C. C., Leonard, K. J., Thal, W. M., and Fulton, J. H. 1986.

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. Phytopathology 76:590-598.
21. Priestly, R. H., and Bayles, R. 1982. Varietal diversification as a means of reducing the spread of cereal diseases in the United Kingdom. J. Natl. Inst. Agric. Bot. 15:205-214.
22. Robinson, R. A. 1976. Plant Pathosystems. Springer-Verlag, Berlin. 184 pp.
23. Vanderplank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.
24. Vanderplank, J. E. 1968. Disease Resistance in Plants. Academic Press, New York. 206 pp.
25. Zadoks, J. C., and Schein, R. D. 1979. Epidemiology and Plant Disease Management. Oxford University, New York. 427 pp.