Predicting Development of Epidemics on Cultivar Mixtures

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ABSTRACT

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A mathematical model for the development of an epidemic on a plant cultivar mixture illustrates the influence of the infection efficiency, spore production rate, proportion of deposited spores, frequency of autodeposition, and composition of the mixture on the genetic composition of the pathogen population and on the long-term rate of disease increase. In the model, the long-term composition of the pathogen population is determined from the long-term rates of disease increase of each pathotype. Alteration of any one of the model parameters may change the long-term composition of the pathogen population qualitatively. Detailed analysis of mixtures with two components showed that a pathotype reproducing on both components will predominate if the frequency of autodeposition is low and both components are present at intermediate frequencies. Predictions for different mixing strategies are given for host-pathogen systems where the host-pathogen interaction is described by a gene-for-gene relationship with "stabilizing selection." These mixing strategies include changing the composition of the mixture, the number of components in the mixture, the frequency of autodeposition (related to crop density), and finally mixing fields instead of plants. A relation was found between the long-term rate of disease increase of a pathotype and its number of virulence genes, and this relation was used for evaluating the different strategies.

Additional key words: autoinfection, complex races, disease resistance, epidemiology, partial resistance.

Yield loss caused by airborne foliar fungi is a serious problem in areas where cereal crops are intensively grown. During the last few decades, disease control has primarily been achieved by transferring single major resistance genes to commercial cultivars. However, because uniformity of a crop over large areas imposes strong selectional forces on pathogen populations, these successively introduced resistance genes have been overcome one by one. Currently, therefore, attempts are being made to reestablish genetic diversity by introducing new cropping methods. Growing multilines or mixtures of cultivars has been successful in decreasing the disease level judged from theoretical models (10,11,15) and field experiments (3,5,17).

There has, however, been considerable argument about the possible evolutionary outcome of the use of mixtures of cultivars (or multilines) with single major resistance genes. The problem is whether the selection for pathotypes able to attack many components leads to a buildup of a "super race" able to attack all components of the mixture and thus erode the resistance of the mixture. Models elucidating this aspect have considered gene-for-gene systems with "stabilizing selection" (16) in which "unnecessary virulence genes" are assumed to reduce the fitness of a pathotype (12). In the present paper, a model is analyzed that enables study of both disease level and composition of the pathogen population. In addition, by defining the model in terms of the quantitative parameters, infection efficiency, and spore production rate, it can be applied to all types of host-pathogen interactions. The model is simplified by assuming unlimited leaf area, but it includes a nontrivial dispersal function represented by the autodeposition.

THE MODEL

The model describes the epidemic development during a disease season in a field of a cultivar mixture of n components. The model assumes that the pathogen reproduces only asexually, that mutations do not occur, that the field is infected by incoming inoculum at the beginning of the season, and that any further external influx of spores does not occur.

The model has four epidemiological parameters. Two of these are reproduction parameters: the *infection efficiency* (the proportion of deposited spores that establish an infection; here, infection means a sporulating colony) and the *spore production rate* (the number of spores produced per day per infection). The other two are deposition parameters: the *proportion of spores deposited* (the proportion of produced spores that are deposited within the field) and the *frequency of autodeposition* (the proportion of deposited spores that are deposited on the donor plant that produced them). Three of the parameters are widely used for describing epidemics on monocultures (18), and the fourth (the frequency of autodeposition) is defined especially for heterogeneous host populations (cf, autoinfection in 14).

The pathogen population is divided into l classes (pathotypes, cf, 14) of individuals with identical parameters with respect to each of the n components of the mixture. The deposition parameters are assumed to be equal among all pathotypes on all components and are designated e ($0 \leqslant e \leqslant 1$, the proportion of spores deposited) and a ($0 \leqslant a \leqslant 1$, the frequency of autodeposition). The reproduction parameters are specific for each pathotype/host component combination; p_{vi} ($0 \leqslant p_{vi} \leqslant 1$) designates the infection frequency and s_{vi} ($0 \leqslant s_{vi}$) the spore production rate for pathotype i on component v. The parameters are unchanged during the disease season.

The epidemic development of pathotype i on an isolated monoculture of component v is considered first. The number of infections on the total leaf area at time t is designated $N_{vi}(t)$. In a small time interval Δt, each infection produces $s_{vi}\Delta t$ spores on the average. The proportion e of these spores will be deposited on leaves, but only the proportion ep_{vi} will succeed as new infections. Hence, the increase in number of infections can be described by the differential equation:

$$\mathrm{d}N_{vi}/dt = ep_{vi}s_{vi}N_{vi}(t). \quad (1)$$

The reproduction parameters enter only as $p_{vi}s_{vi}$ (cf, 18), which is the potential rate of disease increase of pathotype i on component

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v. The term $ep_{vi}s_{vi}$ is the *rate of disease increase* and is designated r_{vi}. The differential equation has the solution:

$$N_{vi}(t) = N_{vi}(0)\exp(r_{vi}t), \tag{2}$$

in which $N_{vi}(0)$ is the initial number of infections. $N_{vi}(0)$ is equal to $p_{vi}I_i$, in which I_i is the amount of primary inoculum of pathotype i. The disease progress curve (Eq. 2) is that of exponential growth, or of a "compound interest disease" with r_{vi} being the infection rate (16).

Next, the development of pathotype i on a mixture of n component cultivars is considered. In the model, the proportion of plants (and of leaf area) of the vth component, m_v,

$$(\sum_{v=1}^{n} m_v = 1)$$

is unchanged during the disease season. Spores that are not deposited on the donor *plant* are dispersed on the components according to the proportions $m_1, \ldots, m$. By arguments similar to those leading to Eq. 1,

$$p_{vi}aes_{vi}N_{vi}(t)\Delta t$$

gives the number of autoinfections from spores of pathotype i produced in the small time interval Δt on component v. The total number of spores that will be deposited on plants other than the donor plant is given by

$$\sum_{k=1}^{n} (1-a)es_{ki}N_{ki}(t)\Delta t$$

and the proportion of these spores that successfully infects component v during the time interval Δt is $p_{vi}m_v$. Hence, the change in number of infections of pathotype i is determined by the system of differential equations:

$$dN_{vi}(t)/dt = eap_{vi}s_{vi}N_{vi}(t) + e(1-a)p_{vi}m_v\sum_{k=1}^{n} s_{ki}N_{ki}(t) \tag{3}$$

in which $N_{vi}(0) = p_{vi}m_vI_i$ for $v = 1, \ldots, n$. The differential equations are solved by means of a standard method for first-order linear differential equation systems with constant coefficients (6). The solutions giving the number of infections of pathotype i on component v at time t are of the form

$$N_{vi}(t) = \sum_{k=1}^{n_i} Q_{kvi}(t)\exp(\lambda_{ki}t),$$

in which $Q_{kvi}(t)$ is a polynomial in t and $\lambda_{1i}, \ldots, \lambda_{n_i i}$ are the $n_i \leqslant n$ different eigenvalues in Eq. 3. The polynominal $Q_{kvi}(t)$ is just a constant except in case of repeated eigenvalues and it depends on the value of I_i.

The total number of infections of pathotype i equals

$$N_i(t) = \sum_{k=1}^{n_i}[\sum_{v=1}^{n} Q_{kvi}(t)]\exp(\lambda_{ki}t). \tag{4}$$

According to the Perron-Frobenius theory of irreducible nonnegative matrixes and their spectral properties (cf, 7), the eigenvalue of largest magnitude, say λ_{1i}, is positive and unique for all relevant parameter values, and the coefficient of exp $(\lambda_{1i}t)$ in Eq. 4,

$$\sum_{v=1}^{n} Q_{1vi}(t),$$

is a constant larger than zero. Hence, for large t the disease progress curve (Eq. 4) increases approximately exponentially at the rate λ_{1i}. Using the definition of eigenvectors and eigenvalues, λ_{1i} is found as the largest value among the solutions to the equation

$$(1-a)\sum_{v=1}^{n}[m_vr_{vi}/(\lambda - ar_{vi})] = 1 \tag{5}$$

if solutions exist; otherwise it equals $\max_v\{ar_{vi}\}$. This *long-term* rate of disease increase of pathotype i *on the mixture* (cf, Eq. 2) is designated R_i and called simply the *disease rate*. According to Eq. 5, R_i depends on the frequency of autodeposition (a), on the composition of the mixture ($m_1, \ldots, m_n$), and on the infection efficiencies ($p_{1i}, \ldots, p_{ni}$), the spore production rates ($s_{1i}, \ldots, s_{ni}$), and the proportion of spores deposited (e) through their products $r_{vi} = ep_{vi}s_{vi}$.

A case often considered is that of all spores being dispersed ($a = 0$). Then

$$R_i = \sum_{v=1}^{n} m_vr_{vi},$$

ie, the long-term rate of disease increase on the mixture is the weighted mean of the rates of disease increase on each component grown in monoculture. When a increases, the disease rate also increases (can only be shown rigorously for $n = 2$ and other special cases) and its maximum is attained for the extreme case ($a = 1$), in which all spores are deposited on the donor plant. Then $R_i = \max\{r_{1i}, \ldots, r_{ni}\}$.

For the development of a heterogeneous pathogen population it is assumed that the pathotypes increase independently. Then Eq. 4 describes the disease progress curve for each of the pathotypes in the population and the disease progress curve for the total pathogen population is

$$N(t) = \sum_{i=1}^{l}\sum_{k=1}^{n_i}[\sum_{v=1}^{n} Q_{kvi}(t)]\exp(\lambda_{ki}t). \tag{6}$$

For large t this disease progress curve increases approximately at the rate $R = \max\{R_1, \ldots, R_l\}$. For a given level of initial inoculum, this parameter indicates the level that the disease will reach in the cultivar mixture, assuming that the amount of healthy host tissue does not become limiting. Corresponding to Eq. 6, the disease progress curve found as the average over the disease progress curves for the same components grown as isolated monocultures over the same area equals

$$\sum_{v=1}^{n} m_v\sum_{i=1}^{l} N_{vi}(0)\exp(r_{vi}t) \tag{6a}$$

(cf, Eq. 2). For large t this disease progress curve increases approximately at the rate: $r = \max\{r_{11}, \ldots, r_{nl}\}$. Thus, $C = r - R$ is a reasonable measure of the long-term difference in disease level between the average performance of an isolated monoculture and the corresponding mixture, ie, the *efficiency of disease control* using the latter approach. However, when different mixtures of the same components are compared, it is more convenient to consider deviations of the *relative* disease rate $\mathfrak{R} = R/r$ from 1. As long as $C > 0$, the disease level attained from a given level of initial inoculum is smaller in the mixture than the average on the components in isolated monocultures.

The composition of the pathogen population can be calculated from Eq. 4 and Eq. 6; the *proportion of infections* of pathotype i at time t is $f_i(t) = N_i(t)/N(t)$. If the epidemic proceeds long enough, the composition of the pathogen population will stabilize. The final proportion of pathotype i, f_i, is determined by the disease rates $R_1, \ldots, R_l$ and the pathotype with the highest disease rate will in relative terms eventually displace all other pathotypes. If the two largest disease rates are equal, say $R_1 = R_2$, then pathotypes 1 and 2 will be found in the ratio

$$f_1/f_2 = \sum_{v=1}^{n} Q_{1v1}(t)/\sum_{v=1}^{n} Q_{1v2}(t) \tag{7}$$

and, if the highest disease rates are nearly equal, the corresponding pathotypes might all persist in detectable proportions for a considerable period.

EXAMPLES OF TWO-COMPONENT MIXTURES

In a two-component mixture ($n = 2$) the expression for the total number of infections of pathotype i (Eq. 4) can easily be found in terms of the parameters r_{1i}, r_{2i}, p_{1i}, p_{2i}, m_1, m_2, and a, by using

$$\lambda_{1i} = [(K_1 r_{1i} + K_2 r_{2i}) + \sqrt{(K_1 r_{1i} - K_2 r_{2i})^2 + 4(1-a)^2 r_{1i} r_{2i} m_1 m_2}]/2$$

$$\lambda_{2i} = [(K_1 r_{1i} + K_2 r_{2i}) - \sqrt{(K_1 r_{1i} - K_2 r_{2i})^2 + 4(1-a)^2 r_{1i} r_{2i} m_1 m_2}]/2 \quad (8)$$

$$Q_{1vi} = I_i p_{vi} m_v (\lambda_{1i} - a r_{ui})/(\lambda_{1i} - \lambda_{2i})$$

$$Q_{2vi} = I_i p_{vi} m_v (a r_{ui} - \lambda_{2i})/(\lambda_{1i} - \lambda_{2i})$$

in which $u = 2$ if $v = 1$ and vice versa, and $K_v = a + (1-a)m_v$.

Three theoretical examples of the development of an epidemic of three pathotypes ($l = 3$) will be considered. The infection efficiencies are given in Table 1 and the spore production rates in Table 2. The values have been chosen to illustrate the behavior of the model in the extreme cases where either all deposited spores of a pathotype or none of them succeed in infecting a cultivar; with 10% of spores being deposited ($e = 0.10$) the largest spore production rate ($r_{21} = 3.0$) corresponds to a 20-fold increase in number of infections in 10 days (cf, Eq. 2).

The effects of differences in reproduction parameters on epidemic development in a 1:1 mixture are illustrated in Tables 3 and 4. In the example in Table 3, pathotype 1, which reproduced only on component 2, increased in proportion; in the example in Table 4, pathotypes 1 and 3 initially increased, but pathotype 1 soon began to decrease. The effect of a difference in mixture composition on epidemic development with equal spore production rates is demonstrated in Tables 4 and 5. In the example in Table 5 the mixture composition was chosen such that the pathotypes 1 and 3 increased simultaneously.

TABLE 1. Interactions (in terms of infection efficiencies[a]) between two plant host components and three pathotypes in a theoretical example

	Pathotype		
Component	1	2	3
1	0	1	1
2	1	0	1

[a] Infection efficiency is the proportion of spores deposited that establish a sporulating colony.

TABLE 2. Three examples of spore production rates[a] for three pathotypes on two plant host components in a theoretical example

		Pathotype		
Example	Host component	1	2	3
A	1	0.0	1.8	1.5
	2	3.0	0.0	1.2
B	1	0.0	1.8	1.5
	2	3.0	0.0	2.4
C	1	0.0	2.7	1.5
	2	3.0	0.0	2.4

[a] Spore production rates are in terms of spores produced per day per infection.

The long-term predictions for the development in the three examples can be read from the magnitude of the disease rates (Table 6). The long-term predominant pathotype is pathotype 1 in the example in Table 3, pathotype 3 in Table 4, and pathotypes 1 and 3 in Table 5. This corresponds reasonably well with the results obtained in 100 days. The predicted order of disease levels (according to R) and disease control (according to C) are also found within 100 days. The initial increase of pathotype 1 in Table 4 is due to a high initial proportion of the poorest pathotype, pathotype 2, compared with that of pathotype 3.

An overall picture of the long-term results for all combinations of the variable parameters m_1 (the proportion of component 1) and a (the frequency of autodeposition) is given in Fig. 1 for three different combinations of spore production rates (Table 2). From Fig. 1A–C it is apparent that, in general, the pathotype that reproduces best on the more common component will be dominant in very uneven mixtures and that the pathotype that reproduces on both cultivars (if it predominates at all) will do so for small values of the frequency of autodeposition and for intermediary mixtures. However, if the latter pathotype reproduces as well as the other pathotypes on each component it will predominate for all combinations of m_1 and a. When both pathotypes 1 and 2 persist, the final ratio between them is (cf, Eq. 7)

$$f_1/f_2 = I_1 m_2 / I_2 m_1 = N_1(0)/N_2(0),$$

so, during the epidemic pathotype 3 decreases without changing the ratio between pathotypes 1 and 2. Conversely, when pathotypes 1 and 3 (or 2 and 3) predominate, the final ratio equals

$$f_1/f_3 = N_1(0)/N_3(0) \times (\lambda_{13} - \lambda_{23})/(\lambda_{13} - a(r_{13} m_2 + r_{23} m_1))$$

and this will only seldom (eg, if $s_{13} = s_{23}$) be identical with the initial ratio.

For varying values of m_1 and a the disease control provided by the mixture can be evaluated from the relative disease rate, $\mathcal{R}$. A small value of $\mathcal{R}$ corresponds to a large efficiency of disease control. Contour lines of $\mathcal{R}$ are shown in Fig. 1A′–C′. Again, the basic predictions apply over a range of values of r_{vi}. If the pathotype reproducing on both cultivars has a fitness disadvantage, the efficiency of the mixture in controlling disease is highest when neither component is rare and the frequency of autodeposition is low. In addition, a mixture with a smaller long-term rate of disease increase than that of the best monoculture (component 1) can

TABLE 3. Change in composition and increase in number of infections during 100 days for the theoretical host-pathogen system defined in Tables 1 and 2, example A[a]

	Frequency of pathotype			Number of infections	
Day	1 $f_1(t)$	2 $f_2(t)$	3 $f_3(t)$	On mixture[b] N(t)	On pure stands[c] N(t)
0	0.42	0.42	0.17	6×10^1	6×10^1
10	0.55	0.29	0.16	2×10^2	7×10^2
20	0.67	0.18	0.15	1×10^3	1×10^4
30	0.77	0.11	0.13	5×10^3	2×10^5
40	0.84	0.06	0.10	2×10^4	4×10^6
50	0.89	0.03	0.08	1×10^5	8×10^7
60	0.92	0.02	0.06	5×10^5	2×10^9
70	0.94	0.01	0.05	3×10^6	3×10^{10}
80	0.96	0.01	0.04	1×10^7	7×10^{11}
90	0.97	<0.005	0.03	7×10^7	1×10^{13}
100	0.98	<0.005	0.02	4×10^8	3×10^{14}

[a] Proportion of spores deposited (e) = 0.10; frequency of autodeposition (a) = 0.01; frequency of component 1 (m_1) = frequency of component 2 (m_2) = 0.50; initial number of spores of pathotype 1 (I_1) = initial number of spores of pathotype 2 (I_2) = 50; and initial number of spores of pathotype 3 (I_3) = 10.
[b] According to Eq. 6.
[c] According to Eq. 6a.

TABLE 4. Change in composition and increase in number of infections during 100 days for the theoretical host-pathogen system defined in Tables 1 and 2, example B. The parameter values are as in Table 3 except for the spore production rates[a]

Day	Frequency of pathotype 1 $f_1(t)$	2 $f_2(t)$	3 $f_3(t)$	Number of infections: On mixture[b] N(t)	On pure stands[c] N(t)
0	0.42	0.42	0.17	6×10^1	6×10^1
10	0.49	0.25	0.26	3×10^2	7×10^2
20	0.50	0.13	0.37	1×10^3	1×10^4
30	0.47	0.06	0.47	8×10^3	2×10^5
40	0.41	0.03	0.56	5×10^4	4×10^6
50	0.34	0.01	0.65	3×10^5	8×10^7
60	0.28	0.01	0.72	2×10^6	2×10^9
70	0.22	<0.005	0.78	1×10^7	3×10^{10}
80	0.17	<0.005	0.83	5×10^7	7×10^{11}
90	0.13	<0.005	0.87	5×10^8	1×10^{13}
100	0.10	<0.005	0.90	4×10^9	3×10^{14}

[a] Proportion of spores deposited (e) = 0.10; frequency of autodeposition (a) = 0.01; frequency of component 1 (m_1) = frequency of component 2 (m_2) = 0.50; initial number of spores of pathotype 1 (I_1) = initial number of spores of pathotype 2 (I_2) = 50; and initial number of spores of pathotype 3 (I_3) = 10.
[b] According to Eq. 6.
[c] According to Eq. 6a.

TABLE 5. Change in composition and increase in number of infections during 100 days for the theoretical host-pathogen system defined in Tables 1 and 2, example B. The parameter values are as in Table 4 except for the composition of the mixture[a]

Day	Frequency of pathotype 1 $f_1(t)$	2 $f_2(t)$	3 $f_3(t)$	Number of infections: On mixture[b] N(t)	On pure stands[c] N(t)
0	0.56	0.27	0.17	6×10^1	6×10^1
10	0.71	0.09	0.21	4×10^2	1×10^3
20	0.75	0.02	0.22	3×10^3	2×10^4
30	0.77	0.01	0.23	3×10^4	3×10^5
40	0.77	<0.005	0.23	2×10^5	6×10^6
50	0.77	<0.005	0.23	2×10^6	1×10^8
60	0.77	<0.005	0.23	1×10^7	2×10^9
70	0.77	<0.005	0.23	1×10^8	5×10^{10}
80	0.77	<0.005	0.23	1×10^9	9×10^{11}
90	0.77	<0.005	0.23	8×10^9	2×10^{13}
100	0.77	<0.005	0.23	7×10^{10}	4×10^{14}

[a] Proportion of spores deposited (e) = 0.10; frequency of autodeposition (a) = 0.10; frequency of component 1 (m_1) = 0.328; frequency of component 2 (m_2) = 0.672; initial number spores of pathotype 1 (I_1) = initial number of spores of pathotype 2 (I_2) = 50; and initial number of spores of pathotype 3 (I_3) = 10.
[b] According to Eq. 6.
[c] According to Eq. 6a.

always be constructed. (In the B′ case, this cannot be read from the figure.) However, for these parameter values, the pathotype able to reproduce on both components dominates (Fig. 1A–C), so the resistance of the crop is apparently eroded.

PREDICTIONS FOR DIFFERENT MIXING STRATEGIES

The comprehensive description of the general predictions of the epidemic, ie, the composition of the pathogen population and the disease control, is based on the long-term disease rates, $R_1, \ldots, R_l$. In case of more than two components in the mixture the expression for R_i is too complicated to be conclusive, so the following simplifications of the model assumptions are made: each of the n components possesses one dominant resistance gene that conditions complete resistance; the corresponding 2^n pathotypes are present in the primary inoculum; pathotypes reproducing on only one component, *simple types*, reproduce better on this component than pathotypes reproducing on at least two components, *complex types*; and all pathotypes reproducing on k ($k \geq 1$) components have a rate of disease increase on these components that is

$$r - (k-1)w \qquad (0 \leq w \leq r/(n-1)),$$

saying that on each of its compatible hosts grown in monoculture, the disease progress curve for these pathotypes is equal to:

$$N_{vi}(t) = N_{vi}(0) \left\{ \exp(r) \, [\exp(-w)]^{k-1} \right\}^t. \qquad (9)$$

This means that the interaction among virulence loci is multiplicative, with w being the decrease in rate of disease increase due to the substitution of one virulence gene for one avirulence gene. Furthermore, r equals the common rate of disease increase of simple types. With these assumptions the long-term rates of disease increase (ie, the eigenvalues corresponding to Eq. 3) can be calculated since separate knowledge of the infection efficiencies and spore production rates are unnecessary (cf, Eq. 5). Since all pathotypes reproducing on k components (ie, being of complexity k), have the same rate of disease increase on compatible cultivars, the largest long-term rate of disease increase among these pathotypes is found for the pathotype(s) reproducing on the k most common components. The disease rate of these pathotype(s) is designated $R(k)$, and from Eq. 5

TABLE 6. Long-term rates of disease increase on the mixture (R_i) for each pathotype in the theoretical systems defined in Tables 3, 4, and 5, and efficiency of disease control (C)[a]

System	R_1	R_2	R_3	C
Table 3	0.165	0.099	0.135	0.135
Table 4	0.165	0.099	0.196	0.104
Table 5	0.211	0.071	0.211	0.089

[a] $C = r - \max(R_1, R_2, R_3)$ in which r equals the long-term average disease rate on the pure stands ($r = 0.30$).

$$R(k) = [a + (1 - a) \sum_{v=1}^{k} m_{(v)}] [r - (k - 1)w], \quad (10)$$

where $m_{(v)}$ is the vth value when $m_1, \ldots, m_n$ are listed in descending order. When the complexity k varies from 1 to n, it can be shown (by looking at $\Delta R(k) = R(k+1) - R(k)$ and $\Delta(\Delta R(k))$ that $R(k)$ increases from k equals 1 to some number k_o and decreases from k equals k_o to n. Thus, k_o is the complexity for which $R = R(k)$, ie, pathotypes compatible with k_o components will predominate. The definition of k_o is equivalent to

$$R(k_o+1) \leqslant R(k_o) \text{ and } R(k_o-1) \leqslant R(k_o)$$

or if $w > 0$,

$$m_{(k_o+1)} (r/w - k_o) \leqslant a/(1-a) + \sum_{v=1}^{k_o} m_{(v)} \leqslant m_{(k_o)}(r/w - k_o + 2) \quad (11)$$

in which the first inequality is cancelled for $k_o = n$ and the second for $k_o = 1$; if $w = 0$ the most complex type will always predominate. The value of k_o depends on the rate of disease increase of simple types compared to the disadvantage of an unnecessary virulence gene, r/w, the frequency of autodeposition, a, and the composition of the mixture $m_1, \ldots, m_n$. This result will now be applied to evaluate different mixing strategies.

At first, the composition of the mixture and the frequency of autodeposition (related to the density of the crop, cf, 3) are changed simultaneously (cf, Fig. 1). A mixture can always be chosen such that its disease level is smaller than that on each monoculture since $R(k) < r$ for all k except when $a = 1$ or $m_{(1)} = 1$ (see Eq. 10). Furthermore, by rearranging the first term in Eq. 10 it is seen that the lowest disease level will be found when $a = 0$ and the mixture is even. The most complex pathotype is most likely to predominate, ie, $k_o = n$, when the proportion of the rarest component in the mixture, $m_{(n)}$, is large, and most of the spores are dispersed, ie, a is small. This is concluded from the condition for $k_o = n$ which according to Eq. 11 is

$$w/r \leqslant m_{(n)} (1-a)/[1+(n-2)m_{(n)} (1-a)]$$

and this inequality is most likely to be satisfied if the right-hand side is large. Similarly, it can be shown that simple pathotypes are likely to predominate if the mixture consists of mostly one component or if most of the effective spores are deposited on their donor plant. In conclusion, when w is positive a strong selection operates favoring complex types when neither component is rare and a considerable exchange of spores occurs, whereas the simple types are favored on mixtures that resemble monocultures. The selection for complex types leads to an erosion of the resistance of the crop, but due to the fitness disadvantage of complexity, the disease control is still effective ($C > 0$).

Another way of changing the mixture is to alter the number of components that constitute it. As an example, the components are evenly mixed. Values of the relative disease rates (R) are obtained from Eq. 10 with $m_{(v)} = 1/n$, and shown in Fig. 2 for four combinations of the relative disadvantage of an unnecessary virulence gene (w/r) and the frequency of autodeposition (a). The long-term prediction is that the effect on the disease control of using an additional component begins to decrease when the number of components has reached a certain magnitude. In addition, the most complex pathotype predominates only in mixtures consisting of few components (depending on the parameter values). The reason for this is that when the mixture

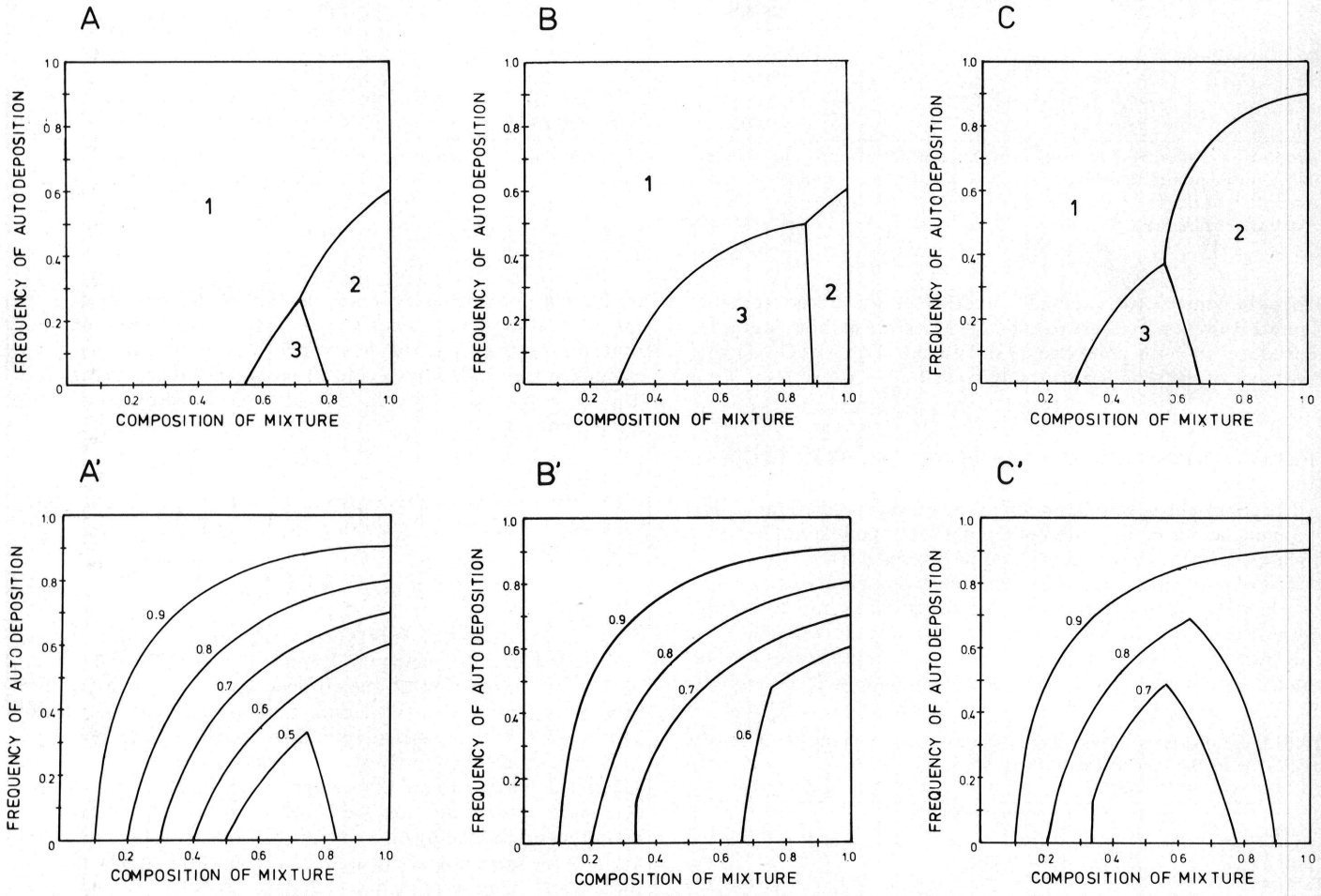

Fig. 1. Predominant pathotype and long-term rate of disease increase in three theoretical examples, A, B, and C in Table 2. **A, B, and C:** The combinations of proportion of component 1 (m_1) in a component mixture, and frequency of autodeposition (a) are divided into three regions in which each of the three pathotypes predominates. **A′, B′, and C′:** Contour lines of the relative long-term rate of disease increase (R) are shown as a function of m_1, and a.

consists of many components the proportion of each will be so small that the pathotype able to reproduce on all components no longer has an advantage. The complexity of the dominant pathotype might be unchanged for several types of mixtures (Fig. 2), and if no autodeposition occurs ($a = 0$), it is fully independent of the number of components in the mixture (cf, Eq. 11).

Finally, the results obtained for mixtures of plants of different cultivars can be extended to describe the epidemic development on mixtures of fields of different cultivars, ie, on a "patchwork" of monocultures. In that case, a is interpreted as the proportion of deposited spores being deposited in the field in which they are produced. It is likely that the value of a will be larger and with that the selection pressure in favor of complex pathotypes will be smaller than if the area were planted with the same components in a cultivar mixture.

DISCUSSION

The model aims at a theoretical description of the basic patterns of the epidemic development, and it thus lacks many biological details (cf, 18). In actual systems, the environment of the pathogen (the crop) alters during the growing season, causing changes in the epidemiological parameters. For instance, the proportion of effective spores and the frequency of autodeposition may vary due to changes in leaf area, and weather conditions; adult plant resistance may cause changes in infection efficiencies and spore production rates. Furthermore, the delay of the epidemic caused by the latent period and the fact that infections do not sporulate throughout the growing season has been neglected.

The model is founded on the assumption that the rate of increase in the number of infections of each pathotype is density-independent, ie, there is no limit in leaf area (Eq. 1). Furthermore, it is assumed that the competition for substrate between pathotypes, as well as the opposing influence of induced resistance and induced susceptibility (for a review, see 3), is negligible, ie, the rate of increase in number of infections is frequency-independent (cf, Eq. 6). These are realistic assumptions if the leaf area of each component during the whole disease season is large compared with the number of infections on it. With this restriction, the assumption of a constant mixture composition is also reasonable as no component increases its leaf area to compensate for the extermination of a highly diseased component.

A much more restrictive assumption is that of no recombination (no sexual reproduction). The influence of this factor on the composition of the pathogen population is difficult to predict. However, appropriate model situations do exist as in some host-pathogen systems (eg, cereal powdery mildew) virtually no sexual reproduction occurs during the disease season. Another fundamental assumption concerns mutation. If the mutation rate from avirulence to virulence is of the same order of magnitude as the fitness disadvantage of an additional virulence ability, a very complex type might predominate even if it was not expected to do so. An opposite effect might appear if the mutation rate is so low that not all possible pathotypes are present in the primary inoculum.

A prediction of the composition of the pathogen population for several years cannot be calculated directly from the model. At first, each time the pathogen infects a new crop the frequency of autodeposition is zero, so the complex types will be especially favored. This changes the starting composition on the new crop but might not greatly influence the general evolutionary pattern. Next, sexual reproduction at the end of each season might influence the development over several years; a preliminary analysis indicates, however, that the eventual outcome is unchanged in simple gene-for-gene systems with few components in the mixture. Finally, it is unlikely that the reproduction parameters are constant during seasons since the pathotypes may become adapted to the mixture by means of mutations in modifier genes (17).

In the examples analyzed, pathotypes able to reproduce on more than one component have some fitness disadvantage. Without this assumption, the long-term prediction is easily given: only the most complex pathotype will increase in frequency. The assumption seems reasonable, however, because it may be hypothesized that the interface pathogen/host for a given genotype of the host can work at an optimal level for only one pathogen genotype and that this pathogen genotype differs among host genotypes. Under the assumption of fitness disadvantage of complexity, the conclusions of the model analysis are:

(i) If the epidemic proceeds long enough, the composition of the pathogen population will stabilize. In most cases, however, it is unlikely that a balanced polymorphism exists.

(ii) Diversity between fields preserves pathotypes of little complexity but might show only a slight efficiency of disease control. Thus, this strategy might give results contrary to the use of diversity within fields.

(iii) The ability of the predominant pathotypes to reproduce on many components of the mixture is a bad predictor for the disease control of the mixture if the fitness pattern and the adaption ability (mutation rate) of the pathotypes are unknown.

(iv) Growing mixtures of many cultivars might stop the "boom and bust cycle" found when using monocultures of single major resistance genes.

(v) The frequency of autodeposition should be studied further, because changes in this parameter influence the disease control within the mixture substantially.

The present model is a generalization of the continuous-time models of Fleming and Person (4) and Jeger et al (9). It includes autodeposition and divides the "rate of increase of inoculum" (4) into three parameters: the proportion of deposited spores, the infection efficiency, and the spore production rate. Furthermore, compared to Fleming and Person's model it goes into much greater detail in terms of the change in the pathogen population with respect to number of virulence genes, and it includes the aspects of nonspecialized pathogens analyzed by Jeger et al (9). The simple discrete-time models with nonoverlapping generations by Groth (8) and Marshall and Pryor (13) are comparable with the simplification of this model (Eq. 9) when measuring time in generations. The term "s" used by Groth (8), and Marshall and Pryor (13) in their multiplicative model corresponds then to $1 - \exp$

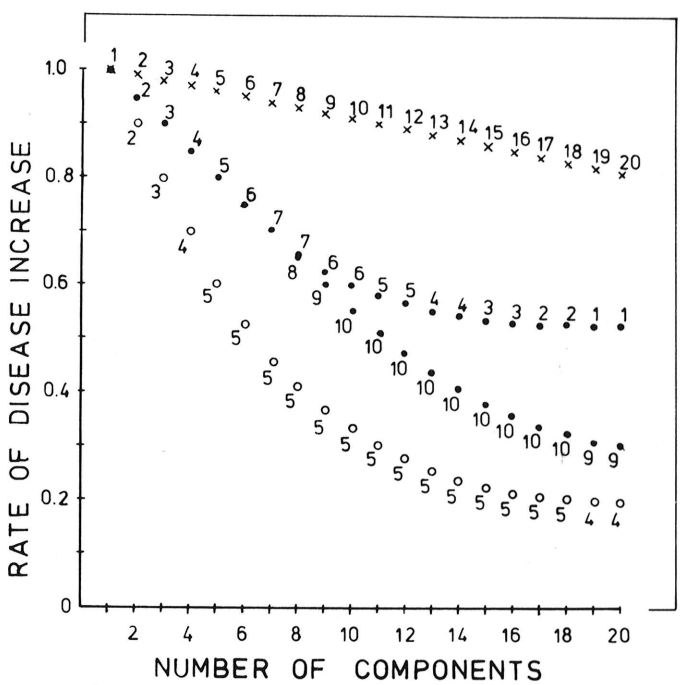

Fig. 2. Influence of the number of components (n) in an even mixture of cultivars on the relative long-term rate of disease increase ($\hat{R}$). The relative disadvantage of an unnecessary virulence gene is $w/r = 0.01$ (×), $w/r = 0.05$ (●), and $w/r = 0.1$ (○), and the frequency of autodeposition is $a = 0.1$ except for the upper curve of the full dotted curves (●) where $a = 0.5$. For each point, the complexity (number of virulence genes) of the predominant pathotype is shown.

$(-w)$, and the general long-term predictions of these three models are similar when assuming no autodeposition.

The discrete-time model with nonoverlapping generations by Kiyosawa and Yabuki (11) describes the changes in proportion of pathogen genotypes from generation to generation. The model assumptions correspond to those of the present model with $a = 0$ and $r_{vi} = \alpha_v + \beta_i$; ie, the rate of disease increase is the sum of a "field susceptibility of the cultivar" and a "multiplication ability of the pathogen genotype" (11). In their model, Kiyosawa and Yabuki give conditions for an "equilibrium of race frequencies." These conditions are not, however, necessarily related to the question of the final composition of the pathogen population or to the largest eigenvalues of the present model.

A somewhat different discrete-time model with overlapping generations was suggested by Barrett (1,2). He defined a measure of autodeposition (ϕ) that depends on a general spore production factor (α), so no autodeposition in the present model corresponds to $\phi = 1$ *and* $\alpha \rightarrow \infty$ (2). This conflict is partly due to the fact that his host-specific parameters are viabilities of infections and as such confound the infection efficiency and the death factor of established infections. Nevertheless, the long-term results shown by Barrett (1) are similar to those found in the present example of a two component mixture.

In conclusion, the present model is much related to most previous models on mixtures, but being more general it enables analysis of the generality of previous predictions. Furthermore, contrary to most previous models, it includes a frequency of autodeposition. It has been stated that this parameter might be redundant (12), but when the term is redefined to be deposition within the donor field as suggested here, it might gain new significance.

LITERATURE CITED

1. Barrett, J. A. 1978. A model of epidemic development in variety mixtures. Pages 129–137 in: Plant Disease Epidemiology. P. R. Scott and A. Bainbridge, eds. Blackwell Scientific Publications Ltd., Oxford, England. 329 pp.
2. Barrett, J. A. 1980. Pathogen evolution in multilines and variety mixtures. Z. Pflanzenkrankh. 87:383-396.
3. Chin, K. M. 1979. Aspects of the epidemiology and genetics of the foliar pathogen, *Erysiphe graminis* f. sp. *hordei*, in relation to infection of homogeneous and heterogeneous populations of the barley host, *Hordeum vulgare*. Ph.D. thesis, University of Cambridge, England. 137 pp.
4. Fleming, R. A., and Person, C. O. 1978. Disease control through use of multilines: A theoretical contribution. Phytopathology 68:1230-1233.
5. Frey, K. J., Browning, J. A., and Simons, M. D. 1977. Management systems for host genes to control disease loss. Ann. N.Y. Acad. Sci. 287: 255-274.
6. Gantmacher, F. R. 1959. The Theory of Matrices. Vol. I. Chelsea Publishing Company, New York. 374 pp.
7. Gantmacher, F. R. 1959. The Theory of Matrices. Vol. II. Chelsea Publishing Company, New York. 276 pp.
8. Groth, J. V. 1976. Multilines and "super races": A simple model. Phytopathology 66:937-939.
9. Jeger, M. J., Griffiths, E., and Jones, D. G. 1981. Disease progress of non-specialised fungal pathogens in intraspecific mixed stands of cereal cultivars. I. Models. Ann. Appl. Biol. 98:187-198.
10. Kampmeijer, P., and Zadoks, J. C. 1977. EPIMUL, a simulator of foci and epidemics in mixtures of resistant and susceptible plants, mosaics and multilines. PUDOC, Wageningen, The Netherlands. 50 pp.
11. Kiyosawa, S., and Yabuki, S. 1976. Modeling on the race frequency change in a host-pathogen system with genes for resistance and avirulence. Jpn. J. Breed. 26:237-246.
12. Leonard, K. J., and Czochor, R. J. 1980. Theory of genetic interactions among populations of plants and their pathogens. Annu. Rev. Phytopathol. 18:237-258.
13. Marshall, D. R., and Pryor, A. J. 1978. Multiline varieties and disease control. I. The "dirty crop" approach with each component carrying a unique single resistance gene. Theor. Appl. Genet. 51:177-184.
14. Robinson, R. A. 1976. Plant Pathosystems. Springer-Verlag, Berlin, Heidelberg, and New York. 184 pp.
15. Trenbath, B. R. 1977. Interactions among diverse hosts and diverse parasites. Ann. N.Y. Acad. Sci. 287:124-150.
16. Vanderplank, J. E. 1968. Disease Resistance in Plants. Academic Press, New York, and London. 206 pp.
17. Wolfe, M. S., and Barrett, J. A. 1980. Can we lead the pathogen astray? Plant Dis. 64:148-155.
18. Zadoks, J. C., and Schein, R. D. 1979. Epidemiology and Plant Disease Management. Oxford University Press, New York and Oxford. 427 pp.