Measuring Disease Progress in Pure and Mixed Stands of Plant Cultivars

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ABSTRACT

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A measure is presented that quantifies disease progress of fungal pathotypes during the exponential phase of the epidemic. This measure, referred to as rate of disease increase (ρ), represents the factor by which the number of infection units is multiplied from one day to another after the initial infection waves have damped. The rate of disease increase depends on four pathotype/cultivar-specific parameters (latent period, infectious period, infection efficiency, spore production rate) and two nonspecific parameters (deposition frequency and autodeposition frequency). Three applications are given. First, it is used to determine the levels of partial resistance of barley cultivars to leaf rust. The calculated ρ values were highly correlated both with total spore production per unit leaf area ($r = 0.97$) and with disease score in the field ($r = 0.81$). The rate of disease increase is particularly useful if the epidemiological parameters display compensatory effects. The second application deals with the long-term composition of pathotype mixtures. The pathotype with the largest ρ value will predominate in the long run. This is again exemplified by using barley leaf rust data. The third application concerns disease control strategies. A host stand should be composed so that the corresponding predominant pathotype has a ρ value smaller than the prevailing pathotype of any other composition. This concept does not presuppose selection against unnecessary genes for virulence. A condition is given under which a cultivar mixture may be more beneficial than each of its components grown in pure stands, and this condition is illustrated by a simple example of two pathotypes and two cultivars.

Additional keywords: autoinfection, epidemiology, optimal cultivar composition.

Plant pathologists mostly use Van der Plank's (29) apparent infection rate (r) to quantify disease progress. This measure has proven useful for comparative studies on sanitation efforts, fungicide application, and cultivar resistance (3,8–10,15). It was shown by Van der Plank (29) that r exhibits wavelike behavior during epidemic development even though latent period (p), infectious period (i), and basic infection rate (R) are assumed to be constant. The waves damp as disease progresses exponentially, and r reaches a constant value that can be calculated from p, i, and R (see reference 30, equation 4.3). A somewhat different measure of disease progress has been suggested by Leonard and Mundt (16). It is a function of latent period, time of peak spore production, time at which sporulation ceases, and total reproduction per generation. Both measures refer to continuous infection processes. For discontinuous processes, Corsten (6) and Oort (21) proposed a measure analogous to Van der Plank's r_l, and Ogle et al (20) introduced a much simpler "measure of relative survival ability." These measures are constructed for epidemics in pure stands. For measuring disease progress in cultivar mixtures, Østergaard (18) suggested the "long-term rate of disease increase." She takes into account infection efficiency, spore production rate, and two deposition parameters, but she considers neither latent nor infectious period. Her measure is therefore inappropriate to quantify disease progress of pathotypes that differ in latent period. This disadvantage is overcome by the measure presented here. It incorporates all basic epidemiological parameters and quantifies disease progress in monocultures and cultivar mixtures during the exponential phase of the epidemic.

Epidemics in monocultures. The measure to be derived is closely related to a time-discrete model previously published by Gumpert et al (11). It describes the increase of the number of infection units (as defined in reference 32) during one disease season. An "infection unit" (called "infection" in reference 11) is the mycelial structure that originates from a single spore. It goes through the following phases of development: pre-sporulation (beginning with spore germination), sporulation, and post-sporulation. Every infection unit, independently of its phase of development, is counted here. Infection units that have passed the latent period are called "lesions." Our model, referring to a single pathotype, has the following form:

$$Y(t) - Y(t-1) = eds[Y(t-p) - Y(t-p-i)], \qquad (1)$$

where $Y(t)$ = number of infection units at day t; p = latent period (days from inoculation to sporulation); i = infectious period (number of days on which a lesion sporulates); e = infection efficiency (the proportion of deposited spores that form sporulating lesions); s = spore production rate (number of spores produced per lesion and day); and d = deposition frequency (proportion of produced spores that are deposited on potential infection sites).

Equation 1 says that the number of sporulating lesions at day t (brackets) multiplied by eds (= Zadok's [31] "daily multiplication factor") gives the number of newly occurring infection units at day t.

According to the theory of linear difference equations (see reference 22, for example), equation 1 has a unique solution if, for $p + i$ consecutive days, the initial values are given. For example,

$$Y(t) = \begin{cases} 0, & -(p+i) < t < 0 \\ eI, & t = 0, \end{cases} \qquad (2)$$

where I is the amount of primary inoculum already deposited on leaves. We remark that only initial values at times $-(p + i) < t \leq 0$ contribute to $Y(t)$ for $t > 0$. Therefore the reader may without loss of generality assume that $Y(t) = 0$ for all $t < 0$. The solution of equation 1 can be determined either numerically by a simple iteration process performed on the computer or algebraically by means of the eigenvalues corresponding to equation 1. These eigenvalues are the roots of the characteristic polynomial (see reference 22, for example) of equation 1:

$$P(\lambda) = \lambda^{p+i} - \lambda^{p+i-1} - eds(\lambda^i - 1) \qquad (3)$$

If $\lambda_1,\ldots,\lambda_l$ are the l ($l \leq p + i$) distinct roots of P and $k_1,\ldots,k_l$, the corresponding multiplicities (repeated roots), then the solution $Y(t)$ is given by:

$$Y(t) = \sum_{\nu=1}^{l} \left(\sum_{\mu=1}^{k_\nu} c_{\nu\mu}\, t^{k_\nu - \mu} \right) \lambda_\nu^t \quad , \quad t > -(p+i). \tag{4}$$

The coefficients $c_{\nu\mu}$ are determined by solving the following system of $p + i$ linear equations which result from combining equations 2 and 4:

$$\sum_{\nu=1}^{l} \left(\sum_{\mu=1}^{k_\nu} c_{\nu\mu}\, t^{k_\nu - \mu} \right) \lambda_\nu^t = 0 \quad , \quad -(p+i) < t < 0$$

$$\sum_{\nu=1}^{l} c_{\nu k_\nu} = eI\,.$$

The asymptotic behavior of the ratio $Y(t)/Y(t-1)$ is studied next. Given arbitrary values of the parameters p, i, e, s, d, and I, the characteristic polynomial P (equation 3) and the solution $Y(t)$ (equation 4) are uniquely determined. Because $\lambda = 1$ is a root of P, the polynomial is of the following form:

$$P(\lambda) = (\lambda - 1)Q(\lambda)\,,$$

where $$Q(\lambda) = \lambda^{p+i-1} - eds(\lambda^{i-1} + \lambda^{i-2} + \ldots + \lambda + 1)\,. \tag{5}$$

The polynomial Q has exactly one positive root λ^*. By a theorem of Cauchy (compare with reference 22, pages 96–97), λ^* is simple, that is, it has multiplicity 1. Moreover, if $i > 1$, λ^* is unique dominant; that is, the moduli of all other roots of Q are $< \lambda^*$ (compare with reference 22, Theorem 12.2). In the following, it is assumed that $i > 1$. Because any root of polynomial Q is also a root of polynomial P, the latter has the unique dominant root $max(1, \lambda^*)$. This root is represented by one of the eigenvalues $\lambda_1,\ldots,\lambda_l$ in equation 4. Without loss of generality, it can be supposed that

$$\lambda_1 = max(1, \lambda^*),$$

$$\text{i.e.,} \quad \lambda_1 > |\lambda_2|, \ldots, |\lambda_l|. \tag{6}$$

Furthermore, two cases concerning the quantity $edsi$ have to be considered.

Case 1: $edsi \neq 1$.
Then $\lambda^* \neq 1$ and λ_1 has multiplicity 1; that is, $k_1 = 1$. By equation 4, we obtain

$$Y(t) = c_{11}\lambda_1^t + \sum_{\nu=2}^{l} \left(\sum_{\mu=1}^{k_\nu} c_{\nu\mu}\, t^{k_\nu - \mu} \right) \lambda_\nu^t. \tag{7}$$

Case 2: $edsi = 1$.
Then $\lambda^* = 1$ and λ_1 ($= 1$) has multiplicity 2; that is, $k_1 = 2$. By equation 4, we obtain

$$Y(t) = (c_{11}t + c_{12})\lambda_1^t + \sum_{\nu=2}^{l} \left(\sum_{\mu=1}^{k_\nu} c_{\nu\mu}\, t^{k_\nu - \mu} \right) \lambda_\nu^t. \tag{8}$$

By means of complex variable theory, it can be shown that the initial condition (equation 2) gives

$$c_{11} \neq 0 \tag{9}$$

in case 1 and

$$c_{11} \neq 0 \quad \text{or} \quad c_{12} \neq 0 \tag{10}$$

in case 2. In view of the inequalities 6, 9, and 10, the conditions of Bernoulli's method (see reference 12, for example) are satisfied; that is, the solution $Y(t)$ has the property

$$\lim_{t \to \infty} Y(t)/Y(t-1) = \lambda_1, \tag{11}$$

where λ_1 is the largest positive root of the characteristic polynomial P. Consequently, it has been proved that for any pathotype, characterized by its parameters p, i ($i > 1$), e, s, and d, and any amount of primary inoculum I, the ratio $Y(t)/Y(t-1)$ has a limit; it is called "rate of disease increase" and is denoted by $\rho (= \lambda_1)$. According to this definition, ρ is the factor by which the number of infection units is multiplied from one day to another after the initial waves have damped. Figure 1 depicts the disease progress curve $Y(t)$ and corresponding ratio $Y(t)/Y(t-1)$ of a hypothetical epidemic. The ratio shows wide fluctuations at the beginning of the epidemic; the amplitude reaches its highest value when the primary inoculum has passed the latent period. As the epidemic turns from the "simple interest" phase ($p \leq t < 2p$) to the "compound interest" phase ($t \geq 2p$), the amplitudes decrease substantially and the curve rapidly approaches ρ. These observations are similar to those of Van der Plank (29,30) with continuous infection processes.

In fast epidemics, lesions that have ceased sporulating (that is, removals) are relatively unimportant, and i can be neglected without incurring a significant error. Equation 4 then can be written in the form of a factorial series (11, equation 4):

$$Y(t) = Y(0) \sum_{\nu=0}^{K} \frac{(eds)^\nu (t - \nu p + \nu)!}{\nu\,!\,(t - \nu p)\,!}, \tag{12}$$

in which $Y(0) = eI$ and $Kp \leq t < (K+1)p$ ($K = 0, 1, \ldots$).

This allows a simple approximation of ρ to be derived. If K is a positive integer (not too small), say $K = 7$, and $t = Kp + 1$, then according to equations 11 and 12, ρ approximately equals

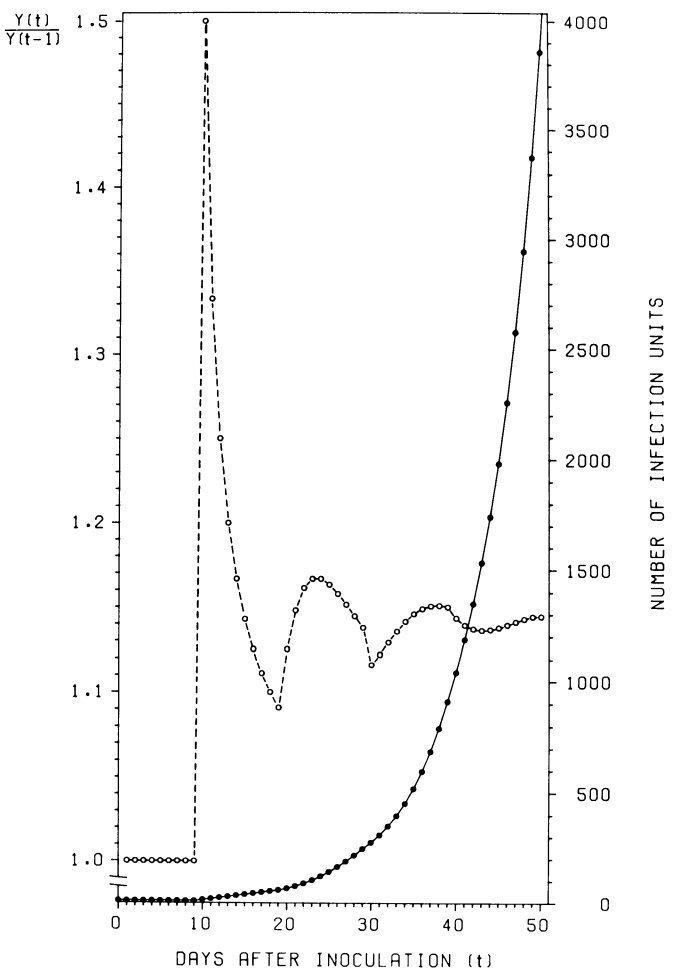

Fig. 1. Disease progress curve (-●—●-) and corresponding ratio $Y(t)/Y(t-1)$ (-○—○-) of a hypothetical epidemic. The number of infection units, $Y(t)$, was calculated according to equation 4, using the following values of parameters: latent period = 10, infectious period = 20, infection efficiency = 0.01, spore production rate = 500, deposition frequency = 0.1, and initial number of spores = 1,000.

$$Y(t)/\,Y(t-1)=\sum_{\nu=0}^{7}\frac{(eds)^{\nu}[(7-\nu)p+\nu+1]!}{\nu!\,[(7-\nu)p+1]!}\Bigg/\sum_{\nu=0}^{7}\frac{(eds)^{\nu}[(7-\nu)p+\nu]!}{\nu!\,[(7-\nu)p]!}.$$

Numerical studies show that the approximate and the exact value differ by less than 10^{-2}, at least for parameters lying within the range found in barley leaf rust.

Epidemics in cultivar mixtures. Let the pathogen population and the cultivar mixture consist of M pathotypes and N components, respectively. The definition of the rate of disease increase in the present case is derived from the following model, describing disease progress of a single pathotype m ($m = 1,...,M$) during the exponential phase of the epidemic (11, equation 5):

$$\begin{aligned}Y_{mn}(t)-Y_{mn}(t-1)&=de_{mn}as_{mn}[Y_{mn}(t-p_{mn})-Y_{mn}(t-p_{mn}-i_{mn})]\\&+de_{mn}(1-a)x_n\sum_{\nu=1}^{N}s_{m\nu}[Y_{m\nu}(t-p_{m\nu})\\&-Y_{m\nu}(t-p_{m\nu}-i_{m\nu})]\end{aligned}\qquad(13)$$

$$n=1,...,N$$

where a = autodeposition frequency (proportion of deposited spores that are deposited on their donor plants) and x_n = proportion of component n ($n = 1,...,N$) in the mixture

$$(\sum_{n=1}^{N}x_n=1)$$

The two summands on the right side of equation 13 represent the number of autoinfections and alloinfections, respectively, caused by pathotype m on component n at day t. The model incorporates time delays (latent and infectious period) and therefore generalizes earlier works of Barrett (1,2), Jeger et al (13), and Østergaard (18).

Given a primary inoculum of I_m deposited spores of pathotype m and the $p_{mn} + i_{mn}$ initial values

$$Y_{mn}(t)=\begin{cases}0\,,&-(p_{mn}+i_{mn})<t<0\\e_{mn}x_nI_m,&t=0\end{cases}\qquad(14)$$

for each component, equation 13 has N unique solutions $Y_{m1},...,Y_{mN}$. They can be determined either by iteration or by a standard method for systems of linear difference equations. The number of infection units caused by pathotype m in the mixture then equals

$$Y_m(t)=\sum_{n=1}^{N}Y_{mn}(t).\qquad(15)$$

The ratio $Y_m(t)/\,Y_m(t-1)$ behaves as in the case of pure stands; it shows wide fluctuations at the beginning of the season but very quickly reaches a limit which is denoted by ρ_m. Analogously to the monoculture case, ρ_m is identical with the largest positive root of the characteristic polynomial of equation 13. This polynomial is given here in the form of a determinant (det) whose elements themselves are polynomial functions:

$$P_m(\lambda)=\det\begin{Vmatrix}f_{11}(\lambda)&\dots&f_{1N}(\lambda)\\\vdots&&\vdots\\f_{N1}(\lambda)&\dots&f_{NN}(\lambda)\end{Vmatrix},\qquad(16)$$

where

$$f_{\nu\mu}(\lambda)=\begin{cases}(\lambda-1)\lambda^{p_{m\nu}+i_{m\nu}-1}-de_{m\nu}[a+(1-a)x_\nu]s_{m\nu}(\lambda^{i_{m\nu}}-1)\,,&\nu=\mu\\-de_{m\nu}(1-a)x_\nu s_{m\mu}(\lambda^{i_{m\mu}}-1)\,,&\nu\neq\mu\end{cases}$$

$$\nu,\mu=1,...,N$$

Consider a cultivar mixture with component frequencies $x_1,...,x_N$ and a pathotype m with rate of disease increase ρ_m. Let ρ_{mn} be the rate of disease increase on component n grown in monoculture. If $p_{m1} = p_{m2} = ... = p_{mN}$ and $i_{m1} = i_{m2} = ... = i_{mN}$, then

$$\rho_m>\sum_{n=1}^{N}x_n\rho_{mn}.$$

If, however, latent or infectious period differs among components, the inequality may be reversed. The identity

$$\rho_m=\sum_{n=1}^{N}x_n\rho_{mn}$$

is satisfied only under certain conditions, for instance, if $p_{mn} = 1$, $i_{mn} \to \infty$ ($n = 1,...,N$) and $a = 0$. If a increases (p_{mn} and i_{mn} arbitrarily chosen), ρ_m increases too and reaches a maximum for $a = 1$ when all spores are deposited on their donor plants. Then

$$\rho_m=max\{\rho_{m1},\dots,\rho_{mN}\},$$

regardless of the proportions of components. These findings are similar to those of Østergaard (18, page 167).

The rate of disease increase, being the largest positive root of the characteristic polynomial (equation 3 or 16), can easily be determined by means of an algorithm for the localization of roots, such as Newton's method (see reference 26 for a corresponding Fortran or Pascal program).

Illustration of ρ by using barley leaf rust data. In the monoculture case, the rate of disease increase depends only on the parameters p, i, e, s, and d but not on the amount of primary inoculum I. The functional dependence is illustrated in Figure 2. Here the parameters e, d, and s are combined multiplicatively into a single quantity; this is justified because ρ depends (besides p and i) only on the product of e, d, and s. It is seen from the figure that the surface is saddle shaped with a positive curvature in p-direction and a negative one in *eds*-direction. If i varies between 21 and 35 days (range found by J. E. Parlevliet, *personal communication*), the surface almost does not change its position, which can be shown numerically (the maximal distance between the surfaces is lower than 10^{-2}).

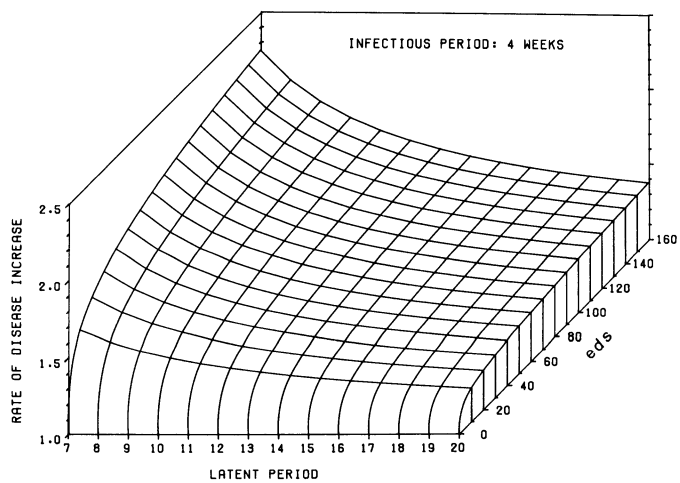

Fig. 2. Influence of latent period and of the product of infection efficiency (e) times deposition frequency (d) times spore production rate (s) on the rate of disease increase (ρ) in the barley leaf rust system. ρ was calculated from equation 3 by Newton's method. At temperatures between 15 and 20 C and lesion densities up to 50 cm^{-2}, the following ranges of epidemiological parameters were as follows (J. E. Parlevliet, *personal communication*): latent period = 7–20, infection efficiency = 0–0.8, and spore production rate = 300–2,000. Shaner and Hess (27) calculated a value of 0.02 for deposition frequency in wheat leaf rust. Assuming an upper limit of 0.1 for this parameter, we obtain a span of 0–160 for the quantity *eds*.

APPLICATIONS

The rate of disease increase can be applied to various problems, such as evaluation of cultivars for resistance, prediction of the long-term composition of pathotype mixtures, and development of disease control strategies.

Evaluation of cultivars for resistance. In Table 1, the components of partial resistance of eight barley cultivars to leaf rust isolate 1-2 are given. Cultivar L 94, having the shortest p and a large product of e and s, obviously is most susceptible. On the other side of the resistance spectrum is cultivar Vada, with the longest p and smallest product of e and s. With the other cultivars, however, a comparison of their partial resistance levels only by means of epidemiological parameters is difficult because of compensatory effects. For instance, Sultan has a smaller product of e and s than Zephyr, but this advantage in resistance is compensated by a shorter p. Gumpert et al (11), who previously used disease progress curves of isolate 1-2 on these two cultivars, could show that Sultan has a somewhat lower level of resistance than Zephyr. These findings are confirmed by the corresponding rates of disease increase given in Table 1. The measure ρ, taking into account all basic epidemiological parameters, reflects the partial resistance level of cultivars more reliably than one single parameter, such as latent period, would. This is particularly useful if the components of resistance display compensatory effects, as in the case of Sultan and Zephyr. Besides the components of partial resistance, total spore production per unit leaf area (measured at heading stage under greenhouse conditions) and disease score in the field also are given in Table 1. The disease score of cultivar L 94 (12.3) corresponds to a disease severity of about 6% (25, Table 1). The rate of disease increase turns out to be highly and significantly correlated both with total spore production per unit leaf area ($r = 0.97$) and with disease score in the field ($r = 0.81$).

Prediction of the long-term composition of pathotype mixtures. In Table 2, the epidemiological parameters of three leaf rust isolates on cultivar Vada are given. The isolates 22 and 11-1 have equal latent periods, infectious periods, and spore production rates, but 11-1 has a higher infection efficiency and is therefore the more aggressive isolate. A comparison of isolates 1-2 and 22, as well as 1-2 and 11-1, only by means of epidemiological parameters is difficult because of compensatory effects. The corresponding rates of disease increase, however, clearly indicate that 11-1 is the most aggressive isolate, followed by 22 and 1-2. Isolate 11-1, mixed with the others, will gain predominance in the long run. This, however, requires that the ranking of isolates is the same irrespective of whether they occur alone or in mixture with each other. Under this condition, it can be shown that in any heterogeneous pathogen population the pathotype with the largest ρ value will predominate in the long run, regardless of its proportion in the original inoculum mixture. The frequency of the predominant pathotype in the mixture may increase continually or may fluctuate, depending on the situation. For instance, if pathotypes reproduce synchronously (equal latent periods) and if the infectious periods are nearly equal, then the predominant pathotype increases monotonically in its frequency, at least in pure stands. This theoretical result is in good accordance with experimental observations (4,14,19). If, on the other hand, pathotypes reproduce asynchronously (different latent periods), then the frequencies may be subject to heavy fluctuations (11).

A strategy for disease control. In a heterogeneous pathogen population, the pathotype with the largest ρ value will predominate in the long run. This holds for pure stands as well as cultivar mixtures. If one of them is changed, the predominant pathotype may change, too. For any cultivar composition, specified by the frequencies $x_1,...,x_N$, let $\rho_m(x_1,...,x_N)$ be the corresponding rate of disease increase of pathotype m ($m = 1,...,M$). Because in the long run disease increases at the rate of the predominant pathotype, $max_m[\rho_m(x_1,...,x_N)]$, the grower's objective is essentially to find that set of x_n's, for which $max_m[\rho_m(x_1,...,x_N)]$ is minimal. This *optimal* cultivar composition is either a monoculture or a mixture, depending on the situation. For instance, if on each host component one and the same pathotype predominates, then at optimum only a single component will be present, namely the one with the greatest amount of resistance to the predominant pathotype. If, however, the predominant pathotype differs among components, a cultivar mixture may be more beneficial than each of its components grown in monoculture. This is illustrated by a simple example of two pathotypes (leaf rust isolates 11-1 and 18) and two cultivars (barley cultivars Berac and Julia). The epidemiological parameters are given in Table 3. The corresponding ρ values indicate that, on cultivar Berac, isolate 11-1 is better adapted than isolate 18, whereas on Julia the situation is reversed. To determine the optimal composition of Berac and Julia, the rates of disease increase are needed for both isolates and any proportions of the two cultivars. The rates are calculated from equation 16 by Newton's method. For the special case of two host components, this equation simplifies to:

$$P_m(\lambda) = \{(\lambda - 1)\lambda^{p_{m1}+i_{m1}-1} - de_{m1}[a + (1-a)x_1]s_{m1}(\lambda^{i_{m1}} - 1)\} \cdot \{(\lambda - 1)\lambda^{p_{m2}+i_{m2}-1} - de_{m2}[a + (1-a)x_2]s_{m2}(\lambda^{i_{m2}} - 1)\} - d^2(1-a)^2 x_1 x_2 e_{m1} e_{m2} s_{m1} s_{m2}(\lambda^{i_{m1}} - 1)(\lambda^{i_{m2}} - 1) \qquad m = 1,2 \tag{17}$$

TABLE 1. Traits of partial resistance of eight barley cultivars to leaf rust isolate 1-2

Trait	Unit[a]	Cultivar							
		L 94	L 98	Mamie	Sultan	Zephyr	Volla	Julia	Vada
Latent period (p)[b]	day	8	9	10	10	11	9	12	15
Infectious period (i)[b]	day	31	28	29	31	32	27	28	21
Infection efficiency (e)[b]	1	0.16	0.09	0.09	0.08	0.08	0.09	0.07	0.05
Spore production rate (s)[b]	day^{-1}	1,120	2,200	1,240	960	1,010	1,400	750	800
Deposition frequency (d)[c]	1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Rate of disease increase (ρ)[d]	day^{-1}	1.62	1.56	1.43	1.39	1.36	1.50	1.30	1.23
Total spore production per unit leaf area[e]	cm^{-2}	100	97	51	43	45	57	25	14
Disease score in the field[f]	1	12.3	11.9	11.4	8.3	8.6	6.1	4.7	3.1

[a] "1" indicates that the parameter is dimensionless.
[b] Latent period = time (days) between inoculation and 50% of pustules just visible (23). Data were calculated from the relative values given by Neervoort and Parlevliet (17, Table 1) and the absolute values for Vada: $p = 15$ (24, Table 2); $i = 21$ (J. E. Parlevliet, *personal communication*); $e = 0.05$ (24, Table 3, setting infection efficiency equal to infection frequency $\times 10^{-2}$); and $s = 800$ (J. E. Parlevliet, *personal communication*).
[c] Assumed values.
[d] Calculated from equation 3 by Newton's method.
[e] From Neervoort and Parlevliet (17, Table 1).
[f] From Neervoort and Parlevliet (17, Table 2). The scores are logarithmic transformations of the number of pustules per tiller; each unit increase on the scale corresponds approximately with a two fold increase in amount of disease (25).

Figure 3 shows the rates of disease increase of both isolates in the cultivar mixture at varying proportions of cultivar Berac. The maximum of both curves (solid line) is minimized at 0.69, which is the optimal proportion of cultivar Berac in the mixture.

For more than two pathotypes, we proceed analogously. First, the maximum curve from the rates of disease increase is constructed and then its minimum is determined. In case of more than two host components, the procedure becomes much more tedious because curves are then to be replaced by surfaces or higher dimensional manifolds. This difficulty, however, is only of a technical, not fundamental, nature.

DISCUSSION

The rate of disease increase, ρ, presented in this paper is derived from a model previously published by Gumpert et al (11). Given the host's composition and the basic epidemiological parameters p, i, e, s, d, and a, ρ quantifies disease progress in pure and mixed stands of plant cultivars. The measure does not account for changes in the weather conditions and their effects on the epidemiological parameters. The applicability of this measure hinges upon the condition that the epidemic stays in its exponential phase throughout the whole disease season; that is, disease increase is density independent. Furthermore, it is assumed that antagonistic or synergistic effects between pathotypes, such as induced resistance or induced susceptibility (for a review, see reference 5), are negligible. The epidemiological parameters of a pathotype are therefore the same irrespective of whether it occurs alone or in mixture. These are realistic assumptions as long as leaf area is large compared to the number of infection units on it.

TABLE 2. Traits of aggressiveness of three leaf rust isolates on cultivar Vada

Trait	Unit[a]	Isolate 1-2	Isolate 22	Isolate 11-1
Latent period (p)[b]	day	15	16	16
Infectious period (i)[c]	day	21	21	21
Infection efficiency (e)[d]	1	0.05	0.08	0.12
Spore production rate (s)[e]	day^{-1}	800	700	700
Deposition frequency (d)[e]	1	0.1	0.1	0.1
Rate of disease increase (ρ)[f]	day^{-1}	1.23	1.24	1.26

[a]See Table 1.
[b]Latent period = time (days) between inoculation and 50% of pustules just visible (23); data (rounded) from Parlevliet (24, Table 2).
[c]From J. E. Parlevliet (*personal communication*).
[d]From Parlevliet (24, Table 3, setting infection efficiency equal to infection frequency $\times 10^{-2}$).
[e]Assumed values.
[f]Calculated from equation 3 by Newton's method.

TABLE 3. Traits of aggressiveness of leaf rust isolates 11-1 and 18 on barley cultivars Berac and Julia

Trait	Unit[a]	11-1 Berac	11-1 Julia	18 Berac	18 Julia
Latent period (p)[b]	day	14	14	14	13
Infectious period (i)[c]	day	28	28	28	28
Infection efficiency (e)[d]	1	0.12	0.15	0.04	0.08
Spore production rate (s)[e]	day^{-1}	900	400	2,100	1,100
Deposition frequency (d)[e]	1	0.1	0.1	0.1	0.1
Rate of disease increase (ρ)[f]	day^{-1}	1.31	1.27	1.29	1.32

[a]See Table 1.
[b]Latent period = time (days) between inoculation and 50% of pustules just visible (23); data (rounded) from Parlevliet (24, Table 2).
[c]From J. E. Parlevliet (*personal communication*).
[d]From Parlevliet (24, Table 3, setting infection efficiency equal to infection frequency $\times 10^{-2}$).
[e]Assumed values.
[f]Calculated from equation 3 by Newton's method.

Being the limit of the ratio $Y(t)/Y(t-1)$, ρ is reached exactly only if $t \to \infty$, assuming that disease is still exponentially increasing. Hence, ρ is an ideal long-term measure and as such can only be approximated by $Y(t)/Y(t-1)$ during the relatively *short* period of a disease season. The approximation is better the more rapidly initial waves fade out. The fading out is accelerated by a short p and (to a lesser extent) a small a, e, s, and d. A variable latent period, as found, for example, in leaf rust of wheat (28), may also have some damping effect on the initial waves. More precisely, if the parameter values are the same as in Figure 1 but latent period is now considered as a discrete random variable with mean value $\bar{p} = 10$, then the early fluctuations would probably be considerably smaller than those shown in Figure 1. This conjecture is supported by findings with continuous infection processes (29, pages 66–67). The rate of disease increase can be applied to efficiently compare the resistance level of cultivars, as was demonstrated by using barley leaf rust data (Table 1). Leonard and Mundt (16, Table 3), who previously dealt with the same problem, used a somewhat different measure, associated with a time continuous model. They chose for the most susceptible cultivar, L 94, a shorter latent period than we did (5 days instead of 8) but the relative values were the same as in Table 1. Their measure (r_1) is, compared to ours, somewhat more highly correlated with total spore production per unit leaf area ($r = 0.98$) and with disease score in the field ($r = 0.88$). This is probably due to the spore production pattern included in their model. However, their model has only been constructed for epidemics in pure stands.

A simple measure for predicting trends in pathotype mixtures has been proposed by Ogle et al (20). The authors do not consider latent and infectious period individually, but instead combine them additively into the so-called "average life span of uredia." In our notation, their "measure of relative survival ability" (20, equation 8) can be written in the form

$$(es)^{1/(p+i-1)}$$

Being a function of p and es, this measure behaves very much like ρ as shown in Figure 2. In contrast to ρ, however, the measure decreases as i increases.

If several cultivars are available, the rate of disease increase can serve as a useful tool for developing disease control strategies. The concept of optimal cultivar compositions presented here improves the ideas of Fleming and Person (7) in three ways. Firstly, it extends the theory by allowing for differences in latent period; secondly, selection against unnecessary genes for virulence

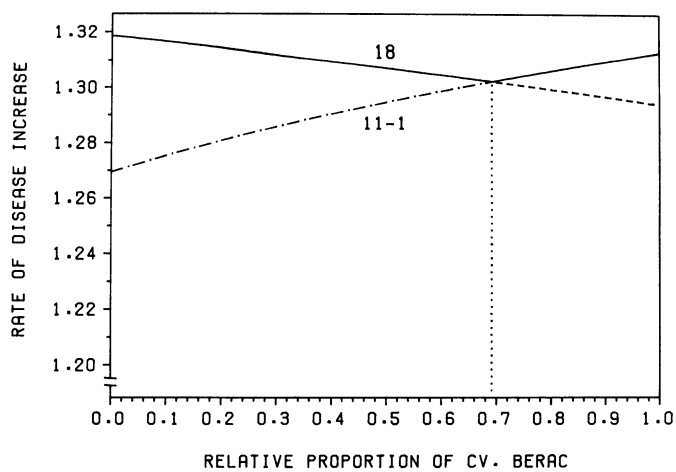

Fig. 3. Rates of disease increase of leaf rust isolates 11-1 and 18 in a mixture of cultivars Berac and Julia as a function of the proportion of cultivar Berac. The rates were calculated from equation 17 by Newton's method, using the parameters given in Table 3 and an autodeposition frequency of 0.1.

is not necessarily operating; and thirdly, our concept takes into account differences in the genetic background of pathotypes as well as cultivars. The optimal cultivar composition essentially depends on the presence of the spectrum of pathotypes. As long as this spectrum remains unchanged, the optimal composition (possibly a monoculture) guarantees maximal protection against the pathogen. After several seasons, however, new, more aggressive pathotypes may emerge owing to migration, mutation, or sexual recombination. Because our concept is based upon the long-term composition of the pathogen population, adjustments of the cultivar composition to the changed pathotype spectrum should be made when the pathogen population has stabilized. A stable state is usually reached much more slowly with cultivar mixtures than with monocultures.

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. (Pflanzenpathol.) Pflanzenschutz 87:383-396.
3. Berger, R. D. 1977. Application of epidemiological principles to achieve plant disease control. Ann. Rev. Phytopathol. 15:165-183.
4. Brown, J. F., and Sharp, E. L. 1970. The relative survival ability of pathogenic types of *Puccinia striiformis* in mixures. Phytopathology 60:529-533.
5. 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.
6. Corsten, L. C. A. 1964. Een kwantitatieve beschrijving van de ontwikkeling van een schimmelpopulatie. Meded. Landbouwhogesch. Wageningen 64-15:1-7.
7. Fleming, R. A., and Person, C. O. 1978. Disease control through use of multilines: A theoretical contribution. Phytopathology 68:1230-1233.
8. Fry, W. E. 1975. Integrated effects of polygenic resistance and a protective fungicide on development of potato late blight. Phytopathology 65:908-911.
9. Fry, W. E. 1977. Integrated control of potato late blight—Effects of polygenic resistance and techniques of timing fungicide applications. Phytopathology 67:415-420.
10. Fry, W. E. 1978. Quantification of general resistance of potato cultivars and fungicide effects for integrated control of potato late blight. Phytopathology 68:1650-1655.
11. Gumpert, F.-M., Geiger, H. H., and Staehle, U. 1987. A mathematical model of the epidemics in homogeneous and heterogeneous host stands. Z. Pflanzenkrankh. (Pflanzenpathol.) Pflanzenschutz 94:206-215.
12. Henrici, P. 1964. Elements of Numerical Analysis. John Wiley & Sons, New York. 336 pp.
13. Jeger, M. J., Griffiths, E., and Jones, D. G. 1981. Disease progress of nonspecialised fungal pathogens in intraspecific mixed stands of cereal cultivars. I. Models. Ann. Appl. Biol. 98:187-198.
14. Keed, B. R. 1968. Wheat rust assessment. Ph.D. thesis. University of Sydney. 125 pp.
15. Latin, R. X., MacKenzie, D. R., and Cole, H., Jr. 1981. The influence of host and pathogen genotypes on the apparent infection rates of potato late blight epidemics. Phytopathology 71:82-85.
16. Leonard, K. J., and Mundt, C. C. 1984. Methods for estimating epidemiological effects of quantitative resistance to plant diseases. Theor. Appl. Genet. 67:219-230.
17. Neervoort, W. J., and Parlevliet, J. E. 1978. Partial resistance of barley to leaf rust, *Puccinia hordei*. V. Analysis of the components of partial resistance in eight barley cultivars. Euphytica 27:33-39.
18. Østergaard, H. 1983. Predicting development of epidemics on cultivar mixtures. Phytopathology 73:166-172.
19. Ogle, H. J., and Brown, J. F. 1970. Relative ability of two strains of *Puccinia graminis tritici* to survive when mixed. Ann. Appl. Biol. 66:273-279.
20. Ogle, H. J., Taylor, N. W., and Brown, J. F. 1973. A mathematical approach to the prediction of differences in the relative ability of races of *Puccinia graminis tritici* to survive when mixed. Aust. J. Biol. Sci. 26:1137-1143.
21. Oort, A. J. P. 1968. A model of the early stage of epidemics. Neth. J. Plant Pathol. 74:177-180.
22. Ostrowski, A. M. 1966. Solution of Equations and Systems of Equations. Academic Press, New York. 338 pp.
23. Parlevliet, J. E. 1976. Evaluation of the concept of horizontal resistance in the barley/*Puccinia hordei* host-pathogen relationship. Phytopathology 66:494-497.
24. Parlevliet, J. E. 1977. Evidence of differential interaction in the polygenic *Hordeum vulgare-Puccinia hordei* relation during epidemic development. Phytopathology 67:776-778.
25. Parlevliet, J. E., and Van Ommeren, A. 1975. Partial resistance of barley to leaf rust, *Puccinia hordei*. II. Relationship between field trials, micro plot tests and latent period. Euphytica 24:293-303.
26. Press, W. H., Flannery, B. P., Teukolsky, S. A., and Vetterling, W. T. 1986. Numerical Recipes. The Art of Scientific Computing. Cambridge University Press, New Rochelle, NY. 818 pp.
27. Shaner, G., and Hess, F. D. 1978. Equations for integrating components of slow leaf-rusting resistance in wheat. Phytopathology 68:1464-1469.
28. Shaner, G., Ohm, H. W., and Finney, R. E. 1978. Response of susceptible and slow leaf-rusting wheats to infection by *Puccinia recondita*. Phytopathology 68:471-475.
29. Van der Plank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.
30. Van der Plank, J. E. 1975. Principles of Plant Infection. Academic Press, New York. 216 pp.
31. Zadoks, J. C. 1971. Systems analysis and the dynamics of epidemics. Phytopathology 61:600-610.
32. Zadoks, J. C., and Schein, R. D. 1979. Epidemiology and Plant Disease Management. Oxford University Press, New York. 427 pp.