Measuring Disease Progress in Pure and Mixed Stands of Plant Cultivars

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ABSTRACT


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, sporulation rate production) 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 sporulation per unit leaf area (r = 0.97) and with disease score in the field (r = 0.81).

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 sporulation, 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, 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, Östergaard (18) suggested the “long-term rate of disease increase.” She takes into account infection efficiency, sporulation 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-sporation (beginning with spor 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:

\[ Y(t) - Y(t-1) = ed_s [Y(t-p) - Y(t-p-i)] \]

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); \( s \) = sporulation 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 \( ed_s \) (= 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,

\[ Y(t) = \begin{cases} 0, & t < 0 \\ e^l, & t = 0 \end{cases} \]

where \( l \) 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:

\[ P(\lambda) = \lambda^{p+i} - \lambda^{p+i-1} - ed_s (\lambda^i - 1) \]
If $\lambda_1, \ldots, \lambda_i$ are the $i$ ($i \leq p + i$) distinct roots of $P$ and $k_1, \ldots, k_n$ the corresponding multiplicities (repeated roots), then the solution $Y(t)$ is given by:

$$Y(t) = \sum_{j=1}^i \left( \sum_{\mu=1}^{k_j} c_{\mu j} t^{\lambda_j} \right) \lambda_j^i, \quad t > -(p + i).$$  \hfill (4)

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

$$\sum_{j=1}^i \left( \sum_{\mu=1}^{k_j} c_{\mu j} t^{\lambda_j} \right) \lambda_j^i = 0, \quad -(p + i) < t < 0$$

and

$$\sum_{j=1}^i c_{\mu j} = e^t.$$

The asymptotic behavior of the ratio $Y(t)/Y(t - 1)$ is studied next. Given arbitrary values of the parameters $p, i, e, s, d, I$, and $L$, 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

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

The polynomial $Q$ has exactly one positive root $\lambda^*$. By a theorem of Cauchy (compare with reference 22, pages 96–97), $\lambda^*$ is simple, that is, it has multiplicity 1. Moreover, if $i > 1$, $\lambda^*$ 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 $\lambda_1, \ldots, \lambda_i$ in equation 4. Without loss of generality, it can be supposed that

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

$$i.e., \quad \lambda_1 > |\lambda_2|, \ldots, |\lambda_i|.$$  \hfill (6)

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

**Case 1: $eds \neq 1$.**

Then $\lambda^* \neq 1$ and $\lambda_1$ has multiplicity 1; that is, $k_1 = 1$. By equation 4, we obtain

$$Y(t) = c_{11} \lambda_1^i + \sum_{j=2}^i \left( \sum_{\mu=1}^{k_j} c_{\mu j} t^{\lambda_j} \right) \lambda_j^i,$$  \hfill (7)

**Case 2: $eds = 1$.**

Then $\lambda^* = 1$ and $\lambda_1 (= 1)$ has multiplicity 2; that is, $k_1 = 2$. By equation 4, we obtain

$$Y(t) = (c_{11} + c_{12}) \lambda_1^i + \sum_{j=2}^i \left( \sum_{\mu=1}^{k_j} c_{\mu j} t^{\lambda_j} \right) \lambda_j^i.$$  \hfill (8)

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

$$c_{11} \neq 0$$  \hfill (9)

in case 1 and

$$c_{11} \neq 0 \quad \text{or} \quad c_{12} \neq 0$$  \hfill (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

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

where $\lambda_1$ 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 $P(\lambda_1)$. According to this definition, $P$ 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 $P$. 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):

$$Y(t) = Y(0) \sum_{k=0}^K \frac{(eds)(t - vp + v)!}{v!(t - vp)!},$$  \hfill (12)

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

This allows a simple approximation of $P$ 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, $P$ 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.](image-url)
Consider a cultivar mixture with component frequencies \( x_1, \ldots, x_N \) and a pathotype \( m \) with rate of disease increase \( \rho_m \). Let \( \rho_m \) be the rate of disease increase on component \( n \) grown in monoculture. If \( \rho_m = \rho_{m1} = \cdots = \rho_{mN} \) and \( i_m = i_{m1} = \cdots = i_{mN} \), 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

\[
\rho_m = \max(\rho_{m1}, \ldots, \rho_{mN}),
\]

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 \( \rho \) 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 \( p \) 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 the direction of \( I \); and a negative one in the \( e, d \) 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 (\( \rho \)) in the barley leaf rust system. \( \rho \) 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 \( \rho \).
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 \( p \), 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 \( p \) 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 \( p \) 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 \( x_1, \ldots, x_N \), let \( \rho_m(x_1, \ldots, x_N) \) be the corresponding rate of disease increase of pathotype \( m \) \((m = 1, \ldots, M)\). Because in the long run disease increases at the rate of the predominant pathotype, \( \max_m[\rho_m(x_1, \ldots, x_N)] \) 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 \( \rho \) 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:

\[
P_m(\lambda) = \frac{[\lambda(1 - \lambda)p_m + \lambda] - \alpha_m[1 + (1 - \alpha)X_0] \mu_m(\lambda^{-m} - 1) \cdot [\lambda(1 - \lambda)p_m + \lambda] - \alpha_m[1 + (1 - \alpha)X_0] \mu_m(\lambda^{-m} - 1)}{-\alpha_m[1 + (1 - \alpha)X_0] \mu_m(\lambda^{-m} - 1)}
\]

\( m = 1, 2 \)

(17)

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

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

\(^a\) 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); \( t = 21 \) (J. E. Parlevliet, personal communication); \( e = 0.05 \) (24, Table 3, setting infection efficiency equal to infection frequency \( \times 10^{-3} \)); 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, \( \rho \), 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 \( a, \rho \) 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

<table>
<thead>
<tr>
<th>Trait</th>
<th>Isolate</th>
<th>I-2</th>
<th>22</th>
<th>11-1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latent period ((p)^a)</td>
<td>day</td>
<td>14</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td>Infectious period ((i)^a)</td>
<td>day</td>
<td>28</td>
<td>28</td>
<td>28</td>
</tr>
<tr>
<td>Infection efficiency ((e)^a)</td>
<td>1</td>
<td>1</td>
<td>0.12</td>
<td>0.15</td>
</tr>
<tr>
<td>Spore production rate ((s)^a)</td>
<td>day</td>
<td>400</td>
<td>200</td>
<td></td>
</tr>
<tr>
<td>Deposition frequency ((d)^a)</td>
<td>1</td>
<td>0.1</td>
<td>0.1</td>
<td></td>
</tr>
<tr>
<td>Rate of disease increase ((p)^a)</td>
<td>day</td>
<td>1.23</td>
<td>1.24</td>
<td></td>
</tr>
</tbody>
</table>

\(a\) See Table 1.

\(b\) Assumed values.

\(c\) 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

<table>
<thead>
<tr>
<th>Trait</th>
<th>Isolate</th>
<th>Berac</th>
<th>Julia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Latent period ((p)^a)</td>
<td>day</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td>Infectious period ((i)^a)</td>
<td>day</td>
<td>28</td>
<td>28</td>
</tr>
<tr>
<td>Infection efficiency ((e)^a)</td>
<td>1</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Spore production rate ((s)^a)</td>
<td>day</td>
<td>400</td>
<td>200</td>
</tr>
<tr>
<td>Deposition frequency ((d)^a)</td>
<td>1</td>
<td>0.1</td>
<td></td>
</tr>
<tr>
<td>Rate of disease increase ((p)^a)</td>
<td>day</td>
<td>1.31</td>
<td>1.27</td>
</tr>
</tbody>
</table>

\(a\) See Table 1.

\(b\) Assumed values.

\(c\) Calculated from equation 3 by Newton's method.

Being the limit of the ratio \( Y(t)/Y(t-1) \), \( \rho \) is reached exactly only if \( t \to \infty \), assuming that disease is still exponentially increasing. Hence, \( \rho \) 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 \( \bar{a} = 10 \), 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, L94, 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 \((\rho)\) is, compared to ours, somewhat more highly correlated with total spore production per unit leaf area \((\bar{r} = 0.98)\) 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

\[
(e)^{(p+1-i)}
\]

Being a function of \( p \) and \( e \), this measure behaves very much like \( \rho \) as shown in Figure 2. In contrast to \( \rho \), 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
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


