Theor Appl Genet (1984) 67:219–230

Methods for estimating epidemiological effects of quantitative resistance to plant diseases *

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Received June 16, 1983
Communicated by G. S. Khush

Summary. A model developed by R.C. Lewontin relating rate of population increase to key parameters of the organism's fecundity curve is described and adapted for use with plant pathogenic fungi. For diseases such as cereal rusts, rice blast, and powdery mildew and downy mildew of cucumber, the sporulation curves for the pathogens have been shown to follow an approximately triangular pattern. In the Lewontin model the key features of the pattern are: A, the time from inoculation to first sporulation (i.e. latent period); T, the time of peak spore production per day; W, the time at which sporulation ceases; and S, the area of the triangle (total reproduction per generation). For exponential increase, the values of A, T, W, and S are related to r_l, the rate of population increase, according to the following equation:

$$\gamma^2 (W-A)/2S = [(e^{-rA}-e^{-rT})/(T-A)] + [(e^{-rW}-e^{-rT})/(W-T)].$$

This equation was used to generate families of curves showing effects on r_l of changes in the position of the triangle (altering latent period) or area (altering reproduction per generation). Data for barley leaf rust, oat crown rust, wheat leaf rust, wheat stem rust, rice blast, cucumber downy mildew, and cucumber powdery mildew were analyzed according to the model to show the effects of different components of resistance on r_l for each disease. Predictions from the model for barley leaf rust were compared with published data for components of resistance and rates of disease increase for eight barley cultivars. For cultivars of similar crop canopy type (two cultivars sparse; six cultivars, dense canopies), the predicted r_l values closely corresponded to observed values. Applications of the model to cultivar mixtures and to integrated control (involving protectant fungicides in combination with quantitative resistance) are also discussed.

Key words: Components of resistance – Quantitative resistance – Epidemiology – Cereal rusts – Sporulation patterns

Introduction

For many plant diseases, particularly those caused by biotropic fungi, monogenic resistance occurs in gene-for-gene relationships in which each major gene for resistance can be rendered ineffective by a corresponding virulence gene in the pathogen (Day 1974). Although the major genes for disease resistance selected in commercial cultivars are highly effective in reducing or preventing reproduction by races of the pathogen that lack the necessary matching virulence genes, they do not appreciably affect reproduction by the virulent races. Therefore, the widespread use of monogenic resistance in a crop imposes extreme selection pressure on the pathogen population that can lead to rapid build-up of pathogenic races with genes for virulence that match the resistance genes used in the crops. With diseases such as stem rust of wheat and powdery mildew of barley, as well as many others, each time a new set of pathogenic races was selected, new major genes for resistance had to be found and incorporated into commercial cultivars of the crop in order to maintain or restore its resistance.

* Cooperative investigations of the US Department of Agriculture, Agricultural Research Service and the North Carolina Agricultural Research Service. Paper No. 8802 of the journal series of the North Carolina Agricultural Research Service, Raleigh, NC 27650, USA

To escape from this "boom or bust" cycle of the build-up of new, virulent races of pathogens following the release of new cultivars, plant breeders have begun to turn to the use of quantitative, polygenically-inherited resistance for many crops. While polygenic resistance does not usually inhibit pathogen reproduction as completely as monogenic resistance does, its advantage lies in its greater durability. Plant pathologists still argue the question of whether it is impossible or merely very difficult for pathogens to adapt to polygenic resistance (Nelson 1978; Vanderplank 1978). They agree, however, that if such adaptation can occur, it will proceed much more slowly than in the case of monogenic resistance involving gene-for-gene relationships with pathogen virulence.

The disadvantage of polygenic resistance, in addition to the greater difficulty in transferring it to commercial cultivars, lies in its quantitative nature. It requires more careful evaluation to recognize quantitative resistance, and it is difficult to measure it and accurately assess its practical value in crop production. As Vanderplank (1968) pointed out, quantitative resistance is at its worst in small, experimental field plots. In typical tests, small plots of susceptible check cultivars are included for comparisons with quantitatively resistant cultivars. Pathogens of polycyclic diseases such as rusts and powdery mildews build up rapidly on the susceptible cultivars and spread into the plots of resistant cultivars. This interplot interference reduces the contrast between susceptible and resistant cultivars and masks the full value that the resistance would have in large commercial fields. For example, Parlevliet (1979) showed that when interplot interference was prevented, the barley cultivars 'L94' and 'Vada' differed by a factor of more than 2500 in the numbers of uredia of *Puccinia hordei* per culm near the end of a field epidemic. On the other hand, when 'L94' and 'Vada' were grown in adjacent 4-row plots, they differed by a factor of only 30 in numbers of uredia per culm.

An alternative approach in assessment of quantitative resistance is the measurement of certain components of resistance in controlled inoculation experiments. Although the disease cycle can be divided into a great many more or less distinct steps, the critical components of quantitative resistance can be thought of as resistance that reduces infection efficiency, extends the latent period from inoculation to sporulation, and reduces sporulation (Parlevliet 1979). Zadoks (1971) suggested that infection efficiency and spore production can be combined into a daily multiplication factor. His simulation studies showed that r, Vanderplank's (1963) apparent infection rate (rate of disease increase), varied greatly with changes in latent period but was less sensitive to changes in the daily multiplication factor.

With sufficiently precise techniques, the parameters infection efficiency, latent period, and spore production per lesion can be measured accurately for cultivars with differing levels of quantitative resistance. Accurate measurements of components of resistance, however, are not sufficient in themselves for accurate assessment of the epidemiological effects of the particular combination of levels of each component characteristic of each resistant cultivar. Zadok's (1971) simulation model and Shaner and Hess' (1978) equations for integrating components of resistance can be used to compare the values of quantitative resistance of different cultivars. Both of these approaches, however, require extensive sequences of complex calculations, and neither approach sufficiently accounts for variations in daily sporulation rates during the infectious periods of individual lesions that typically occur for many pathogens. The purposes of our study were to develop simpler methods for estimating epidemiological effects of quantitative resistance and to demonstrate their application to a variety of plant diseases. The work reported here is an extension of a preliminary study briefly reported by Stefano and Leonard (1978).

Description of the Lewontin model

Lewontin (1965) used the Volterra equation to analyze the effects of changes in certain life cycle components in colonizing species on their intrinsic rates of population increase. In the initial stages of a colonizing episode the population may be assumed to grow exponentially. According to the Volterra equation, during exponential increase

$$\int_0^\infty e^{-rt}\, l(t)\, m(t)\, \mathrm{d}t = 1$$

where e is the base of natural logrithms, r is the intrinsic rate of increase of the population and is equivalent to Vanderplank's (1963) r_1 for exponential increase of disease; $l(t)$ is a function describing the probability that individuals will survive to age t; $m(t)$ is a function describing the number of progeny produced by surviving individuals of age t; and e^{rt} represents exponential increase as it does in Vanderplank's (1963) equation for exponential increase of disease $x/x_0 = e^{r_1 t}$, in which x_0 is the initial amount of disease and x is the amount at time t.

Lewontin (1965) simplified the Volterra equation by combining $l(t)$ and $m(t)$ into one function $V(t)$, which may be interpreted as the number of progeny per individual at t days after its birth. He showed that for many organisms, the function $V(t)$ can be represented as triangular (Fig. 1). The organism matures at age A and begins to reproduce. Its peak reproductive output is reached at age T, after which reproduction declines until it ends at age W. The parameter S in Fig. 1 is the area of the triangle enclosed by $V(t)$ and represents the total reproduction by an individual over its lifespan. This can be expressed as

$$S = \int_0^\infty V(t)\, \mathrm{d}t = \tfrac{1}{2}(W-A) \cdot V(T).$$

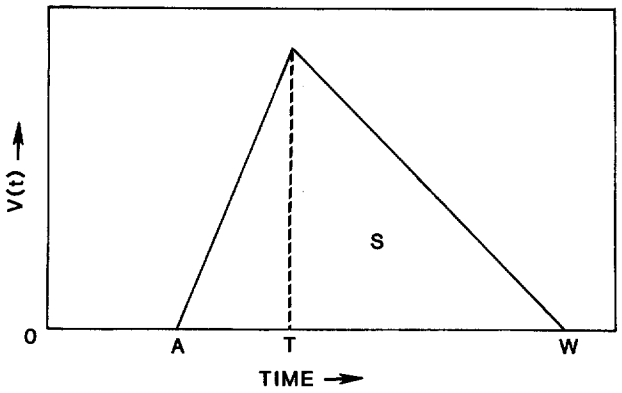

Fig. 1. Generalized, triangular pattern of fecundity for organisms to which the Lewontin model applies. The function $V(t)$ represents the number of progeny per individual at t days after birth. A represents the time of maturation at which reproduction begins; T, the age of peak reproductive capacity; W, the age at which reproduction ceases; and S, the area of the triangle, represents total reproduction per generation

Lewontin (1965) showed that for the parameters as represented in Fig. 1:

$$V(t)=2S(W-t)/[(W-T)(W-A)] \quad t \geqq T$$
$$V(t)=2S(t-A)/[(T-A)(W-A)] \quad t < T$$

Substituting these equations into Lewontin's modified Voltera equation yields the following expression relating r, the rate parameter, to the four critical parameters of the reproduction cycle, A, T, W, and S:

$$r^2(W-A)/2S=[(e^{-rA}-e^{rT})/(T-A)]+[(e^{-rW}-e^{-rT})/(W-T)] \quad \text{(Eq. 1)}$$

We could not solve this equation mathematically for r, but it can be solved numerically for a variety of combinations of values of r, A, T, and W, with S representing the unknown. The solutions can then be used to generate a series of curves showing the effects on r of varying the values of the other parameters.

Application of the Lewontin model to plant pathogenic fungi

Sporulation curves for many important plant pathogenic fungi approximate the triangular pattern used by Lewontin (1965) in his model. Curves for *Pyricularia oryzae* (rice blast) and *Puccinia graminis* f. sp. *tritici* (wheat stem rust) are presented in Figs. 2 and 3, respectively, as examples. We used the data in Figs. 2 and 3 and data from five other plant pathogens (Table 1) to demonstrate the application of Lewontin's model to the estimation of epidemiological effects of quantitative resistance. These pathogens were selected on the basis of availability of data for daily spore production in disease lesions and on the fit of these data to the triangular pattern. Sporulation curves were chosen to represent cases of disease on highly susceptible cultivars in environments highly favorable for disease development.

For each of the pathogens listed in Table 1, the values of A, T, and W were determined for triangles drawn by hand to fit the sporulation curves. Values of r for infection rates on highly susceptible cultivars under environmental conditions highly conducive to disease were selected as best estimates based on published records of disease progress during epidemics of these diseases. Values of S would be extremely difficult to determine directly by experimentation because of the complex nature of this parameter. In the model, S represents the combination of total sporulation, dispersal and deposition of the spores on host plants, survival and successful germination of the spores, and infection efficiency of viable spores on a suitable host. The value of S can be regarded as the number of daughter lesions that result from each parent lesion during one complete pathogen generation.

In calculating changes in r_1 that would result from increasing the latent period of a disease, we assumed

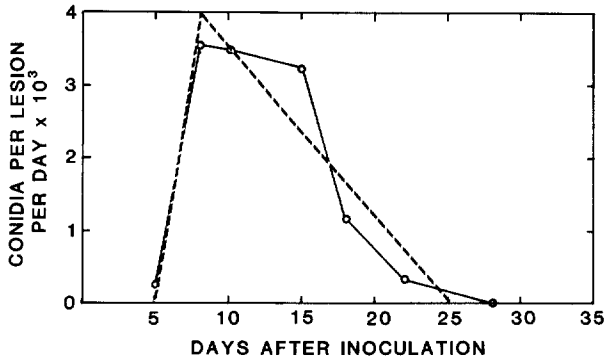

Fig. 2. Daily spore production by *Pyricularia oryzae* in rice blast lesions. Data are from Kato and Kozaka (1974) for sporulation on susceptible rice plants at 25 °C

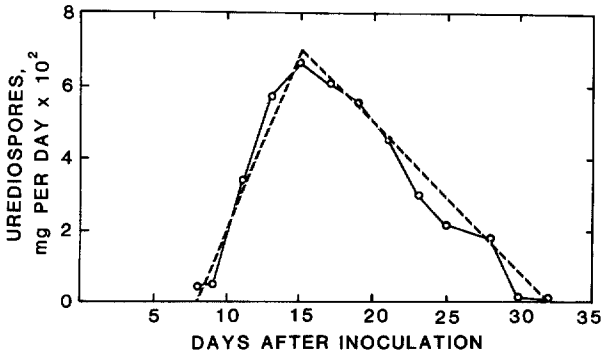

Fig. 3. Daily spore production (in mg. spores per infected plant) by *Puccinia graminis* f. sp. *tritici* in wheat stem rust pustules. Data are from Mortensen and Green (1978) for the susceptible cultivar 'Klein Titan' at 25 °C

Table 1. Times from inoculation to initiation of sporulation (A), peak sporulation (T), and cessation of sporulation (W) derived from representative data for seven foliar pathogens

Pathogen/disease	Parameter values (days)			Data source
	A	T	W	
Puccinia hordei (barley leaf rust)	5	6	15	Teng and Close 1978, Fig. 2C
P. coronata (oat crown rust)	8	13	25	Heagle and Moore 1970, Fig. 3C, cv. 'Coachman'
P. recondita (wheat leaf rust)	6	12	35	Mehta and Zadoks 1977, Fig. 3, treatment D
P. graminis (wheat stem rust)	8	15	32	Mortensen and Green 1978, Fig. 1C, cv. 'Klein Titan'
Pyricularia oryzae (rice blast)	5	8	25	Kato and Kozaka 1974, Fig. 2, 25 °C
Pseudoperonospora cubensis (cucumber downy mildew)	3	6	11	Cohen and Rotem 1971, Fig. 4, 20–25 °C
Sphaerotheca fuliginea (cucumber powdery mildew)	10	18	29	Bashi and Aust 1980, Fig. 2A, 25 °C

that the values of T and W would increase by the same amount as the increase in A (i.e. the values of $T-A$ and $W-A$ were assumed to remain constant as A increased). Thus, the triangular sporulation function, as represented in Fig. 1 was assumed to retain the same shape as it shifted to the right with increases in A, the length of the latent period.

A large number of solutions to Equation 1 were generated for series of values of A and r to produce the families of curves shown in Figs. 4–10. For each disease the situation of a highly susceptible cultivar in an environment very conducive for disease development is represented in the lower right corner of the graphs. For example, with barley leaf rust, we calculated that with a latent period of 5 days a value of S of 46.6 would be required to produce an epidemic with an exponential rate of increase of 0.50 day^{-1}. At that value of S, an increase in the length of the latent period from $A=5$ to $A=8$ would reduce r_1 from 0.50 day^{-1} to approximately 0.36 day^{-1}. To achieve the same reduction in r_1 by reducing S rather than A would require a reduction in S from 46.6 to approximately 17.3. Therefore, under the conditions in which r_1 for leaf rust increase on a susceptible barley cultivar would be 0.50 day^{-1}, resistance that provided a 3-day increase in latent period would be as effective as resistance that resulted in a 63% reduction in either sporulation or infection efficiency.

Other starting points for this analysis could have been chosen. For instance, if the most rapid rate of increase of barley leaf rust that could be expected were $r_1=0.38$, the value of S at $A=5$ days would be 20. At that level of S a 3-day increase in A would reduce r_1 from 0.38 day^{-1} to approximately 0.27^{-1}. A 55% reduction in S would be required to produce the same reduction in r_1 if A remained constant at 5 days.

In this context, it must be emphasized that we have defined latent period as the time from inoculation to the appearance of the first sporulating lesion. In most

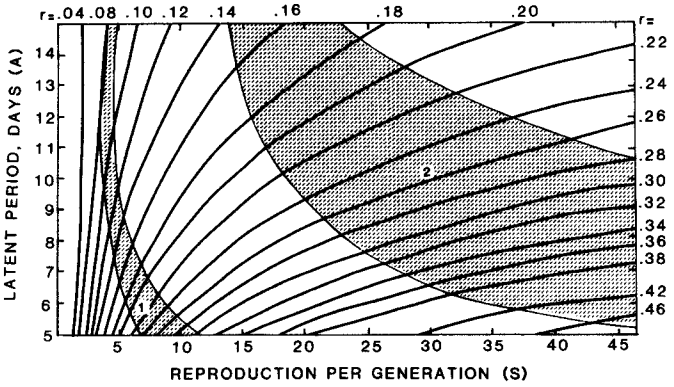

Fig. 4. Barley leaf rust (*Puccinia hordei*)-Relationships of combinations of values of A (latent period) and S (reproduction per generation) to r_1 (rate of disease increase). Values of A, S, and r_1 were generated from Eq. 1 and the relationships among key parameters A, T, and W in the *P. hordei* sporulation curve (Table 1). For resistant cultivars with $A > 5$, the values of T and W are assumed to increase by the same amount as the increase in A. Shaded area No. 1 represents the combinations of parameter values at which a proportionate increase in A will reduce r_1 to the same extent as an equivalent proportionate decrease in S. Shaded area No. 2 represents the combinations of parameter values at which twice as large a proportionate decrease in S is required to match the effect on r_1 resulting from an incremental increase in A. See text for details

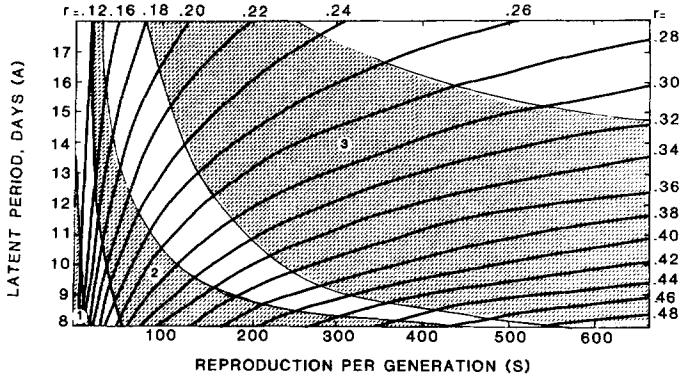

Fig. 5. Oat crown rust (*Puccinia coronata*)-Relationships of combinations of values of A (latent period) and S (reproduction per generation) to r_1 (rate of disease increase). See legend for Fig. 4 and text for details

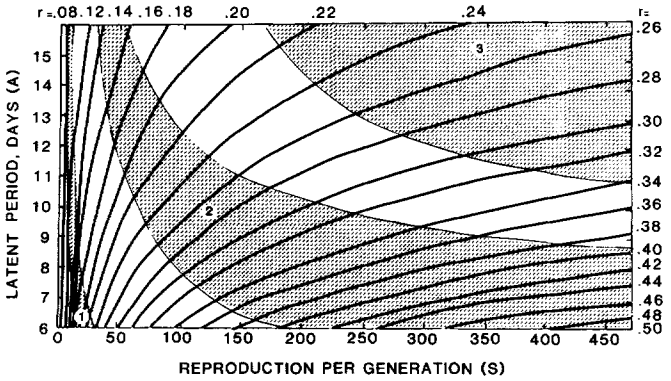

Fig. 6. Wheat leaf rust (*Puccinia recondita*)-Relationships of combinations of values of A (latent period) and S (reproduction per generation) to r_1 (rate of disease increase). See legend for Fig. 4 and text for details

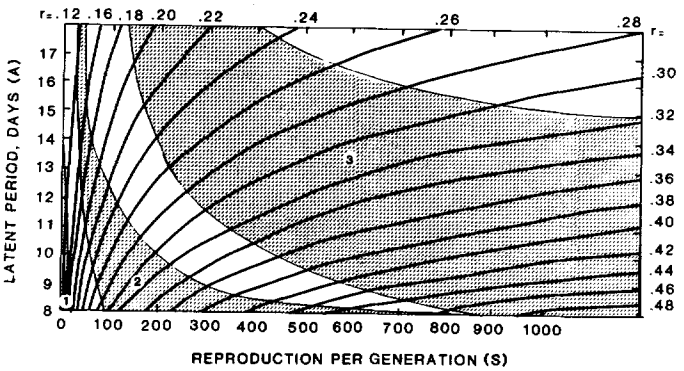

Fig. 7. Wheat stem rust (*Puccinia graminis* f. sp. *tritici*)-Relationship of combinations of values of A (latent period) and S (reproduction per generation) to r_1 (rate of disease increase). See legend for Fig. 4 and text for details

studies in which latent period is evaluated, it is measured as the time from inoculation until the day on which 50% of the eventual total number of lesions began sporulation. Therefore, our values of A are somewhat shorter than the latent period measurements that have been published for the same diseases.

From the slopes of the curves in Figs. 4–10, it can be seen that increases in latent period are relatively more effective in reducing r_1 in situations in which r_1 and S are high than in situations with low values of r_1 and S. Thus, a plant breeder working with a highly susceptible crop in an environment that is highly conducive to disease may choose to concentrate initially on selection for resistance that will increase the latent period of the pathogen. Under conditions in which r_1 is lower, changes in the value of S will be relatively more effective in reducing r_1 still further than similar changes in S would have been under conditions of high r_1. Thus, the relative effectiveness of changes in different components of resistance depends upon the interactions among A, S, and r_1.

To help illustrate the relative effects of changes in A and S in reducing r_1, we calculated values for which a 1-day or a 2-day increase in A would produce a reduction in r_1 identical to that caused by an equivalent percentage decrease in S. For example, in Fig. 4 a 20% increase of A from 5 to 6 days at $S=6.7$ would result in a reduction of r_1 from 0.22 to 0.20. The same reduction in r_1 at $A=5$ days could also be obtained by a 20% decrease in S from 6.7 to 5.4. The curve along the left side of the shaded area labeled No. 1 in Fig. 4 was generated in this way. This relationship holds for any 1-day change along the line, e.g. at $S=5.4$ a 16.7% increase in A from 6 to 7 days would reduce r_1 the same amount as a 16.7% decrease in S from 5.4 to 4.5 at A=6 days.

The curve on the right of the shaded area labeled No. 1 in Fig. 4 was generated from calculations of equivalent changes in r_1 caused by 2-day increases in A or equivalent percentage decreases in S. For example, a 40% increase in A from 5 to 7 days at S=11.8 would result in a reduction of r_1 from 0.32 to 0.24. The same reduction in r_1 could be obtained by a 40% decrease in S from 11.8 to 7.1 at $A=5$ days. The curves for 1-day and 2-day increases in A, and equivalent percentage decreases in S do not follow the same path, because the effect of reducing S becomes greater relative to that of increasing A as the magnitude of the changes increases. For example, a 100% reduction in S would prevent disease increase, whereas a 100% increase in A would merely delay it by doubling the latent period.

For changes in A greater than 2-day increments the corresponding equivalent change in S can be calculated in a step-wise fashion. For instance, a 4-day increase of A from 5 to 9 days at $S=11.8$ would produce a reduc-

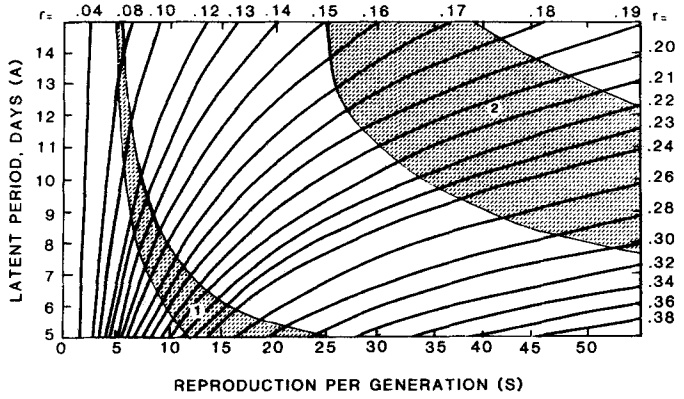

Fig. 8. Rice blast (*Pyricularia oryzae*)-Relationships of combinations of values of A (latent period) and S (reproduction per generation) to r_l (rate of disease increase). See legend for Fig. 4 and text for details

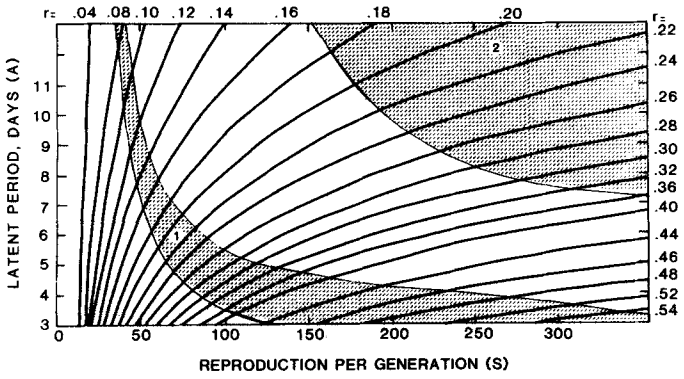

Fig. 9. Cucumber downy mildew (*Pseudoperonospora cubensis*)-Relationships of combinations of values of A (latent period) and S (reproduction per generation) to r_l (rate of disease increase). See legend for Fig. 4 and text for details

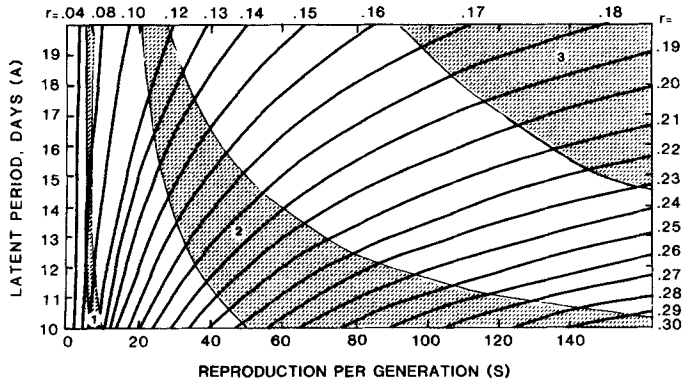

Fig. 10. Cucumber powdery mildew (*Sphaerotheca fuliginea*)-Relationships of combinations of values of A (latent period) and S (reproduction per generation) to r_l (rate of disease increase). See legend for Fig. 4 and text for details

tion in r_l equivalent to that caused by a 40% reduction in S (40% increase in A from 5 to 7 days) from 11.8 to 7.1 followed by a 29% reduction in S (29% increase in A from 7 to 9) from 7.1 to 5.0.

Shaded area No. 1 for each of the diseases illustrated in Figs. 4–10 shows the range of combinations of values of A, S, and r_l at which proportionately equal changes in latent period, A, or pathogen reproduction per generation, S, will cause approximately equal reductions in r_l, the exponential rate of pathogen increase per day. To the left of shaded area No. 1, the reduction in r_l caused by a given increase in A will be less than the reduction in r_l caused by a decrease of equal proportion in S. To the right of shaded area No. 1, the effect of increasing A will be greater. As with barley leaf rust the shaded areas have all been calculated for changes in A of 1- or 2-day increments.

Shaded area No. 2 in Figs. 4–10 represents the combinations of A, S, and r_l at which a given reduction in r_l requires a decrease in S that is twice as great as the proportionate increase in A that would produce the same reduction in r_l. For example, with barley leaf rust (Fig. 4), at $A=10$ and $S=17.5$ a 10% increase in A will reduce r_l as much as a 20% decrease in S would. As with shaded area No. 1, the boundary along the lower left side of the shaded area was determined by calculating changes equivalent to 1-day increase in A or two times the proportionately equivalent changes in S. The boundary along the upper right side of the shaded area was determined for 2-day changes in A and two times the corresponding proportionate changes in S.

In Figs. 5, 6, 7, and 10, there is a shaded area No. 3 as well as shaded areas Nos. 1 and 2. Shaded area No. 3 represents the combinations of values of A, S, and r_l at which the reduction in r_l that would result from a single proportionate incremental increase in A, would require three increments of the same proportionate decrease in S to produce the same effect. Thus, for the values in shaded area No. 1, a 10% decrease in S would be approximately as effective as a 10% increase in A in reducing r_l. At the values represented in shaded area No. 2, a 10% decrease in S would be only about half as effective as a 10% increase in A, and in shaded area No. 3 it would be only about a third as effective. For barley leaf rust (Fig. 4), rice blast (Fig. 8), and cucumber downy mildew (Fig. 9), the combinations of parameters representative of shaded area No. 3 fell outside the region of realistic values included within the graphs.

From Figs. 4–10 one can see that with diseases that have short latent periods on susceptible cultivars (i.e. rice blast with $A=5$ and cucumber downy mildew with $A=3$), reducing S may be nearly as effective as increasing A to reduce r_l. For diseases with longer latent periods (i.e. oat crown rust, $A=8$; wheat stem

rust, $A = 8$; and cucumber powdery mildew, $A = 10$), increasing the latent period will usually be much more effective than reducing pathogen reproduction per generation to reduce r_1.

Although it is convenient to consider the relative influences of A and S separately in determining their effects on r_1, increases in latent period are usually not independent of decreases in pathogen sporulation or infection efficiency. In most quantitatively resistant cultivars, these three traits appear to be highly correlated (Parlevliet 1979). Nevertheless, breeding lines may not have all components of resistance in exactly the same degree. Charts like those in Figs. 4–10 can help the breeder to decide which breeding lines are likely to produce the greatest reduction in r_1. As in the previous analyses, the starting point is the level of r_1 for a susceptible check cultivar in a year with weather suitable for rapid build-up of the disease.

Simplified regression models for estimating epidemiological effects of quantitative resistance

The relationships illustrated in Figs. 4–10 each apply to a single host-pathogen combination, but they might be applied successfully to other diseases for which triangular sporulation functions with similar values of A, T, and W were determined. In an attempt to develop a simpler and more general method of estimating values of r_1 from values of A and S, we tested several regression models. Data for the regressions were taken from the calculated values in Figs. 4–10. For each integer value of A, two values of S (and the corresponding values of r_1) were selected at random from the left half of the graph and two were selected at random from the right half. In the regression models, r_1 was the dependent variable and A, S, $\log_e A$, $\log_e S$, A^2, S^2, $(\log_e A)^2$, and $(\log_e S)^2$ were tested as independent variables. The Statistical Analysis System PROC R SQUARE program for all possible regressions (SAS Institute, Inc 1982) was used to calculate R^2 values for each combination of independent variables. The best two-parameter model had $\log_e A$ and $\log_e S$ as independent variables. Using more than two independent variables added relatively little to the predictive power of the regressions. Therefore, the regression equation $r_1 = b_0 + b_1 \log_e A + b_2 \log_e S$ was chosen as a convenient model for estimating epidemiological effects of changes in latent period and pathogen reproduction per generation.

Regression coefficients and R^2 values for equations for the seven diseases are shown in Table 2. In general, the R^2 values were slightly higher for diseases with long latent periods ($A = 8$–10 days) than for those with short latent periods ($A = 3$–6 days). The regression coefficient for $\log_e S$ (i.e. b_2) was inversely correlated with the value of A for the seven diseases, indicating that changes in S have a relatively greater effect on r_1 for diseases with short latent periods than for those with long latent periods. As a linear approximation from the data for A in Table 1 and b_2 in Table 2, $b_2 \simeq 0.11 - 0.0066\,A$. Values of b_0, the intercept, or of b_1, the regression coefficient for $\log_e A$, were not well correlated with values of A for the seven diseases. For a disease in which A but not T or W is known, a reasonable approximation of the effects of changes in components of resistance might be obtained from an equation based on mean values of b_0 and b_1 from Table 2 and a value of b_2 determined from the regression $b_2 = 0.11 - 0.0066\,A$ as in equation 2.

Table 2. Regression coefficients for the relationships of exponential rate of disease increase (r_1) to latent period (A) and pathogen reproduction per generation (S) for seven diseases expressed in the form, $r_1 = b_0 + b_1 \log_e A + b_2 \log_e S$

Disease	b_0	b_1	b_2	R^2
Barley leaf rust	0.33	−0.15	0.082	0.95
Oat crown rust	0.40	−0.16	0.056	0.97
Wheat leaf rust	0.29	−0.13	0.060	0.94
Wheat stem rust	0.36	−0.15	0.053	0.96
Rice blast	0.22	−0.11	0.072	0.95
Cucumber downy mildew	0.28	−0.14	0.093	0.94
Cucumber powdery mildew	0.43	−0.16	0.048	0.98
Mean	0.33	−0.14		

$$r_1 = 0.33 - 0.14 \log_e A + (0.11 - 0.0066\,A) \log_e S \qquad \text{(Eq. 2)}$$

Examination of the residuals for the fits of equations in Table 2 to the data from Figs. 4–10 revealed that the equations most accurately predicted values of r_1 in Figs. 4–10 that were intermediate. Values of r_1 near 0 or near the highest values in the figures were predicted least accurately by the regression equations. Although the regression equations are probably less accurate than Figs. 4–10 for evaluating the usefulness of quantitative resistance in different cultivars, they could be much more rapid and convenient to use for comparing large numbers of cultivars.

A general equation for comparing effects of latent period and pathogen reproduction per generation

Caswell and Hastings (1980) derived the relationship $k_q \simeq (\lambda / P^*)^q$ as an approximation of the relative importance of developmental time (= latent period) and net fecundity (= reproduction per generation) in determining population growth rate. In Caswell and

Hasting's approximation k_q is the factor by which fecundity must be increased to duplicate the effect of a q-unit decrease in developmental time, $\lambda = e^{r_l}$ where r_l is the population growth rate, and P^* is the average probability of survival of reproducing organisms from one age class to the next. Caswell and Hastings developed their approximation to be independent of the form of the fecundity function, which is equivalent to the sporulation function described in our adaptation of Lewontin's model.

Caswell and Hastings' approximation could be useful in evaluating quantitative resistance to diseases in which the daily sporulation patterns are not known or do not fit the triangular pattern required in Lewontin's model. For this purpose, Caswell and Hastings' approximation can be modified by replacing λ with e^{r_l}, setting $P^* = 1$, and by redefining k_q to be the factor by which pathogen reproduction per generation must be multiplied to duplicate the effect of a q-unit decrease in pathogen latent period. The modified equation is:

$$k_q \simeq e^{qr_l} \qquad \text{(Eq. 3)}$$

Equation 3 says that for a given value of r_l, a decrease in latent period of q days will change r_l to approximately the same extent as would be caused if pathogen reproduction per generation were multiplied by the factor k_q. Of course, we would be interested in resistance that increases rather than decreases latent period, so for resistant cultivars the units of q will be negative days. Correspondingly, the factor k_q will be less than 1 for resistant cultivars. As an example, for barley leaf rust at $r_l = 0.47$ and $S = 0.40$ (Fig. 4), $A = 5$. According to Eq. 3, increasing the latent period from 5 days to 6 days should be equivalent to reducing pathogen reproduction per generation by a factor of 0.63. According to Fig. 4, the 1-day increase in A should reduce r_l from 0.47 to 0.42, and reducing S to 63% of 0.40 (i.e. to 25) should reduce r_l to 0.40. Thus the change in S calculated from Eq. 3 would result in a value of r_l approximately 5% lower than that resulting from the 1-day increase in A. In this case, Eq. 3 slightly overestimated the magnitude of the reduction in S required to match a 1-day increase in A.

The above described procedure was used to determine how well predictions of changes in r_l based on Eq. 3 matched those determined from Figs. 4–10. Eq. 3 worked well for 1-day increases in A. In tests of five or more combinations of values of A and S from each of the seven diseases, the value of r_l resulting from the reduction in S calculated from Eq. 3 never differed by more than 9% from the value of r_l indicated for a 1-day increase in A.

Eq. 3 did not work as well for 3-day increases in A. Approximately five combinations of A and S from each disease were tested and differences as great as 38% were found between values of r_l resulting from the calculated reduction in S and those indicated for 3-day increases in A. The greatest errors, both in number and magnitude, occurred with diseases with short latent periods (i.e. cucumber downy mildew, rice blast, and barley leaf rust). For cucumber powdery mildew, wheat leaf rust, and oat crown rust, none of the errors exceeded 9%. Even with the three diseases with the shortest latent periods, Eq. 3 yielded accurate predictions for reductions in S needed to balance a 3-day increase in A if the increase was from a value of A 5 days greater than the minimum value for the disease. Thus, all predictions from Eq. 3 starting from a base latent period of 8 days or more were within 10% of those indicated for 3-day increases in A.

Overall, the average differences in values of r_l resulting from the calculated reductions in S based on Eq. 3 and those indicated for 1- or 3-day increases in A, were 1.9, 2.8, 2.8, 3.9, 6.1, 6.4, and 9.8% for cucumber powdery mildew, wheat stem rust, oat crown rust, wheat leaf rust, barley leaf rust, rice blast, and cucumber downy mildew, respectively. In 96% of the cases in which differences occurred, Eq. 3 overestimated the magnitude of the reduction in S needed to match the effect of a 1- or 3-day increase in A.

Test of the Lewontin model

In choosing values for the parameters A, T, and W for each disease that we analyzed, we relied on a single sporulation curve that had been published for that disease. Since sporulation curves may differ among susceptible cultivars and among pathogen isolates, the general utility of the Lewontin model for predicting epidemiologcal effects of resistance could be questioned. We believe, however, that several aspects of our analyses provide confidence in their usefulness. First, three of the four families of curves for cereal rust diseases have very similar patterns even though they were based on different sporulation curves. The exception, barley leaf rust, can be explained by its shorter latent period and, especially, by its early peak of sporulation. In these characteristics it resembles cucumber downy mildew and rice blast, which it also resembled in its pattern of the family of curves. Second, the general equation developed by Caswell and Hastings (1980), which does not depend upon a triangular reproductivity function, yielded values generally consistent with the predicted effects from the Lewontin model.

The most convincing test of the Lewontin model would be to apply it to a disease for which reliable values of latent period, infection efficiency, sporulation, and rates of disease increase are available. Table 3

shows the comparisons of observed and predicted values of r_1 for leaf rust on eight barley cultivars. The r_1 values were calculated from data of Neervoort and Parlevliet (1978) using Vanderplank's (1963) Equation 3.3. Relative values for latent period, infection efficiency, and sporulation on these cultivars were published by Parlevliet and van Ommeren (1975). We assumed a value of A of 5 for the most susceptible cultivar, 'L94', and used Fig. 4 to determine the appropriate value of S for $r_1 = 0.29$. The predicted values of r_1 for the other cultivars were determined by calculating their A and S values from their relative values in Parlevliet and van Ommeren (1975).

In this comparison, the Lewontin model appears to give a reasonably accurate prediction for 'L98' but overestimates the effect of resistance in the other cultivars. This could be expected. Neervoort and Parlevliet (1978) indicated that "the cultivars 'L94' and 'L98' are fairly primitive cultivars as compared with the others, which are of a modern, West European type". 'L94' and 'L98' produce fewer tillers and invariably have a much lower canopy density. "Such open crops may reduce the rate of epidemic development because the spores produced may escape in larger numbers than is the case with the denser and shorter crops produced by the modern cultivars" (Neervoort and Parlevliet 1978).

When we removed the two primitive cultivars 'L94' and 'L98', and based the comparisons on the r_1 value observed for 'Mamie', the predicted values for the other five cultivars were much closer to the observed values. In this case we used the A value of 6 calculated from the relative value of 121% of the latent period of 'L94' (Neervoort and Parlevliet 1978), which we assumed to be 5. The results would have been essentially the same if we had set the latent period for 'L94' at 6 days. The S value of 12 was determined from Fig. 4 for $A = 6$ and $r_1 = 0.27$. The close fit of predicted to observed values of r_1, especially for the two most resistant cultivars, 'Julia' and 'Vada', gives us confidence in the utility of the Lewontin model.

We also tested the simplified regression models, which are based on data taken from Fig. 4. In these comparisons we used the S value of 12 for 'Mamie' obtained from Table 3 as the base for calculations of S for the other five cultivars. We eliminated 'L94' and 'L98' from the analysis for the reasons described above. Both the regression equation developed specifically for barley leaf rust and the general regression equation yielded predicted r_1 values that were close to the observed values (Table 4).

Table 3. Comparison of observed rates of disease increase, r_1, for leaf rust on barley cultivars and rate of increase predicted from data on latent periods and reproduction per generation using the relationships in Fig. 4

Cultivar	A [a]	S [b]	Predicted r_1	Observed r_1 [c]
'L94'	5.0	10.0 [b]	0.29 [b]	0.29
'L98'	5.6	9.7	0.26	0.22
'Mamie'	6.0	5.1 (12)	0.17 (0.27)	0.27
'Sultan'	6.3	4.3 (10)	0.14 (0.24)	0.27
'Zephyr'	6.8	4.5 (11)	0.14 (0.21)	0.28
'Volla'	5.9	5.7 (13)	0.18 (0.29)	0.22
'Julia'	7.7	2.5 (5)	0.08 (0.15)	0.14
'Vada'	9.3	1.4 (3)	0.02 (0.08)	0.06

[a] Latent periods (time to first sporulating pustule) are based on relative data from Parlevliet and van Ommeren (1975) assuming that A for the most susceptible cultivar, 'L94', was 5 days

[b] Values of S were calculated from relative data from Parlevliet and van Ommeren (1975) using the value of 10 for cv. 'L94' as the base. The value for 'L94' was determined from Fig. 4 for $A = 5.0$ and $r_1 = 0.29$. Values in parentheses for S and r_1 were calculated using the value 12 for cv. 'Mamie' as the base

[c] Rates of disease increase calculated from data of Neervoort and Parlevliet (1978) for small isolated field plots for June 14 to July 5, 1973 (Vanderplank 1963, eq. 3.3)

Table 4. Comparison of observed rates of disease increase, r_1, for leaf rust on barley cultivars and rates of increase predicted from data on latent periods and reproduction per generation using regression coefficients from Table 2

Cultivar	A [a]	S [b]	Predicted r_1 (specific) [c]	Predicted r_1 (general) [d]	Observed r_1 [e]
'Mamie'	6.0	12	0.26	0.25	0.27
'Sultan'	6.3	10	0.24	0.23	0.27
'Zephyr'	6.8	11	0.24	0.23	0.28
'Volla'	5.9	13	0.27	0.26	0.22
'Julia'	7.7	5	0.16	0.11	0.14
'Vada'	9.3	3	0.09	0.04	0.06

[a] Latent periods based on data from Parlevliet and van Ommeren (1975); see Table 3

[b] Values of S based on data from Parlevliet and van Ommeren (1975). Reproduction per generation on 'Mamie' was chosen as the base for calculations; see Table 3

[c] From the equation $r_1 = 0.33 - 0.15 \log_e A + 0.082 \log_e S$ for barley leaf rust; see Table 2

[d] From the general regression equation $r_1 = 0.33 - 0.14 \log_e A + (0.11 - 0.0066A) \log_e S$; see Table 2 and text

[e] Rates of disease increase calculated from data of Neervoort and Parlevliet (1978) for small isolated field plots for June 14 to July 5, 1975 (Vanderplank 1963, eq. 3.3)

Discussion

We have shown that the triangular pattern of reproduction upon which the Lewontin model depends is

characteristic of the sporulation curves of several important plant pathogens. Data less extensive than those that we cited indicate that sporulation curves for other plant pathogenic fungi also fit the triangular pattern. Aust (1973) presented such evidence for powdery mildew of barley, and Levy and Cohen (1980) showed that sporulation of *Exserohilum turcicum* (northern leaf blight of maize) follows a triangular pattern.

The Lewontin model will not be universally applicable to all plant pathogenic fungi. Sporulation patterns for common bean rust caused by *Uromyces phaseoli* var. 'typica' (Imhoff et al. 1982) and soybean rust caused by *Phakopsora pachyrhizi* (Melching et al. 1979) tend not to have distinct, single peaks of sporulation. For these diseases, Vanderplank's (1963) assumption of uniform sporulation per day during each lesion's infectious period seems reasonable. Therefore, Vanderplank's (1963) equation for r_1 corrected for latent period and removals could be used to generate families of curves similar to those in Figs. 4–10 based on different combinations of latent period and reproduction per generation. Such curves could be used in the same way that we have used those in Figs. 4–10. Another approach would be to use life tables as proposed by Zadoks and Schein (1979) to generate similar families of curves.

We have demonstrated that the Lewontin model can be used to accurately predict epidemiological effects of different combinations of components of resistance of barley to leaf rust. The accuracy of the predictions, however, depends upon having reliable data for latent period, infection efficiency, and total sporulation of resistant cultivars relative to that of the standard susceptible cultivar.

Data on latent period and infection efficiency are relatively easy to obtain in controlled experiments, but some precautions should be observed. First, adult plants should be used because they often express much higher levels of quantitative resistance than do seedlings. For instance, Parlevliet (1976) showed that latent periods for barley leaf rust on flag leaves of resistant barley cultivars differed from latent periods on flag leaves of susceptible cultivars by a much greater degree than did latent periods on primary leaves of resistant and susceptible seedlings. Second, plants grown in the greenhouse may not respond in the same way as plants grown in the field. Extended latent periods for crown rust on resistant oat cultivars can be demonstrated for plants grown outside and transferred to the greenhouse for inoculation, but if the same cultivars are grown in the greenhouse, the latent periods may be similar to those of susceptible cultivars (H. H. Luke, personal communication). Third, latent periods can be affected by the density of infections on plants; higher densities can shorten the latent period (Johnson and Taylor 1976). Therefore, latent periods should be compared on cultivars with relatively similar numbers of infections. Since the Lewontin model applies to the exponential increase phase of epidemics, the disease intensity should be relatively low for comparisons of latent periods.

Measurements of spore production are laborious and are often not attempted for comparisons of relative resistance of cultivars. Several methods that can be used have been summarized by Johnson and Taylor (1976). If determinations of relative spore production on breeding lines is judged to be too time consuming for practical use, predictions of r_1 values could be made on the basis of data for latent period and infection efficiency. Such predictions would usually underestimate the effectiveness of quantitative resistance, because reduced sporulation tends to be associated with the other components of resistance (Parlevliet 1979). As with latent period, the measures of sporulation per infection may be affected by the numbers of infections per plant, with sporulation being reduced at high disease intensity (Parlevliet 1979). The effects of competition among infections in reducing sporulation per infection may also be seen at low infection intensities for some diseases (Leonard 1969; Imhoff et al. 1982).

A final precaution must be mentioned. As seen with barley cultivars 'L94' and 'L98' and barley leaf rust, rates of disease increase may be affected by the canopy structure of the crop as well as by the components of resistance that can be measured in controlled experiments with individual plants. Therefore, the predictions based on the Lewontin model are likely to be less accurate when the plant growth habits are variable among cultivars than when they are uniform.

We have described the use of the Lewontin model for comparisons of the effectiveness of quantitative resistance among cultivars, but the model can also be applied to other situations. For instance, in mixtures of resistant and susceptible plants as in multiline cultivars, the rate of disease increase tends to be proportional to the $\log_e$ of the proportion of susceptible plants in the mixture (Leonard 1969). The degree to which the mixture reduces r_1 can be predicted if the effective generation time of the pathogen is known. It could also be predicted from the Lewontin model by assuming that the effect of the host mixture would be equivalent to multiplying S by the proportion of susceptible plants. This assumption implies that spores produced on susceptible plants within the mixture are randomly dispersed among resistant and susceptible plants. For rusts of small grains and for rice blast, this assumption appears to be justified (Leonard and Czochor 1980).

Another application of the Lewontin model could be in interpreting the epidemiological effects of

protectant fungicides. The efficacy of the fungicide in a monocyclic inoculation test can be used as a measure of the degree to which S would be reduced by the fungicide. We may assume that the main effect of the protectant fungicides is to reduce infection efficiency and that the rate of lesion expansion and sporulation will be much less affected (Bruck et al. 1981). Using the Lewontin model one could calculate expected equivalency of fungicide efficacy and levels of quantitative resistance in known cultivars in reducing rates of disease increase (Fry 1975, 1978).

The Lewontin model and the Caswell-Hastings equation apply to exponential increase of populations. With severe epidemics of plant diseases the pathogen populations increase to levels well beyond the threshold at which available, uncolonized host tissue becomes limiting. At these levels the use of the Lewontin model or the Caswell-Hastings equation may not be valid. On the other hand, the analyses will work for the early stages of epidemics and can be used to predict when threshold levels of disease will occur with different cultivars or control measures. Furthermore, when the control measures are highly effective, they may delay build-up of disease so that it stays in the exponential phase throughout most of the growing season.

The studies of Zadoks (1971) and Parlevliet and van Ommeren (1975) showed that changes in latent period produced greater relative changes in r_1 than did changes of similar magnitude in other components of resistance. The Lewontin model and the Caswell-Hastings equation reaffirm this conclusion but also demonstrate that the relative effects of changes in latent period depend upon the starting value of r_1 and also upon the key parameters of the sporulation curve. Proportionate increases in latent period will have their greatest effect relative to similar proportionate decreases in reproduction per generation when r_1 is high and the latent period is long. For diseases with short latent period, it may be more effective to reduce reproduction per generation rather than to increase the latent period. Of course, plant breeders must work with the range of variation available to them. If a breeder finds very little variation in latent period for the pathogen among his breeding lines but large ranges of variation in infection efficiency or sporulation, he will be wise to select for reduced values of S rather than spend too much effort trying to increase the latent period. Our analyses should help plant breeders adjust their selection strategies to take best advantage of the components of resistance available to them. This should be true not only for the diseases that we have described, but also for many other, less studied diseases. The examples presented here can be used to predict the behavior of other diseases with similar characteristics.

Acknowledgements. The contributions of Margaret Stefano, Martin W. Imhoff, and Clementine Zimmerman to various stages of this study are gratefully acknowledged.

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