Letter to the Editor

Multilines and Super-Races—a Reply

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The tendency among the supporters and critics of multilines and cultivar mixtures has been to use the results of mathematical models of pathogen response without appreciating the assumptions of the models. The recent letter by J. V. Groth (1) on the evolution of so-called "super-races" will promote renewed interest in this field. However his model has a number of assumptions which limit its usefulness; the response of the pathogen is not as easily predictable as Groth's model implies. We offer here a simple model in which Groth's model is included as a special case, and which leads to slightly different conclusions. Our model is not intended to be comprehensive but only to demonstrate the subtlety of the operating evolutionary forces. The mathematical model in this paper is part of an extensive analysis of pathogen response to cultivar mixtures. Furthermore, we feel that there are a number of points in Groth's letter which require clarification:

1) The assumption of complete lethality of nonvirulent genotypes on resistant hosts is a special case (assumption iii). In the field, and thus in a general model, scope must be allowed for those pathogen genotypes which commonly occur and which are not lethal on noncorresponding hosts.

2) In relation to the first variation of the model: the model relies on the fitnesses of the virulence alleles acting additively (assumption iv). Fitnesses are, by definition, probabilities of survival and reproduction. If the virulence genes act independently, then the only way in which the fitness of a "super-race" can be defined is by multiplication of the fitnesses of each of the virulence genes. If the selection coefficients (s in Groth's terms) are very small, then 1.0 minus the sum of the selection coefficients is approximately the same as the product of the fitnesses, viz:

$$(1-s)^n = 1 - ns + \frac{n(n-1)s^2}{2!} - \frac{n(n-1)(n-2)s^3}{3!} + \ldots\ldots$$

If s is very small, we may neglect higher terms of s, and obtain:

$$(1-s)^n \cong 1 - ns$$

If s is greater than 0.1 this approximation is no longer reasonable; even when $s = 0.1$ and $n = 2$ the approximation is 1.23% in error. Hence Groth's "simple" variation is a special case of the general multiplicative model, for very small values of s. The multiplicative model offers the simplest possible genetic model in the absence of any real data on the way virulence genes affect fitness.

3) It is obvious that the values of the selection coefficient (s) will be different for each pathogen on each host and at different times during epidemic development; eg, through the effects of adult plant resistance. As a first approximation, therefore, in the absence of relevant data, and to simplify the mathematics, s can be assumed to be the same on all hosts and constant during the season. Even if s is constant, the *mean fitness* of each pathogen genotype will vary during the season, where *mean fitness* is the fitness of each pathogen genotype averaged over all components and weighted by its frequency on each component at a given point in time. Groth (1) implies that his value for the mean fitness (R_m) will be constant during disease development since he states on page 937: "Complex races which reproduce better ($R > 1$) than the original simple races will be selected for, and will eventually replace the simple races."

This will only be true if the asexual generations of the pathogen are discrete; ie, those lesions producing spores die before their offspring reinfect the host plants; this is obviously unrealistic. Consider, for example, a two-component multiline ($n = 2$) with each component at 50%. By using Groth's assumptions, the following fitness matrix is obtained:

Host	Pathogen			
	V_AV_B	v_AV_B	V_Av_B	v_Av_B
R_A	0	1.0	0	1.0−s
R_B	0	0	1.0	1.0−s
Mean fitness	0	0.5	0.5	1.0−s

[in which the mean fitness of the simple races is

$$R_1 = 1.\frac{1}{n}$$

and the mean fitness of the complex race is

$$R_m = \frac{m}{n}(1-s),$$

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in which m is the number of virulence genes carried].

Groth then uses these mean fitness values on the assumption that they will apply in succeeding generations. However, those lesions already established at the end of the first asexual generation will have fitnesses during the second asexual generation of 1.0 and $(1.0 - s)$ for the simple and complex races, respectively. The spores, having been released, reinfect the different hosts at random and the new infections will have different mean fitnesses from those of their parents. The net mean fitness of each pathogen genotype after the first asexual generation must take into account the fact that the mature lesions and new infections will be subject to different selection pressures, since spores are dispersed and mature lesions are not. The relative change in fitness of simple and complex races thus depends on the rate at which the spores produced by the simple race fail to find a susceptible host; this in turn depends on the rate of reproduction. Since mature lesions do not die immediately after releasing their first spores, the nonrandom distribution of pathogen genotypes on hosts must be allowed for and, hence, the time from initial infection becomes an important factor. Groth's model assumes complete redistribution of pathogen genotypes over all hosts: this is only true at the start of the first generation or if the generations are discrete. Only under these conditions will the mean fitnesses remain constant.

Although many pathogens produce large quantities of spores daily, the rate of increase of infection is often low, due to the large amount of wastage. Therefore it is necessary to introduce a constant, α, which measures the realized growth rate and not the absolute reproductive capability of the pathogen. A full list of the assumptions underlying the model on which this paper is based can be found in Barrett (2).

Thus, if the reproduction rate for all races is $(1 + \alpha)$, where 1.0 represents the parent lesion, and if $\alpha = 1.0$, the population doubles in each generation, so that over, say 20 generations the population increases by $(1.0 + 1.0)^{20}$; ie, approximately one millionfold. If all of the spores produced are released into a general pool for redistribution over both components of the simple multiline to produce new infections, then the number of new infections, expressed as a proportion of all individuals is $\alpha/(1.0 + \alpha)$. The mean fitness of the infections of the simple races in the first generation is $1/n$; in the second asexual generation it will be:

$$R_1 = \frac{1.1}{1 + \alpha} + \frac{\alpha}{1 + \alpha} \cdot \frac{1}{2} = \frac{1 + .5\alpha}{1 + \alpha}$$

The mean fitness of the new infections of the complex race over the period Δt (the period of an asexual generation), will be $(1 - s)$ as the multiline, under Groth's assumptions, offers them a uniform environment. The magnitude of s is dependent on the length of the time interval over which it is measured; eg, if $s = 0.1$ over one time interval, then over two time intervals its value becomes $s' = 1 - (1 - s)^2 = 0.19$.

After the second generation the mean fitness of the simple races remains constant, since the reproduction rate and spore dispersal rates are constant. If α is very large the mean fitness of the simple races tends to $1/n$; ie, the parent lesions make up an insignificant proportion of the population size in the next "asexual generation"; this is equivalent to the parents dying and is identical to Groth's (1) model. Further, if the simple races can grow at all on noncorresponding hosts, their mean fitnesses will change continuously; given sufficient time they will converge on an equilibrium value (Barrett, Wolfe, and O'Donald, *unpublished*).

Taking Groth's multiplicative model and allowing for the dispersal of spores alone in each interval of time, the expression for the mean fitness of complex races relative to that of the simple races now becomes:

$$R_1 = \frac{n + \alpha}{n(1 + \alpha)}$$

$$R_m = \frac{1(1 - s)^{m-1}}{1 + \alpha} + \frac{(m/n)\,\alpha\,(1 - s)^{m-1}}{1 + \alpha}$$

$$= \frac{(1 - s)^{m-1}(n + m\alpha)}{n(1 + \alpha)}$$

$$R = \frac{R_m}{R_1}$$

$$R = \frac{(1 - s)^{m-1}(n + \alpha m)}{n + \alpha}$$

where α is the proportion by which the population grows in each time interval Δt.

s is the selection against virulence genes on non-compatible hosts when present in a multiple virulent pathogen genotype.

m is the number of virulence genes in the pathogen genotype.

n is the number of components in the multiline; all components are assumed to carry a different resistance gene and to be present in the multiline in the proportion $1/n$.

To obtain turning points, differentiating with respect to m, we obtain:

$$\frac{\partial R}{\partial m} = \frac{n}{n + \alpha}(1 - s)^{m-1} \ln(1 - s) + \frac{\alpha}{n + \alpha}[m \ln(1 - s) + 1](1 - s)^{m-1}$$

Turning points are obtained when $1/(n + \alpha) = 0$ and when $(1 - s)^{m-1} = 0$; these are obviously trivial solutions; and when

$$\hat{m} = \frac{-\alpha - n \ln(1 - s)}{\alpha \ln(1 - s)}$$

where $\hat{m}$ is the value of m which maximizes or minimizes the value of R. Differentiating again with respect to m to determine whether

$$\left.\frac{\partial R}{\partial m}\right|_m$$

is a minimum or maximum point, we obtain

$$\frac{\partial^2 R}{\partial m^2} =$$

$$\frac{(1-s)^{m-1} \ln (1-s)}{n+\alpha} [n \ln (1-s) + \alpha m \ln (1-s) + 2\alpha]$$

which on substituting $\hat{m}$ for m gives a maximum when α and n are positive. Negative values of these parameters would be biologically meaningless. Substitution of different values of α, n, and s give a wide range of possible results, which can be roughly classified into three types.

(i) $\hat{m} \leqslant 1$: simple races will predominate.
(ii) $2 < \hat{m} < n-1$: intermediate races will be selected.
(iii) $\hat{m} \geqslant n$: the most complex race will displace all other races.

TABLE 1. Values of $\hat{m}$ required to maximize R, for ranges of values of s and α[a]

s	$\hat{m}$			
	$\alpha = 0.1$	$\alpha = 0.5$	$\alpha = 1.0$	$\alpha = 10.0$
0.1	−10.51	5.49[b]	7.49[b]	9.29[b]
0.25	−16.52	−0.52	1.48[c]	3.28[b]
0.5	−18.56	−2.56	−0.56	1.24
0.75	−19.28	−3.28	−1.28	0.52

[a]Where $\hat{m}$ is the number of virulence genes which maximize the fitness of the pathogen (R), s is the selection acting against each virulence allele in the absence of a corresponding host. The symbol α represents a measure of the realized rate of reproduction.

[b]A complex race will displace simpler races.

[c]The fitnesses of complex and simple races are equal, leading to a neutral equilibrium. In all other cases simple races will predominate.

The curve of R is not symmetrical about $\hat{m}$.

If m lies in the interval (1,2) or (n − 1, n) the genotype selected will depend on the value of $\hat{m}$ so the fitness of each of the genotypes must be calculated separately to determine which will displace the other. For example, consider a two-component multiline (n = 2). Table 1 gives the values of $\hat{m}$ for different values of s and α.

The most obvious characteristic of this model is its sensitivity to small changes in the reproduction rate. A small change in α can be sufficient to switch the result from a Type (i) to a Type (ii) outcome. Until more accurate measurements of the rate of growth of pathogens and the selection forces acting on them are available it will not be possible to predict with any certainty whether or not multilines will select complex or intermediate races, or if simple races will predominate. It is certain, that whatever equilibria are produced, they are likely to be neutral or unstable (Barrett, Wolfe, and O'Donald, *unpublished*).

In contrast to Groth's model, $\hat{m}$ is not only a function of s but also of the reproduction rate and of the number of components in the multiline. Even with the inclusion of these parameters it is still a simple model especially with the restrictions placed on the fitnesses. A more general model, of which this is a special case, is to be published elsewhere (Barrett, Wolfe, and O'Donald, *unpublished*).

LITERATURE CITED

1. GROTH, J. V. 1976. Multilines and "super-races": a simple model. Phytopathology 66:937-939.

2. BARRETT, J. A. 1978. A model of epidemic development in variety mixtures. Pages 129–137 *in* P. R. Scott and A. Bainbridge, eds., Plant disease epidemiology. Blackwell's Scientific Publications. 329 p.