Letter to the Editor

Estimating Parasitic Fitness of Plant Pathogenic Fungi: A Theoretical Contribution

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Mode (6) proposed a mathematical model to describe the manner in which the proportions of two races in a mixture will change when the two races have different reproductive rates. This model was used by Leonard (4) in studies of mixed *Puccinia graminis* f. sp. *avenae* populations with what he described as "reasonably good fit."

Horsten (3) also used the same model in his evaluation of parasitic fitness of carbendazim- and edinphos-resistant genotypes of *Septoria nodorum*. MacKenzie (5) explicitly related Mode's model with Vanderplank's apparent infection rate and observed that

$$\ln W = r_1 - r_2 \text{ and}$$
$$W = e^{r_1 - r_2}$$

in which W = relative parasitic fitness of the less fit race; r_1 = apparent infection rate of the less fit race; and r_2 = apparent infection rate of the more fit race.

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In Mode's terminology

$$(1 - S)^n = e^{(r_1 - r_2)\,t}$$

thus,

$$n\ln(1 - S) = (r_1 - r_2)\,t \rightarrow \ln(1 - S) = [(r_1 - r_2)\,t]/n$$

in which r_1, r_2 retain the meanings given above, S = coefficient of selection, and n = number of generations.

To summarize, if:

x_0, x_t = proportions or amounts of disease caused by a less fit (weaker) race at times 0 (beginning of epidemic) and t, respectively.

y_0, y_t = proportions or amounts of disease caused by a more fit (stronger) race at the same time.

r_1 = apparent infection rate for x

r_2 = apparent infection rate for y

then according to Mode (6) and MacKenzie (5)

$$x/y = (x_0/y_0)\,e^{(r_1 - r_2)t}$$

and

$$\ln(x/y) = \ln(x_0/y_0) + (r_1 - r_2)\,t \qquad (1)$$

Thus, the increase of proportion of the strong race can be predicted by the intensity ($r_1 - r_2$), the duration (t) of selective pressure, and its initial occurrence, when the favorable selective pressure starts to operate. Fleming and Person (2) have pointed out that one of the assumptions underlying Eq 1 is that "competitive or synergistic interactions between different units of inoculum on the common host are negligible"; ie, that the model is valid only during the logarithmic stage of an epidemic.

However, competition between the two races for the host's susceptible sites is liable to occur after the epidemic's logarithmic stage and this is the problem this paper attempts to solve.

Mathematical Model for Competing Races

When decreasing availability of a host's susceptible sites begins to become limiting, increase in amounts of disease can be described by:

$$dx/dt = r_1x(1-x-y) \quad (2)$$
$$dy/dt = r_2y(1-x-y) \quad (3)$$

in which; x, y, r_1, and r_2 have the meanings ascribed to them in Eq 1. At the same time to an observer, total visible disease $z = x + y$, and

$$dz/dt = r_3z(1-z) \quad (4)$$

Eq 2, 3, and 4 cannot be solved explicitly; however, Eq 2 and 3 can be used to calculate x_t/y_t. For $x + y = z$, from Eq 2 and Eq 3:

$$(1/x)\,(dx/dt) = r_1(1-z)$$
$$(1/x)\,(dy/dt) = r_2(1-z)$$

Since x and y are functions of t, by the chain rule:

$$(1/x)\,(dx/dt) = d\ln x/dt = r_1(1-z)$$
$$(1/y)\,(dy/dt) = d\ln y/dt = r_2(1-z)$$

Multiplying the above by r_2 and r_1, respectively, and subtracting the second equation from the first gives:

$$r_2(d\ln x/dt) - r_1(d\ln y/dt) = 0$$
$$d/dt(\ln x^{r_2} - \ln y^{r_1}) = d/dt[\ln(x^{r_2}/y^{r_1})] = 0 \text{ or}$$
$$\ln(x^{r_2}/y^{r_1}) = C \text{ (a constant)}$$

The constant $C = \ln(x_0^{r_2}/y_0^{r_1})$, in which x_0, y_0 the amounts of disease at the onset of the competition; ie, when $z \simeq 0.05$,

if $$\ln(x_t^{r_2}/y_t^{r_1}) = \ln(x_0^{r_2}/y_0^{r_1})$$
$$x_t^{r_2}/y_t^{r_1} = x_0^{r_2}/y_0^{r_1} \text{ or } x_t^{r_2-r_1}(x_t/y_t)^{r_1} = x_0^{r_2-r_1}(x_0/y_0)^{r_1}$$

and $$\ln(x_t/y_t) = \ln(x_0/y_0) + [(r_2-r_1)/r_1]\ln(x_0/x_t) \quad (5)$$

which again allows prediction of the increase of proportion of the strong race as a function of the intensity of selective pressure $[(r_2-r_1)/r_1]$, its initial occurence (x_0/y_0), and a factor that combines the effect of time and increased occupation of hosts' susceptible sites by the weak race $[\ln(x_0/x_t)]$.

Application of the Model

The difference between Eq 1 and 5 can best be illustrated when y_0 and y_t are relatively small; ie, when the "strong" race occurs in relatively small, but increasing, proportions. In practice this situation occurs in the early stages of selection of a fungicide-resistant race that occurs at low frequency in a pathogenic population with a fungicide-sensitive race at high frequency.
In this case, since y_0 and y_t are very small

$$\ln[x_t/(1-x_t)] = \ln[x_0/(1-x_0)] + r_1t$$

and $\ln(x_0/x_t) = \ln[(1-x_0)/(1-x_t)] - r_1t$. If this is substituted and suitably rearranged, Eq 5 becomes:

$$\ln(x_t/y_t) = \ln(x_0/y_0) + (r_1-r_2)\,t + [(r_2-r_1)/r_1]\ln[(1-x_0)/(1-x_t)] \quad (6)$$

It should be noted that during the logarithmic stage of an epidemic since $x_t = x_0e^{r_1t}$ Eq 5 becomes:

$$\ln(x_t/y_t) = \ln(x_0/y_0) + [(r_2-r_1)/r_1]\ln(x_0/x_0e^{r_1t}) = \ln(x_0/y_0) + (r_1-r_2)\,t$$

ie, identical to Eq 1.

What Eq 6 says is that the proportion of the strong race will increase more slowly than Eq 1 would predict because some of the susceptible sites available for its multiplication will have already been occupied by the weak race.

It also says that for a given ratio r_2/r_1 this "delaying" effect will be greater the higher the value of r_1 is, and that for a given value of r_1, this "delaying" effect will be greater the higher the ratio r_2/r_1.

Theoretical calculations that illustrate the difference between Eq 1 and 5 (or Eq 6) are presented in Table 1. At the end of a 40-day epidemic there is a very significant divergence between predictions based on the two equations. The log/time linear model (Eq 1) predicts a higher proportion of the strong race.

Most of the data in the literature such as those produced by Ruppel (7) or Horsten (3) cannot be used to verify the model presented in this paper. They have been generated by one or more consecutive passages (inoculations and reisolations) started with a mixture of known initial composition of sensitive and resistant spores. Thus apparent infection rates cannot be calculated and at the same time no judgement can be made as to whether the disease level at each passage was sufficiently high to cause competition for susceptible sites.

The only set of data found that could be used in a model comparison were those presented by Dovas et al (1) and subsequently discussed by MacKenzie (5).

The results of this comparison are presented in Table 2.

The comparison is not very conclusive. In the unsprayed "sensitive" strain epidemic the proportion of the resistant strain

TABLE 1. Comparison of two models to predict changes in frequencies of a "strong" race and a "weak" race of a plant pathogen in a theoretical epidemic[a]

Time (days)	Predicted values of y/x[b]	
	Eq 1[c]	Eq 5[d]
0	1:10,000	1:10,000
10	1:1,123	1:1,245
20	1:148	1:229
30	1:18.2	1:45.8
40	1:2.24	1:13.9

[a] Initial inoculum of the "weak" and "strong" races are $x_0 = 0.05$ and $y_0 = 0.05 \times 10^{-4}$, respectively; rates of increase of the "weak" and "strong" races are $r_1 = 0.07$ and $r_2 = 0.28$, respectively.
[b] y/x = (amount of "strong" race)/(amount of "weak" race).
[c] $\ln(x/y) = \ln(x_0/y_0) + (r_1-r_2)\,t$ (Eq 1).
[d] $\ln(x/y) = \ln(x_0/y_0) + [(r_2-r_1)/r_1]\ln(x_0/x_t)$ (Eq 5).

TABLE 2. Observed and predicted ratios of "weak" to "strong" races (x/y) of plant pathogens in mixed epidemics[a]

Time (days)	"Sensitive unsprayed"[a] x/y			"Mixed strain unsprayed"[a] x/y		
	Observed	Predicted		Observed	Predicted	
		Eq 1[b]	Eq 5[c]		Eq 1[b]	Eq 5[c]
0	15.66:1	(no estimate)		3.35:1	(no estimate)	
24	4.88:1	5.42:1	10.30:1	1.13:1	0.55:1	1.51:1
38	3.17:1	3.19:1	5.99:1	0.77:1	0.23:1	0.67:1

[a] Data of Dovas et al (1). "Sensitive unsprayed": $r_1 = 0.06423$ and $r_2 = 0.10842$. "Mixed strain unsprayed": $r_1 = 0.08307$ and $r_2 = 0.15819$.
[b] $\ln(x/y) = \ln(x_0/y_0) + (r_1-r_2)\,t$ (Eq 1).
[c] $\ln(x/y) = \ln(x_0/y_0) + [(r_2-r_1)/r_1]\ln(x_0/x_t)$ (Eq 5).

increased faster than even the linear log/time model would predict. In the unsprayed "mixed strain" epidemic, the proportion of the resistant strain increased slower and a better fit was provided by the model presented in this paper.

On balance, this evidence does not conclusively favor either model. One additional note is necessary to relate the data in Table 2 to MacKenzie's calculation. For the change in proportions of strains in the unsprayed plots inoculated with the "sensitive strain" MacKenzie used the apparent infection rate of this epidemic ($r = 0.07$) as the rate of increase of the "sensitive strain" in comparison with the rate of increase of the "resistant strain" ($r = 0.0926$) in unsprayed plots inoculated with the "resistant strain." This clearly overestimates the rate of increase of the "sensitive strain," because the "sensitive strain" epidemic was, in fact, caused by a mixture of an initially low proportion of the "resistant strain" with the "sensitive strain." Therefore, the observed infection rate was higher than the real infection rate of the "sensitive strain" alone, because the contaminant "resistant strain" reproduced faster and increased in frequency. This is apparent if one considers once more the basic equations describing the process:

$$dx/dt = r_1x(1-x-y),$$
$$dy/dt = r_2y(1-x-y),$$
$$dz/dt = r_3z(1-x-y),$$

in which x and r_1 are amount of disease and infection rate for the "sensitive strain," y and r_2 are amount of disease and infection rate for the "resistant strain," and $z = x + y$, and r_3 are observed amount of disease and the accordingly measured infection rate. Actually, r_3 is not a constant, but instead r_3 varies as the ratio x/y changes througout the epidemic.

Since obviously

$$dx/dt + dy/dt = dz/dt,$$

it then follows that

$$r_3 = (xr_1+yr_2)/(x+y),$$

and although this relation reflects "instant" values and, in general, cannot be used in calculations, it illustrates that using r_3 to estimate r_1, when $r_2 > r_1$, and in a time interval within which x/y decreases substantially, will lead to an overestimate of r_1 and, accordingly, an underestimate of $r_1 - r_2$.

The apparent infection rates used in Table 2 have been calculated using the method suggested by Vanderplank (8) (p. 109, Exercise 3) and are a closer approximation to reality.

Practical Implications

The model presented in this paper as well as the one developed by Mode (6) can be used to predict changing proportions of races or genotypes that, in a certain set of conditions, exhibit different apparent infection rates. Mode's model should be valid only in the early stages of an epidemic, but the model presented in this paper should work in the early as well as the later stages because it allows for the effect of competition. The production of field data that would allow verification of these models would be welcomed. Mixtures of fungicide-sensitive and -resistant races seem to be extremely useful for such studies, because they permit easy and measurable manipulation of selective pressure intensity and duration by fungicide application, and they allow practical measurement of the effects of those changes on genotype frequencies.

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