Letter to the Editor

Estimating Relative Fitness in Plant Parasites: Some General Problems

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In a recent letter to the editor, Skylakakis (8) examined the relative rate of replacement of one race of an asexually reproducing plant pathogen by a second race when both races compete for available infection sites. An earlier letter to the editor by MacKenzie (7) and a reply by Groth and Barrett (2) derived expressions describing the changes in the relative frequencies of two asexually reproducing pathogen races in the absence of competition. In this letter, a general treatment of the rate of replacement of one asexually reproducing race by a second is offered in which the treatments of MacKenzie (7), Groth and Barrett (2), and Skylakakis (8) are included as special cases.

If a pathogen population includes two races with numbers, N_1 and N_2, having intrinsic rates of increase, r_1 and r_2, respectively, then the instantaneous rates of change in the numbers of each race can be given by:

$$dN_1/dt = r_1 N_1 f_1(N_1,N_2) \quad (1)$$

and

$$dN_2/dt = r_2 N_2 f_2(N_1,N_2) \quad (2)$$

in which $f_1(N_1,N_2)$ and $f_2(N_1,N_2)$ are functions that modify the growth rates and combine the effects of intra- and inter-race competition. With epidemic foliar disease, it is often more convenient to use the proportion of leaf area affected by disease as an index of disease severity, in which case the equations become:

$$dx/dt = r_x x f_x(x,y) \quad (3)$$

and

$$dy/dt = r_y y f_y(x,y) \quad (4)$$

in which x and y are the proportions of the leaf area affected by each of the two races, r_x and r_y are the respective apparent infection rates of increase, and $f_x(x,y)$ and $f_y(x,y)$ correspond to the functions $f_1(N_1,N_2)$ and $f_2(N_1,N_2)$ above.

Case 1. If host tissue is not limiting (ie, there is no intra-race competition and there is no competition or interaction between races) then $f_1(N_1,N_2) = f_2(N_1,N_2) = 1.00$ and

$$dN_1/dt = r_1 N_1 \text{ and } dN_2/dt = r_2 N_2 \quad (5)$$

and the growth equations are those for exponential growth as used by MacKenzie (7) and Groth and Barrett (2). In equations 3 and 4 there is an implicit assumption that host tissue is potentially limiting and hence $f_x(x,y) \neq 1.00$ and $f_y(x,y) \neq 1.00$ although at very low levels of disease severity $f_x(x,y)$, $f_y(x,y) \cong 1.00$.

Case 2. If host tissue is limiting, then the rates of increase of the two races may be described by the Lotka-Volterra competition equations (1):

$$dN_1/dt = r_1 N_1[1 - (N_1/K_1) - \alpha(N_2/K_1)] \quad (6)$$

and

$$dN_2/dt = r_2 N_2[1 - \beta(N_1/K_2) - (N_2/K_2)] \quad (7)$$

in which K_1 and K_2 are the maximum numbers of race 1 and race 2, respectively, that can be maintained by the host tissues (ie, the "carrying capacities") and α and β are constants that determine the form of interaction between the two races: if $\alpha = 0$, then race 2 does not compete for resources with race 1 and equation 6 reduces to the logistic growth equation for race 1; if $\alpha > 0$, then race 2 competes for resources with race 1 and reduces the amount of resource available to race 1; if $\alpha < 0$, then the rate of increase of race 1 is enhanced by the presence of race 2 (similarly for β in equation 7).

For the model of disease severity (equations 3 and 4), we obtain:

$$dx/dt = r_x x(1 - x - \alpha' y) \quad (8)$$

and

$$dy/dt = r_y y(1 - \beta' x - y) \quad (9)$$

in which α' and β' correspond to the constants α and β in equations 6 and 7. If $\alpha' = 1.0$ and $\beta' = 1.0$, then equations 8 and 9 are identical to those used by Skylakakis (8).

If $K_1 = K_2 = K$, then, by dividing both sides of equations 6 and 7 by K, we obtain:

$$(dN_1/K)/dt = (r_1 N_1/K)[1 - (N_1/K) - \alpha(N_2/K)]$$

and

$$(dN_2/K)/dt = (r_2 N_2/K)[1 - \beta(N_1/K) - (N_2/K)]$$

but N_1/K and N_2/K are the proportions of leaf area of the host infected by race 1 and race 2, respectively, and consequently:

$$N_1/K = x \text{ and } N_2/K = y.$$

Thus, relating equations 1 and 2 to equations 3 and 4 and permitting the interchangeable use of numbers or disease severity at this level of analysis.

It then follows from equations 1 and 2 and by inference for equations 3 and 4 that:

$$d(N_1/N_2)/dt = [N_2(dN_1/dt) - N_1(dN_2/dt)]/N_2^2$$

$$= [r_1 N_1 N_2 f_1(N_1,N_2) - r_2 N_1 N_2 f_2(N_1,N_2)]/N_2^2.$$

$$\therefore d(N_1/N_2)/dt = (N_1/N_2)[r_1 f_1(N_1,N_2) - r_2 f_2(N_1,N_2)]$$

and similarly,

$$d(x/y)/dt = (x/y)(r_x f_x(x,y) - r_y f_y(x,y))$$

Case 3. If $f_1(N_1,N_2) = f_2(N_1,N_2) = f(N_1,N_2) = 1.00$ as in case 1 above, then,

$$d(N_1/N_2)/dt = (N_1/N_2)(r_1 - r_2)$$

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or

$$d(p/q)/dt = (p/q)(r_1 - r_2)$$

in which,

$$p = N_1/(N_1 + N_2) \text{ and } q = N_2/(N_1 + N_2)$$

as obtained by MacKenzie (7) and Groth and Barrett (2).

Case 4. If $f_x(x,y) = f_y(x,y) = (1 - x - y)$ as discussed in case 2 above, then:

$$d(x/y)/dt = (x/y)(1 - x - y)(r_x - r_y)$$

and the rate of replacement of one race by the other will be slower than when both races are increasing exponentially in absence of competition (cf, cases 1 and 3 above) because $(1 - x - y) < 1.0$. Since $(1 - x - y)$ will change during the course of an epidemic, the magnitude of the rate of change of $d(x/y)/dt$ will also change, as pointed out by Skylakakis (8).

Case 5. If, for example, $f_1(N_1, N_2) \neq f_2(N_1, N_2)$, as in the general Lotka-Volterra competition equations above (case 2), then:

$$d(N_1/N_2)/dt = (N_1/N_2)\{r_1[1 - (N_1/K_1) - (\alpha N_2/K_1)] - r_2[1 - (\beta N_1/K_2) - (N_2/K_2)]\}$$

and it is possible for the magnitude and direction of $d(N_1/N_2)/dt$ to change during the course of an epidemic, depending on the values of $r_1, r_2, K_1, K_2, \alpha, \beta$, and the starting values of N_1 and N_2. Thus, it is possible for either race to show a net increase during an epidemic under different conditions. Similarly, the Lotka-Volterra competition equations applied to disease severity yield:

$$d(x/y)/dt = (x/y)(r_x(1 - x - \alpha' y) - r_y(1 - \beta' x - y))$$

By analogy with Haldane's original method for the estimation of relative fitness (3), the expression $(r_1 f_1(N_1,N_2) - r_2 f_2(N_1,N_2))$ could be considered to be the "relative fitness" of race 1 to race 2 but this "relative fitness" will vary during the course of an epidemic unless there is no intra-race or inter-race competition. When there is competition, the estimator of "relative parasitic fitness" proposed by MacKenzie (7), viz " ....all one needs to do is to subtract the larger r from the smaller r (less-fit isolate)," is inappropriate. Indeed, no single numerical "relative fitness" value can describe the behavior of the ratio of the number (or frequencies) of the two races when there is competition. Under these conditions it is necessary for the functions $f_1(N_1,N_2)$ and $f_2(N_1,N_2)$ to be stated explicitly, perhaps in the form $(f_1(N_1,N_2) - f_2(N_1,N_2))$, as well as the intrinsic rates of increase, r_1 and r_2, and the estimated values substituted in the expression or listed with it, ie, the intrinsic rates of increase *and* the form of the fitness functions are required to describe the rates of change in pathogen populations. Similarly, if disease is measured in terms of disease severity, the apparent infection rates and the corresponding fitness functions should be used.

Some practical considerations. The models outlined above are drawn from two sources. Those describing changes in *population numbers* are drawn from ecology and their analogues describing changes in *disease severity* from plant pathology. In the form of differential equations in which they have been presented above, it is possible to show how they are related to one another. However, the relationship between the different forms of these equations masks some important differences in the concepts, which underlie the models and the practical problems in applying them to real data. In order to clarify this point, the following list describes some of the main assumptions underlying simple population growth models and is adapted from Pielou (6):

Assumption 1. Abiotic factors are sufficiently constant not to affect reproduction and survival rates. In other words, it is assumed that the parameters in the models are constant and, equally, the forms of interaction between organisms contained in the models are assumed to be constant, eg, the form of competition between different races is constant at all population densities.

Assumption 2. The population either has no age structure or maintains a stable age structure.

Assumption 3. Reproduction and survival rates respond instantly, without lag, to population density changes.

Assumption 4. "Crowding" affects all population members equally.

Assumption 5. Population growth is density dependent even at the lowest densities.

Assumption 6. The population under study constitutes a single population; on average, all individuals are exposed to the same range of factors.

It is only when all of the conditions implicit in these assumptions are fulfilled that it is possible to consider using the growth equations, solving them (where solutions exist), estimating the parameters, and fitting the data to the estimated curves. It should be immediately clear to any plant pathologist that these assumptions cannot be fully met in many plant diseases. Indeed, Vanderplank (9) pointed out that despite the superficial similarity between the logistic growth equation and his equations describing rates of change in disease severity, the equations are not identical because assumption 1 (above) cannot be met in most plant diseases. However, it is implicit in his argument that all of the other assumptions are assumed to be more or less fulfilled. Consequently, he stresses that the application of his equations can only be applied during very short intervals of time, although he does concede that "where r stays nearly constant over the whole observed course of an epidemic," the logit transformation allows r to be estimated. In this case, the application of his equations is identical to that of the logistic equation (see above). His derivation of R (the basic infection rate) and R_c (the corrected basic infection rate) represent a further attempt to overcome some of the other assumptions implicit in his model. Despite the distinction between logistic growth and infection processes so heavily stressed by Vanderplank, the logit transformation appears to be one of the most popular ways of fitting disease progress curves. Much of the development of the theory of disease progress is based on the assumption, either explicitly or implicitly, that the logistic growth equations adequately describe epidemic development (eg, Jowett et al [4]). Indeed, Zadoks and Schein (10) define r as the "apparent infection rate" *or* "the logistic infection rate."

Furthermore, a consideration of the mathematical assumptions on which the exponential growth, logistic growth, and Lotka-Volterra-type competition equations are based reveals that the equations themselves are simplifications of more complex expressions selected as the simplest mathematical expressions with the required behavior, mathematical tractability, and parameters that can be interpreted as having biological meaning (Lotka [5]).

A major difference between the models of infection processes developed by Vanderplank (9) and the logistic growth model is that the resource exploited by the pathogen or parasite is itself a living organism that may be growing. Consequently, the estimation of the apparent infection rate confounds the intrinsic rate of increase of the pathogen or parasite with the growth rate of the host plant, since "disease severity" is standardized to an estimate of the total available host tissue, which itself may be changing during the course of an epidemic. The consequences of this will be quite different in the logistic model and in the disease severity model. In the former, expansion of host tissue will appear as a change in the "carrying capacity," K, but in the latter as a change in the apparent infection rate, r. It is not difficult to envisage a situation in which the application of the logistic equation will give a positive rate of change in the number of infections but a negative rate of change in disease severity. Indeed this can sometimes be observed in spring cereals infected with foliar diseases in the U.K. at the tillering stage of growth, when plants seem to "grow away from the disease."

Both population growth and disease severity models have a place in plant pathology, but their use and interpretation must be tempered with an appreciation of the underlying assumptions on which they are built.

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