Letter to the Editor

Selection Pressures and Plant Pathogens: Stability of Equilibria

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Leonard (3) developed a mathematical model to describe the dynamics and equilibria of the genotypic frequencies of host resistance genes and pathogen virulence genes, given various parameters of selection intensity and fitness of host and pathogen. Examination of the model, however, shows that the equilibrium is unstable; this carries implications concerning the validity of Leonard's conclusions. For this discussion, we shall develop Leonard's model and examine it from two viewpoints, analytical and numerical.

We begin by defining the following: n is the frequency of virulence gene *V* in the haploid pathogen; m is the frequency of pathogen avirulence gene *v*, so that $n + m = 1$; p is the frequency of dominant resistance gene *R* in the diploid host; q is the frequency of recessive susceptible gene *r*, $(p + q = 1)$; t is the effectiveness of resistance $(0 \leqslant t \leqslant 1)$, so for genes expressing high resistance, t is about 1; k is the cost of virulence to the pathogen of the virulence gene in a given avirulent population; and a is the advantage conferred to the virulent pathogen genotype on resistant host genotypes relative to the avirulent pathogen on the nonresistant host.

Hence, Leonard develops the following table (Table 1).

Now we have (in Leonard's notation) that the frequency for the genotypes of the next cycle of pathogens is

$$n' = \frac{n\,[1 - k + (1 - q^2)\,a]}{1 - (1 - q^2)\,t + n\,[(1 - q^2)\,(a + t) - k]}$$

Eq. 1

From this, Leonard derives that the change in n for one cycle is

$$\Delta n = \frac{n\,(1 - n)\,[(1 - q^2)\,(a + t) - k]}{1 - (1 - q^2)\,t + n\,[(1 - q^2)\,(a + t) - k]}$$

Eq. 2

Leonard claims that "If the host and pathogen populations are allowed to come to equilibrium, so that $\Delta n = 0$,

$$1 - q^2 = k/\,(a + t) \ldots".$$

This is misleading. Equation 1 implies that there is no change in host gene frequency, and that equilibrium conditions are obtained for only the pathogen gene frequency, given a fixed host gene frequency. Equilibrium is reached when $n = 0$, or $n = 1$, or if the host gene frequency is fixed at $q = [1 - k/\,(a + t)]^{1/2}$; i.e., $1 - q^2 = k/\,(a + t)$, then the pathogen gene frequency will remain unchanged for any value of n between 0 and 1. This development does not say anything about equilibrium frequencies of the resistant host genotypes.

Leonard's model for selection in the host population introduces two further parameters, c, the cost to the pathogen of the resistance gene, and s. The concept of s gives us pause. Having previously defined, on page 207, fitness as ". . . a measure of the reproductive success of individuals. . .", Leonard states on page 211 "The term s in Table 3 represents severity of disease. The actual loss in fitness due to disease in the host is also proportional to the fitness of the pathogen infecting the host." This *assumption*, made to simplify the model development, while not unreasonable as an approximate description, may be strictly invalid; consider, for example, the case of tolerant cultivars that appear to be susceptible but yield as if they were resistant.

If we grant this assumption we arrive at Leonard's table of the relative fitnesses of host genotypes (Table 2).

Assuming that the entire host population is attacked, we have the frequency of the genotype of the next cycle of hosts to be

$$p' = \frac{p\,[1 - c - s\,(1 - t) + ns\,(k - a - t)]}{1 - s + nsk + (1 - q^2)\,[ts - c - ns\,(a + t)]}$$

Eq. 3

from which Leonard derives the change in frequency of p to be

$$\Delta p = \frac{pq^2\,[ts - c - ns\,(a + t)]}{1 - s + nks + (1 - q^2)\,[ts - c - ns\,(a + t)]}$$

Eq. 4

which is zero at equilibrium. The correct interpretation is that $\Delta p = 0$ if $p = 0$, $p = 1$, or, if the pathogen gene frequency is fixed at $n = (ts - c)/(ts + as)$, then $\Delta p = 0$ for any value of p between 0 and 1. Equation 3 makes no allowance for changes in pathogen gene frequency; in

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fact, n is implicitly assumed to be fixed.

Analysis of nontrivial equilibrium: Analytical approach.—We may now consider the nontrivial equilibrium point given by the two equations

$$n = (ts - c)/(ts + as) \qquad (ts > c)$$

$$1 - q^2 = k/(a + t) \qquad (k < a + t).$$

Eq. 5

We shall use n, m, p, and q to denote the nontrivial equilibrium frequencies, and n_j, m_j, p_j, and q_j to denote frequencies for the jth cycle. Thus

$$p_{j+1} = \frac{p_j [1 - c - s(1 - t) + n_j s(k - a - t)]}{1 - s + n_j sk + (1 - q_j^2)[ts - c - n_j s(a + t)]}$$

Eq. 6

and

$$n_{j+1} = \frac{n_j [1 - k + (1 - q_j^2) a]}{1 - (1 - q_j^2) t + n_j [(1 - q_j^2)(a + t) - k]}$$

Eq. 7

We can express this as

$$p_{j+1} = f(p_j, n_j) \text{ and } n_{j+1} = g(p_j, n_j)$$

where the f and g indicate two functions of the variables p_j and n_j. Thus, for the equilibrium values,

$$p = f(p, n), \qquad n = g(p, n).$$

Parenthetically, we note that we are assuming that the pathogen affects reproductive capacity, not viability, so that p and n remain unchanged during each cycle. If infection affects viability, the model might better be written

$$p_{j+1} = f(p_j, n_j) \text{ and } n_{j+1} = g(p_{j+1}, n_j),$$

Eq. 8

but note that

$$n_{j+1} = g[f(p_j, n_j), n_j] = h(p_j, n_j);$$

i.e., a different function of p_j and n_j.

Some discussion of the stability of equilibria, with examples in biology, is given by Lotka (4); however, for completeness, we shall develop the following for the host-pathogen genotype case. Consider first the approximation using Taylor's theorem for the multivariable case [which may be found in any advanced calculus text; e.g.; Fulks (2)],

$$f(p_j, n_j) \doteq f(p, n) + (p_j - p)\frac{\partial f(p, n)}{\partial p} + (n_j - n)\frac{\partial f(p, n)}{\partial n}$$

which leads to the form,

$$\begin{pmatrix} p_{j+1} \\ n_{j+1} \end{pmatrix} \doteq \begin{pmatrix} p \\ n \end{pmatrix} + \begin{pmatrix} \frac{\partial f}{\partial p} & \frac{\partial f}{\partial n} \\ \frac{\partial g}{\partial p} & \frac{\partial g}{\partial n} \end{pmatrix} \begin{pmatrix} p_j - p \\ n_j - n \end{pmatrix}$$

The matrix of partial derivatives, called the Jacobian, is evaluated at the equilibrium point. The approximation holds if p_j, n_j are close to the equilibrium. Let $p_j - p = \delta_j$, $n_j - n = \epsilon_j$, be the deviations from the equilibrium of the frequencies at the jth cycle. The expression may then be written as

$$\begin{pmatrix} \delta_{j+1} \\ \epsilon_{j+1} \end{pmatrix} = X \begin{pmatrix} \delta_j \\ \epsilon_j \end{pmatrix} \quad \text{where X is the Jacobian,}$$

or $d_{j+1} = X d_j$ where d_j is a column vector.

This is in the form that Bodmer (1), amongst others, discusses. The stability of the system is indicated by the

TABLE 1. Relative fitness of pathogen genotypes on susceptible and resistant hosts

Pathogen genotype	Frequency	Relative fitness of pathogen genotypes: on *rr* (susceptible)	*Rr*, *RR* (resistant)	
		q^2	2pq	q^2
v (avirulent)	m	1	$1 - t$	
V (virulent)	n	$1 - k$	$1 - k + a$	

TABLE 2. Relative fitness of host genotypes interacting with avirulent and virulent pathogens

Host genotype	Frequency	Relative fitness of host genotype afflicted by avirulent (*v*) or virulent (*V*) pathogen	
		m	n
RR (resistant)	p^2	$1 - c - s(1 - t)$	$1 - c - s(1 - k + a)$
Rr (resistant)	2pq		
rr (susceptible)	q^2	$1 - s$	$1 - s(1 - k)$

latent roots (or characteristic roots or eigenvalues) of X. Generally, if the dominant root is less than 1 then the equilibrium is stable, and vice versa. Parenthetically, we may note that because we are using an approximate analysis, if the dominant root is very close to or equal to 1, then the analysis is inconclusive.

The roots may be complex, of the form $\lambda = \alpha + i\beta$ where $i^2 = -1$ and neither α nor β is zero. In this case, the system will spiral inwards if $(\alpha^2 + \beta^2)^{1/2} < 1$ and outwards if $(\alpha^2 + \beta^2)^{1/2} > 1$.

To examine the stability of this specific case, we differentiate the functions represented by Equations 6 and 7 with respect to p and n, and determine their values at the equilibrium.

$$\left.\frac{\partial f}{\partial p}\right|_{eq} = 1,$$

$$\left.\frac{\partial f}{\partial n}\right|_{eq} = \frac{ps\,(k - a - t)}{1 - s + nsk} = x_{12}, \text{ say,}$$

$$\left.\frac{\partial g}{\partial p}\right|_{eq} = \frac{2qn\,(1 - n)\,(a + t)^2}{a + t - kt} = x_{21},$$

$$\left.\frac{\partial g}{\partial n}\right|_{eq} = 1.$$

We now consider the characteristic roots of the Jacobian:

$$\begin{vmatrix} 1 & x_{12} \\ x_{21} & 1 \end{vmatrix}$$

Here the solution for λ is obtained from the equation

$$(1 - \lambda)^2 - x_{12}\,x_{21} = 0.$$

So $$\lambda = 1 \pm (x_{21}\,x_{12})^{1/2}$$

and the dominant root is $\lambda = 1 + (x_{21}\,x_{12})^{1/2}$

where $$x_{21}\,x_{12} = \frac{2pqnms\,(a + t)^2\,(k - a - t)}{(1 - s + nsk)\,(a + t - kt)}$$

which is negative for any values of a, k, s, and t that give a nontrivial equilibrium value. Therefore, the dominant root of the system is complex with $(\alpha^2 + \beta^2)^{1/2} > 1$, so the system spirals outwards from the equilibrium. The equilibrium point therefore is unstable.

[Similarly we can show that the equations (Eq. 8) lead to a Jacobian with latent roots such that $[\alpha^2 + \beta^2]^{1/2} = 1$.]

Numerical approach.—The analysis establishes that the equilibrium point is not locally stable. We also can show instability by a numerical approach. Write a computer program for the recursive expressions Eq. 6 and 7, start with values of p_0, n_0 close to, but not equal to, the equilibrium point and examine a printout, or a graphical display of (p_1, n_1), (P_2, n_2), ... for a number of iterations. While this does not "prove", in the mathematical sense, the stability or otherwise of the equilibrium, it does provide data that can be clearly followed. We shall consider that an end point is reached when n_j or p_j becomes sufficiently close to 0 or 1 that stochastic effects ensure a fairly high probability of fixation. As a caution though, we might point out that the end point of an unstable system may be somewhat affected by the numerical accuracy of the computation, especially where 1,000 or more iterations may be necessary. Little emphasis should be placed either on the exact number of iterations required to reach the end point, or on the value of n_j or p_j that is not zero.

Figure 1 plots the last spiral of the system for $a = 0.1$, $c = 0.01$, $k = 0.3$, $s = 0.2$, and $t = 1.0$. The program started with values of p_0, n_0 within 0.1% of the equilibrium values, and took about 1,800 iterations to reach the end point. Each spiral took about 70 iterations.

CONCLUSION

In misinterpreting the implications of Equations 2 and 4, Leonard clearly errs, and the conclusions he draws concerning the conditions for stabilizing selection are invalid. Furthermore, the correct interpretation of his model, demonstrated here, shows that it is inherently unstable and is not, therefore, an explanation of stabilizing selection, such as that observed in the stable host-pathogen systems existent in the "fertile crescent" area of the Middle East. Models that include, for

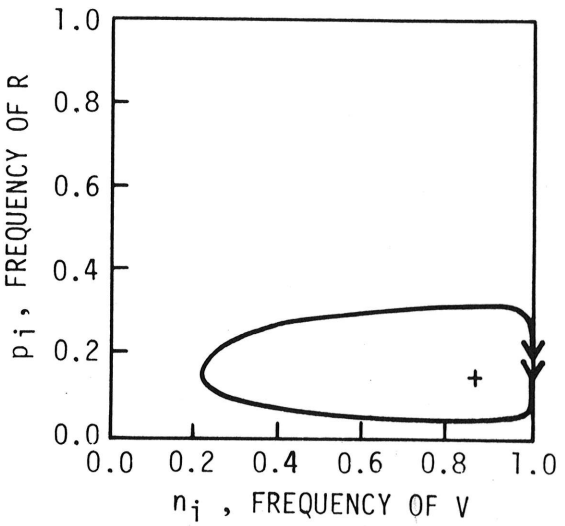

Fig. 1. Numerical analysis of the model. Equations 6 and 7 (see text) were used to calculate respectively the frequency n_j of the virulence gene *V* in the haploid pathogen, and the frequency p_j of the dominant resistance gene *R* in the diploid host. The initial values were within 0.1% of the nontrivial equilibrium (marked +). The graph shows the locus of the last cycle of iterations before n_j became unity.

example, competition between pathogens, varying environmental effects, or having only a proportion of the host attacked may be more satisfactory.

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