

Letter to the Editor

In Response to 'Selection Pressures and Plant Pathogens: Stability of Equilibria'

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According to Sedcole (3), Leonard's (1) model of selection pressures in plant host-pathogen systems does not fit the observed behavior of these systems in nature. There might be several reasons for Sedcole's conclusion: (i) the model contains inaccurate information, (ii) the model is accurate as far as it goes but it omits some factor(s) important in nature, (iii) Sedcole misinterpreted the model, or (iv) Sedcole misinterpreted the behavior of natural host-pathogen systems.

Sedcole (3) suggested some areas in which he feels that the model may be inaccurate, but the main basis for his criticism is his demonstration that the nontrivial equilibrium point is unstable. This lack of a stable equilibrium point raises some interesting questions about the model, but (as shown below) the consequences of instability of the equilibrium point differ from those he described.

Before discussing the implications of the unstable equilibrium point, there are some specific areas of confusion in Sedcole's (3) interpretation of the model to be resolved. First, the meaning of the term stabilizing selection as Sedcole used it is different from its meaning in Leonard's (1) paper. Leonard used the term stabilizing selection in the way that Van der Plank (4) described it in the context of genes for virulence in plant pathogens. In Van der Plank's use of the term, stabilizing selection is selection that favors alleles for avirulence, as opposed to directional selection that favors alleles for virulence. Sedcole used the term stabilizing selection in its proper genetical sense. In a single-locus model, stabilizing selection means the forces that maintain gene frequencies near their equilibrium values (2). That is, stabilizing selection would act to reduce frequencies of genes for virulence when they were above the equilibrium level, but would act to increase their frequencies when they were below equilibrium levels. Thus, stabilizing selection should be represented as the combination of two opposing forces that lead to a stable equilibrium. Sedcole is correct in saying that a model without a stable equilibrium point cannot be an explanation of stabilizing selection in its proper genetical sense. It should be pointed out, however, that there is nothing in the model or in Sedcole's analysis of it that would indicate that there is not a force of selection that acts against unnecessary genes for virulence [i.e., stabilizing selection *sensu* Van der Plank (4)].

To avoid this kind of confusion in the future, plant pathologists should discontinue current usage of stabilizing selection in plant pathology as selection favoring alleles for avirulence on susceptible host plants. It would be better to refer to this type of selection simply as selection against unnecessary virulence. As illustrated in Leonard's (1) model, selection against unnecessary virulence is essential to the existence of equilibrium points (whether stable or unstable) at intermediate gene frequencies.

Sedcole (3) questioned the correctness of Leonard's (1) assumption that loss of host fitness due to disease is proportional to the fitness of the pathogen genotype infecting the host. He suggested that differences in tolerance among host genotypes would make this assumption invalid. It seems obvious to us that when disease damage is proportional to the rate of pathogen increase, a pathogen genotype of greater fitness should cause greater disease damage in the host than a less-fit pathogen genotype would cause. This is all that is implied in the assumption. A host genotype with more tolerance may be damaged less than a genotype with less tolerance, but the assumed relationship between more-fit and less-fit pathogen genotypes should still hold true. Even on the tolerant host plant, the pathogen genotype with greater fitness should cause more damage than a less-fit pathogen genotype would cause. If significant changes in tolerance occur as pleiotropic effects of major genes for resistance, that condition could be added to the model, but there is no evidence available now to suggest that it should be.

Sedcole (3) suggested that the model assumes that the entire host population is attacked. This is a misinterpretation. The model assumes that host plants are infected at random, so that all plants have an equal probability of becoming infected, but it is not necessary that every plant in the host population must be infected. Of course, an infection will be successful only if the genotypes of the host and pathogen are compatible. The probability of a compatible infection depends on the genetic compositions of the host and pathogen populations. There is no provision in the model for protected sites where part of the host population may grow but the pathogen cannot enter and cause disease. Such a provision is sometimes included in predator-prey models.

Sedcole's (3) statements that equations 1 and 3 (in his letter) make no provisions for changes in host gene frequencies and pathogen gene frequencies, respectively, also may be a misinterpretation. In the model the frequencies of resistant and susceptible plants are

assumed to remain constant during the growing season, but the frequency of genes for virulence in the pathogen population may change according to forces of selection. In the next growing season a new host population is initiated from seeds produced in the preceding season. A change in frequencies of resistant and susceptible plants may occur depending on the relative numbers and viability of seeds produced by each genotype in the previous season. Seed production by resistant and susceptible host genotypes depends upon the amount of disease suffered by each, which in turn is related to the composition of the pathogen population as well as to the suitability of the environment for disease increase. The model assumes that the gene frequencies in the pathogen population do not change during the interval between growing seasons, but a factor for differential survival can easily be added to the model. Thus, under the assumptions of the model, changes in genetic composition of host and pathogen populations occur in a series of alternate steps. During the growing season the pathogen population adjusts to the host population as it exists in that season. The new host population in the next growing season represents an adjustment to the disease damage caused by the pathogen population of the previous growing season. Over the course of a year each population may change, and the change in each population may cause a complementary change in the other population. Thus, $n_{j+1} = g(p_j, n_j)$ and $p_{j+1} = f(p_j, n_{j+1})$.

Sedcole (3) determined mathematically that the nontrivial equilibrium point in the model at $n = (ts - c)/(ts + as)$ and $1 - q^2 = k/(a + t)$ is not stable. In addition to the nontrivial equilibrium point, there are also eight trivial equilibrium points at: $n = 0, p = 0$; $n = 1, p = 0$; $n = 1, p = 1$; $n = 0, p = 1$; $n = 0, 1 - q^2 = k/(a + t)$; $n = 1, 1 - q^2 = k/(a + t)$; $n = (ts - c)/(ts + as), p = 0$; and $n = (ts - c)/(ts + as), p = 1$. Using an analysis similar to Sedcole's we have shown that each of these trivial equilibrium points also is unstable. This means that if the gene frequencies are displaced slightly from any of the nine equilibrium points, they will not return to that equilibrium point. What exactly they will do is beyond the scope of the analysis used by Sedcole, because that analysis applies only to the area in the immediate vicinity of the equilibrium point.

It can be shown mathematically that if host and pathogen populations are of unlimited size, it is impossible for n or p to reach 1 or 0 within a finite number of generations unless either n or p is already equal to 1 or 0. In the model,

$$\begin{aligned} n + \Delta n &= \frac{n[1 - (1 - q^2)t] + (mn - n^2)[(1 - q^2)(a + t) - k]}{1 - (1 - q^2)t + n[(1 - q^2)(a + t) - k]} \\ &= \frac{n[1 - (1 - q^2)t] + n[(1 - q^2)(a + t) - k]}{1 - (1 - q^2)t + n[(1 - q^2)(a + t) - k]} \end{aligned}$$

From this it can be seen that if $n < 1, n + \Delta n < 1$, and if $n > 0, n + \Delta n > 0$. Therefore, if host and pathogen populations were of infinite size, the frequencies of genes for virulence and resistance would cycle forever around the nontrivial equilibrium point unless the frequencies were originally at equilibrium. Although populations

never can be infinitely large it is worth considering this point, because for reasonably large populations forward and reverse gene mutation may effectively prevent gene frequencies from reaching 1 or 0. Thus, the perpetual cycling of gene frequencies may be a characteristic of finite as well as infinite populations.

Mathematically defining the behavior of gene frequencies in this model at values other than the equilibrium frequencies is an extremely complex problem. Therefore, Sedcole's (3) use of a numerical analysis appears to be the best available approach. However, Sedcole's numerical analysis is incorrect. Using either of two independently developed computer programs for the model (one by H. E. Schaffer, Department of Genetics, North Carolina State University and one by R. J. Czochor), we find that gene frequencies do not spiral outward in the way that Sedcole described. We used the same parameters as Sedcole ($a = 0.1, c = 0.01, k = 0.3, s = 0.2$, and $t = 1.0$) and started at values of $n_0 = 0.8626$ and $p_0 = 0.1462$ that are within 0.1% of the equilibrium values. We ran the analysis through 200 iterations and found no detectable outward spiral; the track of the third cycle around the equilibrium point was essentially the same as that of the first. Using these same values except that $p_0 = 0.1329$ (at equilibrium $\hat{p} = 0.1472$), we found that n reached a maximum value of 0.907 in the first cycle and oscillated with a period of about 70 iterations per cycle, but the value of n never exceeded 0.907 at any time even though the analysis was continued through 3,000 iterations. Again, the last cycle followed

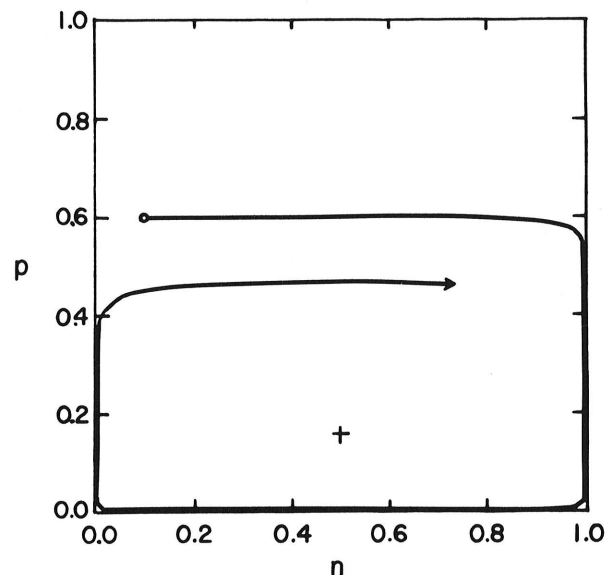


Fig. 1. Change in gene frequencies in a theoretical host-pathogen system with n = frequency of the virulence gene, p = frequency of the resistance gene, $n_0 = 0.1$ and $p_0 = 0.6$ are initial gene frequencies, and the following parameters exist: cost of virulence (k) = 0.3, cost of resistance (c) = 0.1; difference in fitness of the virulent pathogen genotype on the resistant host compared with the susceptible host (a) = 0.0, effectiveness of resistance (t) = 1.0, and suitability of the environment for disease development (s) = 0.2. The nontrivial equilibrium point is represented by the cross.

essentially the same track as the first.

Under some conditions we find an inward spiral from the starting point toward the equilibrium point. For example, using the parameters $a = 0.0$, $c = 0.10$, $k = 0.3$, $s = 0.2$, and $t = 1.0$, we started an analysis at $n = 0.10$ and $p = 0.60$. Under these conditions the period of oscillations varied from more than 150 iterations per oscillation to less than 90. We found that the gene frequencies spiraled inward markedly in the first cycle and less in subsequent cycles (Fig. 1). After more than 900 iterations (nine cycles) there was still a detectable inward spiral of the gene frequencies. We did not continue the analysis beyond the ninth cycle, but obviously the spiral could not continue all the way to the equilibrium point because the equilibrium point is unstable. Therefore, there must be a limit cycle beyond which the inward spiral could not continue. At this stage in our study of the model we suspect that the model has many concentric limit cycles so that the amplitude of gene frequency oscillations depends largely on the point at which gene frequencies start in the system.

We do not agree with Sedcole's (3) conclusion that the model is not consistent with the behavior of gene frequencies in the 'stable' host-pathogen systems in the

Middle East. From what we know of these systems we systems with stable equilibrium points or more typical of systems that have unstable equilibrium points but stable limit cycles. Either type of system tends to retain genes for resistance and virulence in host and pathogen populations, respectively.

We are exploring many modifications of the model such as including provisions for gene mutations, variable weather conditions, etc. and will report the conclusions of these studies in a later paper.

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