Stability of a Gene-for-Gene Coevolution System Under Constant Perturbations

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Accepted for publication 8 April 1998.

In 1977, Leonard proposed a model of host-parasite interactions to account for polymorphisms of host resistance and pathogen virulence in natural pathosystems (6). The latest version of the model (updated by Leonard in 1994 [7]) is

$$\begin{aligned} n_{i+1} &= g(p_i, n_i) = \frac{n_i[1-k+(1-q_i^2)a]}{1-(1-q_i^2)t+n_i[(1-q_i^2)(a+t)-k]} \\ p_{i+1} &= f(p_i, n_{i+1}) = \frac{p_i[1-c-s(1-t)+n_{i+1}s(k-a-t)]}{1-s+n_{i+1}ks+(1-q_i^2)[ts-c-n_{i+1}s(a+t)]} \end{aligned} \quad (1)$$

in which n_i and m_i ($n_i + m_i = 1$) are the frequencies of genes for virulence and avirulence in the parasite population at the end of the i th host generation, respectively; p_i and q_i ($p_i + q_i = 1$) are the frequencies of genes for resistance and susceptibility in the host population, respectively; relative fitness of the avirulent parasite on the resistant host is $1-t$, where t represents the effectiveness of the resistance; relative fitness of the virulent parasite on the susceptible host is $1-k$, where k is the cost of virulence; c is the fitness cost of resistance; s is the severity of disease; and $1-k+a$ is the relative fitness of the virulent parasite on the resistant host, where a determines the type of selection in the parasite population. When $a = 0$, there is a cost of virulence even when it is necessary to parasitize the resistant host. This is considered a hard selection, in which the presence of the virulence gene causes a reduction in the intrinsic rate of reproduction by the parasite (7,10). When $a = k$, virulence has a fitness cost when it is unnecessary, as it is when a virulent parasite infects a susceptible host. This can be considered a soft selection, in which the relative fitness of the virulent parasite is reduced in competition with the avirulent parasite on a susceptible host (7,10).

The model has a single internal equilibrium point (EP; n_e, p_e), at which selection pressures are balanced and there is no change in the frequency of either resistance or virulence. The internal EP can be calculated by the following equation set:

$$\begin{aligned} 1-q_e^2 &= k/(a+t) \\ n_e &= (ts-c)/(ts+as) \end{aligned} \quad (2)$$

in which $p_e = 1 - q_e$. The model also has four trivial EP: (0, 0), (0, 1), (1, 0), and (1, 1).

Leonard's model (equation set 1) describes host-parasite interactions in a natural system. However, many host-parasite systems

are subsystems in an agroecosystem disturbed by farming activities. Without perturbations, the dynamics of the frequencies (n_i, p_i) of a host-parasite system would proceed generation by generation as described by previous studies (7), but under perturbations from farming activities, the dynamics of the frequencies (n_i, p_i) might be quite different. Ecologists have found that the dynamic behaviors of population models would be changed drastically if a constant perturbation is considered (11,14), and use of perturbations has been suggested as a possible measure to control dynamics of ecological or physical systems (2,11,14).

Plant pathologists have noted that under some types of perturbations, the dynamic behaviors of the frequencies (n_i, p_i) in Leonard's model would be changed. Specific types of natural perturbations to Leonard's model have been studied for nonagricultural systems, such as pathogen migration (8), mutation, and random drift of a pathogen population (5). These studies showed that the coevolution process of the host-parasite system described by Leonard's model is sensitive to some types of perturbations and caused us to consider the dynamic behaviors of Leonard's model under other possible perturbations, especially those related to agricultural activities.

An important expected type of perturbation due to agricultural practices is directed incremental input of resistance genes into a host-parasite system. An agricultural crop can be considered part of a host-parasite coevolving system if the host genotype frequencies are controlled or partly controlled by human choices rather than by natural selection. For example, in managed grassland stands, selected populations of a grass species may be grown in close proximity with wild populations of the same species. In such instances, parasites may be distributed randomly and spread freely between the wild and bred populations, and managers may periodically introduce increasing frequencies of resistant grasses into managed grasslands. Another example is isolated areas of developing countries where commercial production of seeds is not available and land races of some crops are genetically mixed and may coevolve with their parasites. Annual improvement in resistance is limited only to exchanges of seeds between farmers for plants with better yields, which often are more resistant. These are two examples of host-parasite systems perturbed by an incremental input of resistance genes in the host population in each generation.

The purpose of our study is to investigate the coevolution dynamics of the host-parasite system described by Leonard's model under the condition that in each generation there is a constant percentage of resistance genes, λ , added to the host population. Our study provides additional insight into the host-parasite interactions described by Leonard's model, which may be useful for analysis and prediction of a real host-parasite system (1,12).

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Publication no. P-1998-0430-010
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LEONARD'S MODEL UNDER CONSTANT PERTURBATIONS

If a constant percentage of a resistance gene, λ , is added to the host population at the beginning of each generation, then Leonard's model under this perturbation can be described as

$$\begin{aligned} n_{i+1} &= g(p_i, n_i) \\ p_{i+1} &= f(p_i, n_{i+1}) + \lambda \end{aligned} \quad (3)$$

in which $g(p_i, n_i)$ and $f(p_i, n_{i+1})$ are defined in equation set 1 as the frequencies of virulence and resistance at the end of the $i + 1$ generation; n_{i+1} and p_{i+1} ($p_{i+1} = f(p_i, n_{i+1}) + \lambda$) are the initial frequencies of the next generation; and λ is a positive constant smaller than 1 and satisfies the condition $p_{i+1} \leq 1$ for every i . For convenience, we term equation set 3 the modified Leonard's model, which describes a perturbed host-pathogen system. Parameters a , c , k , s , and t have the same biological meanings as in equation set 1.

In equation set 3, n_{i+1} and p_{i+1} are the initial frequencies of the $i + 2$ generation. The frequencies for virulence and resistance at the end of the $i + 1$ generation are $g(p_i, n_i)$ and $f(p_i, n_{i+1})$, respectively. For example, n_0 and p_0 are the initial frequencies for the first generation, and $g(p_0, n_0)$ and $f(p_0, n_1)$ ($n_1 = g(p_0, n_0)$) are the frequencies at the end of the first generation. The initial frequencies for the second generation are $n_1 = g(p_0, n_0)$ and $p_1 = f(p_0, n_1) + \lambda$. This is an important difference in the definitions of n_i and p_i for the modified and original Leonard's models. In the original Leonard's model, n_{i+1} and p_{i+1} are considered either the frequencies at the end of the $i + 1$ generation or the initial frequencies of the $i + 2$ generation, because no perturbations are considered. To avoid confusion, we use (n_{i+1}^*, p_{i+1}^*) to denote $(g(p_i, n_i), f(p_i, n_{i+1}))$ the frequencies of virulence and resistance at the end of the $i + 1$ generation to distinguish them from (n_{i+1}, p_{i+1}) , which were the initial frequencies for the $i + 2$ generation. The interactions between (n_{i+1}, p_{i+1}) and (n_{i+1}^*, p_{i+1}^*) are $n_{i+1}^* = n_{i+1}$ and $p_{i+1} = p_{i+1}^* + \lambda$.

One of the salient points in studying Leonard's model is the stability of the internal EP, because the model may contain clues to the causes of stable polymorphisms in natural pathosystems. Leonard (7) suggested that the equilibria in the model may be stable in biological terms, even though they do not meet the mathematical criteria of stability. Mathematically, a stable EP means that any changes in gene frequencies of the pathogen or host away from the EP will result in a return to it. We investigated the interactions among perturbation, λ , parameter set a , c , k , s , and t , and stability of the internal EP of the modified Leonard's model (equation set 3). We found that the dynamic behaviors differ significantly from those under no perturbations (equation set 1), particularly in relation to stability of the internal EP. The results of the simulations are presented below to illustrate the effects of perturbation, λ , at different magnitudes on the dynamic behaviors of host-parasite interactions. The first part of the simulation results is from the original Leonard's model under no perturbations (equation set 1), which is used as a frame of reference for the simulation results from the modified Leonard's model (equation set 3).

DYNAMIC BEHAVIORS OF THE MODIFIED LEONARD'S MODEL

Stability of the internal EP of Leonard's model as described by equation set 1. The internal EP of equation set 1 is determined by parameters a , c , k , s , and t . When parameter values are given, the internal EP of the host-parasite system can be calculated by equation set 2. Using equation set 1, we ran simulations with 30 sets of parameter values, and in all simulations, frequencies n_i and p_i converged toward the internal EP. Simulations with the following parameter sets illustrate the model behavior:

- (i) $a = 0$, $c = 0.03$, $k = 0.3$, $s = 0.8$, and $t = 1$ (lower fitness cost)
- (ii) $a = 0$, $c = 0.3$, $k = 0.3$, $s = 0.8$, and $t = 1$ (higher fitness cost)

The internal EP (n_e, p_e) calculated by equation set 2 is (0.96250, 0.16334) for the first parameter set and (0.6250, 0.16334) for the second parameter set. The simulation results are presented in Figure 1, which shows that if the initial gene frequencies (n_0, p_0) are inside the unstable limit circle (7), frequencies (n_i, p_i) spiral inward toward the internal EP but do not reach it in a very large number of generations (60,000 generations), even if the initial frequencies (n_0, p_0) are very close to the internal EP (Fig. 1C and F). The internal EP, therefore, does not appear to be stable in a mathematical sense. However, because the deviations of frequencies (n_i, p_i) from the internal EP (Fig. 1C and F) are very small, the gene frequencies can be considered biologically stable. The results of our simulations, including those illustrated in Figure 1, agree with published results on the stability of the internal EP (3,4,6–10,13). The simulations also were extended over a much greater number of generations than previously have been tested. Our simulations confirm that the EP in Leonard's model is stable in a biological sense, although it may not meet mathematical criteria for stability (7).

Stability of the modified Leonard's model. When a constant proportion of a resistance gene, λ , is added to the host population in Leonard's model at each generation, the structure of the model is changed, and the dynamic behaviors of frequencies (n_i, p_i) in the host-parasite system also are changed. The internal EP (n_e, p_e) of the modified Leonard's model (equation set 3) is different from that of the original model (equation set 1) and can be calculated as

$$\begin{aligned} n_e &= g(p_e, n_e) \\ p_e &= f(p_e, n_e) + \lambda \end{aligned}$$

The frequencies of the perturbed model at the EP are

$$\begin{aligned} p_e &= 1 - q_e \\ n_e &= \frac{\lambda[(1 - q_e^2)(ts - c) + (1 - s)] + p_e q_e^2 (ts - c)}{p_e q_e^2 s(a + t)} \end{aligned} \quad (4)$$

in which $1 - q_e^2 = k/(a + t)$.

Because the virulence gene frequency, n_e , should be less than 1 and more than 0, i.e., $0 < n_e < 1$, the level of perturbation, λ , should satisfy the following condition:

$$0 < \frac{\lambda[(1 - q_e^2)(ts - c) + (1 - s)] + p_e q_e^2 (ts - c)}{p_e q_e^2 s(a + t)} < 1 \quad (5)$$

In other words, λ is restricted to

$$\lambda < (as + c)p_e q_e^2 / [(1 - q_e^2)(ts - c) + (1 - s)]$$

We define the threshold as

$$\lambda_0 = (as + c)p_e q_e^2 / [(1 - q_e^2)(ts - c) + (1 - s)] \quad (6)$$

If $\lambda > \lambda_0$, there would be no internal EP for equation set 3 and no basis for discussing the convergence of frequencies (n_i, p_i) or (n_i^*, p_i^*) . Because $n_i^* = n_i$, $p_i^* = p_i - \lambda$, and λ is a small constant, the dynamic behaviors of frequencies (n_i^*, p_i^*) are consistent with that of (n_i, p_i) , and the internal EP of (n_i^*, p_i^*) is $(n_e, p_e - \lambda)$.

We used parameter sets 1 and 2 to test the modified Leonard's model. The simulation results on the dynamics of frequencies (n_i, p_i) are presented in Figures 2 and 3, corresponding to the first and second data sets, respectively. We let $\lambda = 0.0005$ and 0.0021 and $\lambda = 0.01$ and 0.05 for the first and second data sets, respectively. Because $n_i^* = n_i$, $p_i^* = p_i - \lambda$, and the four values of λ we considered were very small, the phase planes from simulations on frequencies (n_i, p_i) and (n_i^*, p_i^*) are almost the same. Therefore, only phase planes on frequencies (n_i, p_i) are presented.

Based on the simulation results, we found the following features of the modified Leonard's model: (i) the internal EP of the modified Leonard's model was stable (Figs. 2 and 3), and frequencies (n_i, p_i) reached the internal EP (n_e, p_e) within finite generations when perturbation, λ , was considered. (ii) Under different levels of perturbation, λ , the rates of convergence of frequencies (n_i, p_i) to the internal EP were different. For the first parameter data set, it took $\approx 6,000$ generations for frequencies (n_i, p_i) to reach the internal EP and $\lambda = 0.0005$ compared to $\approx 1,700$ generations and $\lambda = 0.0021$ (Fig. 2B and D). For the second data set, it took ≈ 300 and ≈ 70 generations to converge the internal EP under $\lambda =$

0.01 and 0.05, respectively (Fig. 3B and D). (iii) There exists a value, $\lambda_{\max} \leq \lambda_0$, such that when $\lambda = \lambda_{\max}$ frequencies (n_i, p_i) will converge to the stable internal EP at the greatest rate; this is a direct inference of the second feature. Because under different perturbation levels (all the perturbation levels should be less than λ_0) the convergence rates of (n_i, p_i) are different, there should be a $\lambda_{\max}$ among all the possible perturbation levels.

Equation 6 is obtained from equation 5: $0 \leq n_e \leq 1$. In fact, the use of perturbation, λ , should keep frequencies (n_i, p_i) within the interval $0 \leq p_i \leq 1$ and $0 \leq n_i \leq 1$ for all generations. The situation becomes very complex if the interactions between λ and all the

Fig. 1. Phase planes from simulations, using Leonard's host-pathogen interaction model (equation set 1 in text), with two sets of parameters: **A through C**, $a = 0$, $c = 0.03$, $k = 0.3$, $s = 0.8$, and $t = 1$: the internal equilibrium point (EP) is (0.92650, 0.16334); and **D through F**, $a = 0$, $c = 0.3$, $k = 0.3$, $s = 0.8$, and $t = 1$: the internal EP is (0.6250, 0.16334). $\times$ indicates the position of the internal EP in the system coordinates. T is the number of simulation generations. The initial and final frequencies are denoted by $p(0)$ and $p(T)$ for the resistant host and by $n(0)$ and $n(T)$ for virulence in the parasite. The final frequencies ($n(T)$ and $p(T)$) in **B** and **E** are used as the initial frequencies in **C** and **F**, respectively. Parameters are defined in text.

intervals of $(0 \leq p_i \leq 1, 0 \leq n_i \leq 1)$, $i = 1, 2, \dots$ are taken into consideration. Therefore, $\lambda < \lambda_0$ is a necessary condition for frequencies (n_i, p_i) of a pathosystem to converge to the internal EP, but it is not a sufficient condition, i.e., if the internal EP of a perturbed dynamic system is stable, λ must be less than λ_0 , but $\lambda < \lambda_0$ may not ensure a stable internal EP. This point is illustrated by the simulation results of the two parameter sets mentioned above. The threshold value of λ_0 for the first parameter set calculated by equation 6 is 0.00796. Simulations show that when $\lambda > \lambda_0$, the internal EP does not exist, when $\lambda_0 > \lambda > 0.0021$ the internal EP exists but is unstable, and when $\lambda \leq 0.0021$ the internal EP exists and is stable. The threshold value of λ_0 for the second parameter set is 0.098. When $\lambda > \lambda_0 = 0.098$, the internal EP does not exist, or else the internal EP exists and is stable.

DISCUSSION

The perturbations of host-parasite dynamics studied by Leonard (8) and Kirby and Burdon (5) are natural perturbations. We studied perturbations resulting from farming activities. Although the two types of perturbations are different, both can change the stability of Leonard's model and speed up the rate of convergence of the host-parasite system. This may explain why the slow coevolution process described by the original Leonard's model (Fig. 1) is rare in real agriculture systems and why the predictions of many theoretical models do not fit well with real data from agricultural systems.

In a host-parasite system, we may be unable to control natural perturbations, such as mutation and gene migration of the pathogen population, but we may be able to control the deployment of resistance genes in the host population. Thus, more studies are needed on the interaction between the magnitude of parameter λ and the dynamic behaviors of frequencies (n_i, p_i) . Equation 6 shows that to have a stable pathosystem the added proportion of the resistance gene, λ , cannot be arbitrarily large when the values of the parameter set of Leonard's model are given. Ecologically, it shows that we can alter a host-parasite system by increasing the proportion of resistance genes, but the alteration (the magnitude of λ) should be within the range permitted by the parameters that define the system (the right side of equation 6 is a function of the parameter set). Otherwise, the convergence of frequencies (n_i, p_i) or (n^*_i, p^*_i) of a system would be impossible, which means that a stable host-parasite system cannot be achieved.

For each combination of a parameter set, there may be a $\lambda_{\max}$ in $(0, \lambda_0)$, such that when $\lambda = \lambda_{\max}$ frequencies (n_i, p_i) correspond to the fastest convergence rate to the stable internal EP. However, it is difficult to find $\lambda_{\max}$. For example, the value of λ_0 corresponding to the first data set ($a = 0$, $c = 0.03$, $k = 0.3$, $s = 0.8$, and $t = 1$) is 0.00796, and the value in the second data set ($a = 0$, $c = 0.3$, $k = 0.3$, $s = 0.8$, and $t = 1$) is 0.098. In the first data set, $\lambda_{\max}$ seems to be 0.0021, because in a series of simulations with different values of $\lambda \leq 0.0021$, the larger λ is the faster the convergence. With the second data set, however, the rate of convergence of the model

Fig. 2. Phase planes on frequencies (n_i, p_i) from simulations with the modified Leonard's host-pathogen interaction model (equation set 3 in text) under smaller fitness cost of resistance, c . The parameter set is $a = 0$, $c = 0.03$, $k = 0.3$, $s = 0.8$, and $t = 1$. $\times$ indicates the internal equilibrium point (EP) of Leonard's model under no perturbations (equation set 1 in text: 0.96250, 0.16334), and $+$ indicates the stable internal EP of the modified Leonard's model. **A and B**, The stable internal EP of the modified Leonard's model (equation set 3) is (0.964856, 0.16334); **C and D**, the stable internal EP of the modified Leonard's model (equation set 3) is (0.972395, 0.16334). $p(T)$ and $n(T)$ are the frequencies of the resistant host and virulence in the parasite at the beginning of generation T , respectively. Because the internal EPs of the Leonard's model and the modified Leonard's model are very close, there is some overlap in $+$ and $\times$. Parameters are defined in text.

Fig. 3. Phase planes on frequencies (n_i, p_i) from simulations with the modified Leonard's host-pathogen interaction model (equation set 3 in text) under larger fitness cost of resistance, c . The parameter set is $k = 0.3$, $a = 0$, $t = 1$, $c = 0.3$, and $s = 0.8$. The difference between Figures 3 and 2 is the value of parameter c . The internal equilibrium point (EP) of Leonard's model without perturbations (equation set 1 in text), indicated by $\times$, is (0.6250, 0.16334). The stable internal EP of the modified Leonard's model is indicated by $+$. **A and B**, The stable internal EP of the modified Leonard's model is (0.66315, 0.16334); **C and D**, the stable internal EP of the modified Leonard's model is (0.81638, 0.16334). $p(T)$ and $n(T)$ are the frequencies of the resistant host and virulence of the parasite at the beginning of generation T , respectively. Parameters are defined in text.

does not necessarily increase with the increase of λ . When $\lambda = 0.05$, the rate of convergence is faster than when $\lambda = 0.01$, but for some values of $\lambda > 0.09$, the rate of convergence of the model is slower than that for $\lambda = 0.05$. Currently, we can only try to approach $\lambda_{\max}$ by repeated computer simulations.

The difference between the two data sets is in the values of parameter c , which reflects the fitness cost of cultivar resistance. Fitness cost was low (0.03) in the first data set and relatively high (0.3) in the second data set. In our simulation, a low fitness cost of resistance corresponds to a small perturbation threshold, λ_0 , and a slow rate of convergence of frequencies (n_i, p_i) to the internal EP. Similarly, we can use simulations to examine the effect of other parameters on the rate of convergence of the host-parasite system under different levels of perturbations.

In this study, we only examined the dynamic properties of Leonard's model under constant perturbations characterized by a constant increment of resistance genes in a host population. In practice, perturbations can be caused by other types of complex activities that are difficult to analyze quantitatively. For example, if in generation i the added proportions of a resistant cultivar (λ_i) are different, Leonard's model under this kind of perturbation would be

$$\begin{aligned} n_{i+1} &= g(p_i, n_i) \\ p_{i+1} &= f(p_i, n_{i+1}) + \lambda_i \end{aligned} \quad (7)$$

in which $\lambda_i \geq 0$ and satisfies the condition $p_{i+1} \leq 1$. Equation set 7 is more general than equation set 3, but the internal EP of the

model, if it exists, is difficult to express as a mathematical formula, and the coevolution process is difficult to predict. Because the values of λ_i are different, the dynamic behaviors of frequencies (n_i, p_i) and (n^*, p^*) might be quite different and should be discussed separately. We may need to study the more complex perturbations if Leonard's model is to be used to analyze host-parasite interactions in an agricultural system.

The investigation of the dynamic behaviors of Leonard's model under perturbed environments can help us analyze host-parasite interactions in agroecosystems and lead us to a better understanding of the host-parasite coevolution process in the real world.

ACKNOWLEDGMENTS

Journal paper J-17863 of the Iowa Agriculture and Home Economics Experiment Station, Ames. Project 3384 supported by Hatch Act and State of Iowa funds. We thank K. Leonard and the two reviewers for valuable suggestions and S. Sanogo and Y. Luo for critical reviews.

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