

## A MODEL OF INDUCIBLE DEFENSE

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Hosts often amplify the expression of defensive structures or chemicals in response to an increasing probability of attack by parasites, herbivores or predators (Harvell, 1990). I address two questions here. First, what is the optimal pattern of amplification by the host in response to an increasing abundance of parasites? Second, what are the population dynamics of the host-parasite system when the hosts follow an optimal pattern of amplification?

I use an extension of the basic Lotka-Volterra model to describe the effects of hosts and parasites on each other:

$$\begin{aligned} dh_i/dt &= h_i \left[ v(1 - \alpha\xi_i) - d(1 + \delta\xi_i) \right. \\ &\quad \left. - m(1 - \gamma\xi_i)p \right. \\ &\quad \left. - \sum_{j=1}^N h_j [v(1 - \alpha\xi_j) \right. \\ &\quad \left. - d(1 + \delta\xi_j)] \right] \\ dp/dt &= p \left[ -s + b \sum_{j=1}^N h_j(1 - \gamma\xi_j) \right], \quad (1) \end{aligned}$$

where  $h_i$  is the numerical abundance of the  $i$ th host genotype,  $i = 1, \dots, N$ , and  $p$  is the numerical abundance of the parasite. The model has the usual demographic parameters of the Lotka-Volterra formulation:  $v$  and  $d$  are the host birth and death rates,  $m$  is the morbidity and mortality per parasitic attack, and  $b$  and  $s$  are the parasite birth and death rates. All of these parameters are greater than zero. The host population has density dependent competition for limited resources represented by the term with summation over  $h_j$  in the  $dh_i/dt$  equations. The carrying capacity of hosts is standardized to one.

The host has the ability to induce defensive struc-

tures or chemicals in response to parasite density. The level of induction for a host of genotype  $i$  is  $\xi_i$ , which may depend on the abundance of parasites, the abundance of hosts, and the parameters of the model. The value of  $\xi_i$  ranges from zero to one. Induced defense has both costs and benefits. The costs are reduced fecundity by a proportion  $\alpha\xi_i$  and increased death rate by a proportion  $\delta\xi_i$ . The benefit is reduced morbidity and mortality as a result of parasitic attack by a proportion  $\gamma\xi_i$ . The ability of a parasite to gain from an attack on a host of genotype  $i$  is reduced by the proportion  $\gamma\xi_i$ , thus the overall birth rate of a parasite is  $b \sum_i h_i(1 - \gamma\xi_i)$ . The cost and benefit parameters all range between zero and one.

The optimal pattern of induction can be obtained by finding the maximum of  $dh_i/dt$  as a function of  $\xi_i$ . It is easy to show that  $dh_i/dt$  is monotonically increasing in  $\xi_i$  if  $m\gamma p > (v\alpha + d\delta)(1 - h_i)$ , is monotonically decreasing in  $\xi_i$  if the inequality is reversed, and is independent of  $\xi_i$  if the relation is an equality. Any host genotype  $i$  that follows the optimal pattern of induction will have a rate of increase,  $dh_i/dt$ , that is greater than the rate of increase for any genotype that does not follow the optimal pattern. Thus no host polymorphism is maintained and we can drop the  $i$  and  $j$  subscripts.

The optimal pattern of induction is

$$\xi = \begin{cases} 1 & p > \frac{(v\alpha + d\delta)(1 - h)}{m\gamma} \\ 0 & p < \frac{(v\alpha + d\delta)(1 - h)}{m\gamma} \end{cases}, \quad (2)$$

where the level of induction has no effect on fitness when equality holds. The level of induction is more likely to be high when parasite pressure,  $p$ , is high, as would be expected. A bit surprising at first glance is the fact that induction is more likely to be high when

host abundance,  $h$ , is high. This apparently occurs because, at high density, the rate of growth ( $dh/dt$ ) of the host population is low, and thus there is a higher premium on surviving parasitic attack rather than on producing new offspring.

The conditions in Equation (2) define an optimal transition between the induced and uninduced states given the host and parasite densities, ( $h$ ,  $p$ ). If we assume that the genetic equilibrium has been obtained, then the population dynamics of host and parasite can be examined by study of Equation (1). The equilibrium of the system is

$$h^* = \frac{s}{b(1 - \gamma\xi)} \quad (3)$$

$$p^* = \frac{[v(1 - \alpha\xi) - d(1 + \delta\xi)](1 - h^*)}{m(1 - \gamma\xi)}$$

This equilibrium is locally stable when  $h^*$  is between zero and one, and  $p^*$  is greater than zero.

Combining the optimal level of induction, Equation (2), with the location of the equilibrium, yields the conditions for the host to be induced at equilibrium

$$\xi = \begin{cases} 1 & \gamma > \frac{(v\alpha + d\delta)}{v - d} \\ 0 & \gamma < \frac{(v\alpha + d\delta)}{v - d} \end{cases} \quad (4)$$

where the level of induction is a neutral character when equality holds. These conditions can be translated to read: induction is favored at equilibrium when the rate of increase in resistance benefits with increasing induction,  $\gamma$ , is greater than the marginal rate of increase in resistance costs with increasing induction,  $(v\alpha + d\delta)/(v - d)$ .

The global dynamics of the system are peculiar because of the sharp transition between induced and uninduced states shown in Equation (2). Many of the general features of the dynamics can be seen in Figure 1. The line through the middle of the figure shows the transition for the host between induced and uninduced states given in Equation (2). When below the line the host is uninduced and the system follows a spiral toward an attracting point given by the symbol "U" in the figure that would be an equilibrium if the host never induced [Equation (3) with  $\xi = 0$ ]. The spiral trajectories from the uninduced region soon cross the transition line at which the host induces. Trajectories starting in the induced region spiral toward the equilibrium for the induced state. The equilibria for the induced and uninduced states are always on the same side of the transition line; the side is determined by the conditions in Equation (4).

When a trajectory hits the transition line, there are two possible outcomes. First, if the dynamical flow in the opposite state is away from the line, then the trajectory will continue. This is shown in the trajectory farthest to the left and the two trajectories farthest to the right in Figure 1. Second, if the direction of flow is toward the line from both sides, then the system may move reasonably quickly along the line until the flow on one side changes direction, or the system may move

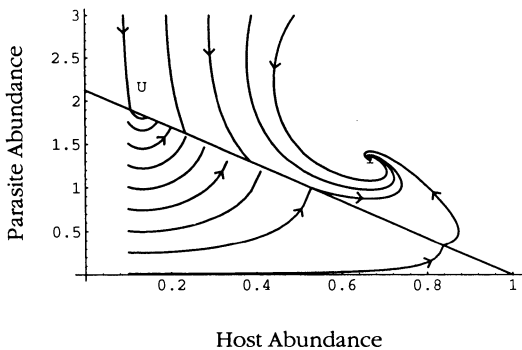


FIG. 1. The dynamics of a host-parasite system with inducible defense, where the host follows the optimal amplification pattern given in Equation (2). Details are explained in the text. The parameters for this figure are  $v = 3$ ,  $d = 0.5$ ,  $m = 1$ ,  $b = 6$ ,  $s = 0.8$ ,  $\alpha = 0.5$ ,  $\delta = 0.5$ ,  $\gamma = 0.8$ .

very slowly while staying close to the transition line. The dynamics near the line are difficult to examine because of the discontinuous transition between induced and uninduced states. The particular parameters in Figure 1 show a case in which the flow moves toward the line from both sides over a considerable distance. For many other parameter combinations, the length of the transition line over which opposite flows meet is smaller and the system moves more smoothly toward the equilibrium determined by Equation (3) and Equation (4).

Four conclusions can be drawn from this simple model. (1) Natural selection favors a phenotypically plastic response by hosts with a sharp transition between induced and uninduced defensive states. (2) High parasite density favors induction of defense, as expected. (3) Surprisingly, high host density also favors a transition to the induced state because density dependent competition places a higher premium on surviving parasitic attack. (4) The forces favoring induction follow a simple rule when the system is at both genetic equilibrium for patterns of host induction and ecological equilibrium for the abundance of host and parasites: the hosts are favored to be in the induced state when, with increasing level of induction, the rate of increase in resistance is greater than the marginal costs of induction imposed on the intrinsic rate of increase.

The model presented here assumes that hosts assess and immediately adjust their phenotype in response to the probability of attack. This may be a reasonable approximation for certain chemical or cellular defenses. By contrast, structural defenses tend to develop slowly in response to parasite challenge in nudibranchs, barnacles, bryozoans, and other aquatic invertebrates (Harvell, 1990). Developmental lags lower the correlation between expression of defense and actual challenge; this unpredictability may lower the induction threshold (Clark and Harvell, 1992).

The induction of defense in response to parasite abundance is sharp in some organisms but graded in others (Harvell, 1990). The present model predicts a sharp transition because of the linear relationship as-

sumed between the costs and benefits of induction. Nonlinear cost-benefit functions may cause a graded response that follows a pattern similar to the marginal value rule in conclusion (4) above.

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