

Notes and Comments

Does Host Self-Regulation Increase the Likelihood of Insect-Pathogen Population Cycles?

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The ability of insect pathogens (in particular viruses) to cause long-term stable limit cycles in their hosts was first demonstrated theoretically by Anderson and May (1980, 1981). Since then, there has been extensive discussion and controversy about whether the cycles seen in temperate forest insects (Varley 1949; Baltensweiler 1964; Baltensweiler et al. 1977) are caused by pathogens (Myers 1988) and whether the addition of greater biological detail to the simple Anderson and May model increases or decreases the likelihood of cycles. The Anderson and May model is a set of coupled ordinary differential equations, related to the Lotka-Volterra predator-prey model, in which neither the host nor the pathogen shows any density-dependent self-regulation. Since the type of forest insect that shows long-term cycles frequently severely depletes its food supply, it is natural to ask whether the addition of host self-regulation makes cycles more likely. This was first done by Bowers et al. (1993), who concluded that density dependence was unlikely to increase the probability of population cycles, although the opposite conclusion was reached the following year by Dwyer (1994). The apparent contradiction was reconciled by White et al. (1996), who showed that the two studies had incorporated host self-

regulation in different ways within Anderson and May's basic framework.

All the possible examples of pathogen-induced population cycles come from temperate forest insects with discrete generations inhabiting highly seasonal environments, while the models discussed above are all phrased in continuous time with perfectly overlapping generations. Here, we argue that the way to add greater biological realism to the Anderson and May model and to move toward the parameterization of real systems is to change the model framework to one that more accurately reflects the natural history of the system. Previously, we have developed a two-part model, consisting of a "within-generation" component phrased as time-delayed differential equations that describe the age-structured interaction between the host and pathogen within the summer, and a "between-generation" element that maps the densities of host and pathogen at the end of one season onto the densities at the beginning of the next (Briggs and Godfray 1996; see also Godfray et al. 1994 and Rohani et al. 1994 for a similar host-parasitoid model). Versions of the model incorporating biological assumptions equivalent to those of Anderson and May do not predict limit cycles, a finding that partly reflects the well-known destabilizing properties of time lags (May 1974b) and partly reflects the fact that in the seasonal model (and in nature) infected hosts are killed before reproductive maturity. These models can predict cycles equivalent to those found by Anderson and May, but so far this has required the inclusion of a long-lived reservoir for the pathogen (perhaps in the soil) or some degree of vertical transmission (Briggs and Godfray 1996). Our purpose here is to ask whether the addition of host density dependence changes the likelihood of limit cycle population dynamics, with this more natural model framework.

Briefly, at the beginning of the season, the host population enters a developmental stage (of length T_L , typically the larval or part of the larval stage), during which they are susceptible to the pathogen. Hosts that escape pathogen attack and survive to the end of the season mature into

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adults and produce eggs that become susceptible larvae in the following season. Hosts that succumb to the pathogen go through an incubation phase before dying and releasing infectious pathogen particles into the environment. An important parameter is Φ , the ratio of the incubation period (T_i) to the length of time that susceptible hosts are present during the season (T_L). If Φ is small, the pathogen is able to go through a number of replication cycles within a single host generation. The “free-living” pathogen particles decay in the environment, possibly at different rates within and between seasons. (For full details of the structure of the model, see table 1 and app. A.)

The instantaneous risk of pathogen infection is given by the function $k \ln(1 + \nu P/k)$, where P is pathogen density, ν is the transmission rate when the pathogen is rare, and k is the extent to which transmission is influenced by pathogen density. When k is large, the risks of infection increase linearly with pathogen density (the Anderson and May assumption). As k gets smaller, there is increasing density dependence in pathogen efficiency, with the risks of infection increasing sublinearly with pathogen density (for further description and defense of this transmission function, see Reeve et al. 1994; Briggs and Godfray 1995, 1996).

It is well known that, in structured predator-prey models, the order in which reproduction, natural enemy attack, and self-regulation occur can influence the predicted population dynamics (May et al. 1981). We explored these effects by investigating three versions of the model in which host density dependence was included before, during, or after the window of host susceptibility to the pathogen (see app. A). We model density-dependent self-regulation by including an additional instantaneous per capita risk of mortality that is proportional to the host population density (aL where L is larval host population density and a is the strength of density dependence). The overall strength of self-regulation is described by $\alpha = aT_d$, where T_d is the length of time over which the density dependence operates.

Our understanding of the results of this fairly complex model is greatly enhanced by examining the limiting case in which there is time for only a single round of pathogen infection during the host’s vulnerable window ($\Phi \geq 1$). In certain cases, this model simplifies to a pair of coupled difference equations (app. B) similar to those used to investigate the effect of host density dependence in insect host-parasitoid systems (May et al. 1981). However, there are two main differences between the insect-parasitoid and

Table 1: Summary of variables and parameters for the full seasonal age-structured model

	Description	Value
Variable:		
$L(t, \tau)$	Susceptible host larval density at time t in generation τ	...
$I(t, \tau)$	Infected host larval density at time t in generation τ	...
$P(t, \tau)$	Pathogen density at time t in generation τ	...
$\psi(P[t, \tau])$	The nonlinear infection rate, a function of pathogen density	...
$S(\tau)$	Host survival before and after susceptible window (this may be density-dependent; see app. A)	...
$D_L(t, \tau)$	Host mortality not due to the pathogen during the susceptible period (this may be density-dependent; see app. A)	...
Parameter:		
F	Host fecundity	2.0
T_L	Stage length of susceptible hosts	50
T_i	Stage length of infectious period	1–50
T_s	Length of season	100
d_L	Density-independent death rate of susceptible hosts	.0
d_i	Density-independent death rate of infected hosts	.0
d_p	Density-independent death rate of pathogen particles	.001
γ	Number of pathogen particles released on death of infected host	100
α	Strength of self-regulation	.5–.001
ν	Transmission coefficient	.01
k	Strength of pathogen nonlinear transmission	.01–10
σ_p	Pathogen overwinter survival	.75
σ_L	Host overwinter survival	1.0

Note: Parameter values in bold are those varied in the figures.

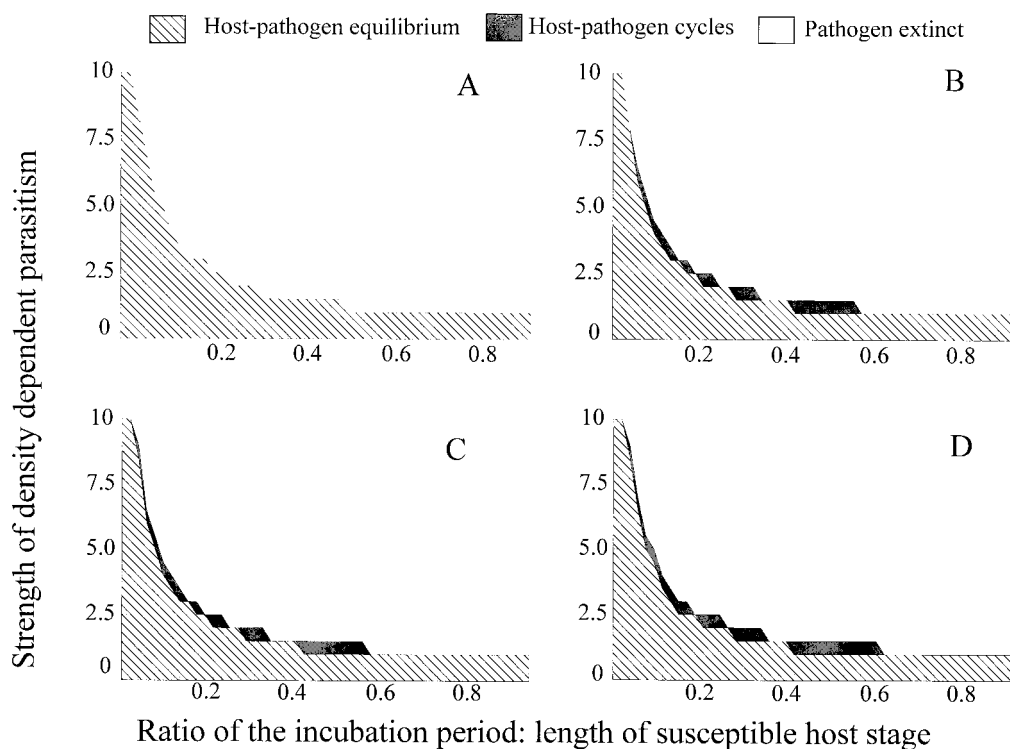


Figure 1: Region of local stability (in the density-dependent parasitism versus relative incubation period plane) for (A) the seasonal age-structured model in which host density dependence is absent. Note that many cycles of pathogen infection ($\Phi < 0.1$) require less nonlinearity in pathogen transmission ($kT_L \gg 1.0$) for the interaction to be stable. In the limit ($\Phi \rightarrow 1.0$), the host-pathogen interaction is stable only when $kT_L < 1.0$. B, Host density dependence is introduced and acts before parasitism. C, Host density dependence acts after parasitism. D, Host density dependence acts during the season. Note that in all cases B–D, the introduction of host density dependence increases the region over which the host-pathogen interaction persists but in general does not increase the likelihood of host-pathogen cycles. In the absence of pathogens, hosts persist at a stable equilibrium (parameters: $\alpha = 0.001$, $F = 2.0$).

the insect-pathogen systems. The first is that, while most parasitoids produce one or a few new parasitoid offspring from a parasitized host, pathogens such as viruses can produce in the order of 10^7 – 10^{10} new infectious particles from an infected host. The second difference is that, while parasitoids live at most for a single season, pathogen particles potentially can remain infectious for a number of years. Previous models that lacked host density dependence found that these two differences have little effect on population dynamics (Briggs and Godfray 1996). Here, the number of particles released from an infected host had a simple scaling effect on the equilibrium densities, and the carryover of pathogen between seasons affected the rate of convergence to equilibrium but not the position of the stability boundary.

In the absence of host density dependence, persistent consumer-resource limit cycles do not occur in either the insect-pathogen or the insect-parasitoid versions of the discrete-time model. The only two types of dynamics observed are a stable host-pathogen equilibrium, if the non-

linearity in the pathogen transmission is sufficiently strong ($kT_L < 1$) and divergent oscillations with eventual extinction of the pathogen if the nonlinearity is weak ($kT_L > 1$). In appendix B, stability analysis shows that, for certain ranges of parameter values, the addition of host density dependence allows persistent limit cycles to occur. In the host-pathogen discrete-time model when $\gamma = 1$ (the parasitoid version), persistent host-pathogen cycles occur only in parameter regions where the pathogen has little impact on host equilibrium density. High levels of carryover of pathogen between years expand the region in which persistent cycles occur and extend it to regions in which the pathogen can have a relatively high impact on the density of the host.

Adding density dependence to the seasonal model may or may not increase the likelihood of population cycles. Numerical exploration of the parameter space indicated that this depended chiefly on the strength of host density dependence and the fraction of the infectious pathogen that survived between years. Consider first relatively weak

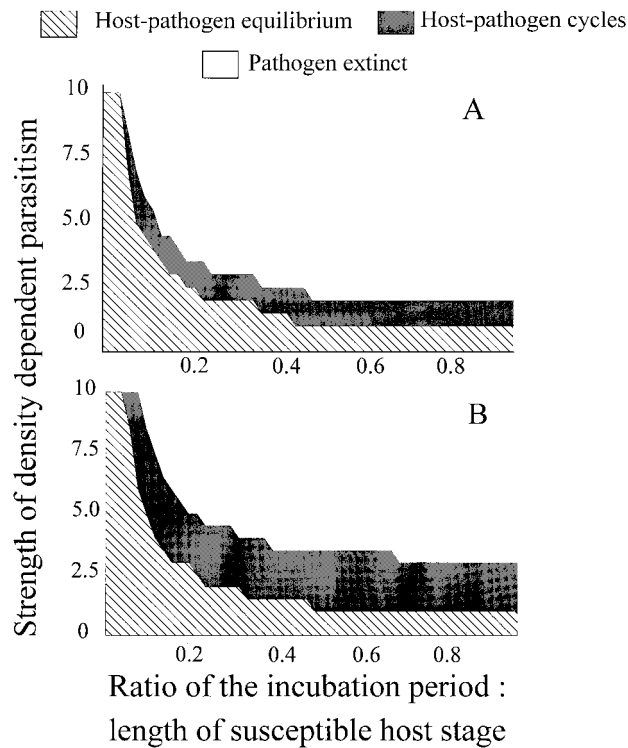


Figure 2: Local stability regions (in the density-dependent parasitism versus relative incubation period plane) for a more powerful host self-regulation acting (A) before parasitism and (B) after parasitism. Note the increased likelihood of cycles with the increased level of host density dependence (cf. fig. 1B–D). Parameters: $\alpha = 0.5$, $F = 2.0$.

density dependence ($\alpha = 0.001$; when $\Phi = 1$, the carrying capacity of the host in the absence of the pathogen is approximately 700 for the parameter values in table 1 compared with an equilibrium determined purely by the pathogen of 0.04 when the transmission parameter $k = 1$). The addition of density dependence has little effect on the likelihood of population cycles (fig. 1B–D), with these predicted in only a small region of parameter space between regions of stability and instability. Where the density dependence is included in the life cycle of the host has little effect on the dynamics.

Now consider more powerful host self-regulation ($\alpha = 0.5$; when $\Phi = 1$, the carrying capacity of the host is now 1.38). The region of population cycles is much greater (fig. 2A, B) because the host density dependence operates at sufficiently low population densities to prevent the destabilizing divergent oscillations. Increasing the carryover of infectious pathogens between years also in-

creases the likelihood of cycles. This occurs because a long half-life of infectious pathogens in the environment prevents the rapid recovery of host populations after an epizootic that leads to destabilizing diverging oscillations. In the absence of host self-regulation, increased persistence of pathogens in the environment is a stabilizing factor in continuous-time insect-pathogen models (Anderson and May 1981; Briggs and Godfray 1995) while allowing the pathogens to survive between years can affect stability in discrete-time insect-pathogen models (app. B).

Whether adding host self-regulation to continuous-time insect-pathogen models increases the likelihood of population cycles (Bowers et al. 1993; Dwyer 1994; White et al. 1996) depends on the extent of the stabilizing mechanisms that are already present. As White et al. (1996) pointed out, Bowers et al. (1993) added host density dependence to a model in which cycles were already possible. In this case, the stabilizing mechanism was reproduction by infected hosts. The addition of host self-regulation in this case simply added an additional damping effect and did not increase the region in which cycles occur. Dwyer (1994) added host density dependence to a model in which infected hosts were not able to reproduce (which is more realistic for temperate forest insect systems in which pathogens generally kill infected hosts when they are still in the larval stage, not allowing for reproduction). As in the basic host-pathogen discrete-time model (app. B), this baseline model predicts divergent oscillations and Dwyer (1994) found that the addition of density dependence was able to stabilize these divergent cycles and to produce persistent host-pathogen limit cycles. Although we have not explored all possible seasonal insect-pathogen interactions with host density dependence, the results found here (along with those in Briggs and Godfray 1996) suggest that insect pathogens in temperate forest systems have the potential to drive long-period population cycles, provided there is some mechanism present (such as host density dependence, vertical transmission, or a long-lived pathogen reservoir) that can overcome the strongly destabilizing effect of the between-season time lag. The models indicate the critical parameters that need to be evaluated in order to assess the role of pathogens in generating cycles. However, a firm answer to the question of whether pathogens cause cycles in forest insects will only come from field studies. What we believe are needed are more studies such as that on gypsy moth (*Lymantria dispar*; Dwyer and Elkington 1993; d'Amico et al. 1996; and included references) where insect-pathogen models of the type developed here and by Dwyer and Elkington (1993) are parameterized from field data.

APPENDIX A

Equations

Equations for the seasonal age-structured model with the functions for density dependence at different stages in the life cycle.

Between Season

$$L(0, \tau + 1) = F \times L(T_s, \tau) \times S(\tau) \times \sigma_L;$$

$$I(0, \tau + 1) = 0;$$

$$P(0, \tau + 1) = P(T_s, \tau) \times \sigma_P.$$

Within Season

$$\frac{d[L(t, \tau)]}{dt} = -\psi[P(t, \tau)] \times L(t, \tau) - D_L(t, \tau) \times L(t, \tau);$$

$$\frac{d[I(t, \tau)]}{dt} = \psi[P(t, \tau)] \times L(t, \tau) - M_I(t, \tau) - d_I \times I(t, \tau);$$

$$\frac{d[P(t, \tau)]}{dt} = \gamma \times M_I(t, \tau) - d_P \times P(t, \tau),$$

where

$$M_I(t, \tau) = \psi[P(t - T_I, \tau)] \times L(t - T_I, \tau) \times \exp(-d_I \times T_I).$$

Version 1

Density dependence acts at the beginning of the season, after host reproduction but before the window of host susceptibility to the pathogen.

$$S(\tau) = \exp[-\alpha \times F \times L(T_s, \tau)];$$

$$D_L(t, \tau) = d_L.$$

Version 2

Density dependence acts during the window of susceptibility to the pathogen.

$$S(\tau) = 1;$$

$$D_L(t, \tau) = a \times L(t, \tau) + d_L.$$

Version 3

Density dependence acts after the window of susceptibility to the pathogen.

$$S(\tau) = \exp [-\alpha \times L(T_s, \tau)];$$

$$D_L(t, \tau) = d_L.$$

APPENDIX B

Discrete-Time Limiting Cases of Seasonal Age-Structured Model

In this appendix, we analyze the limiting case of the seasonal age-structured host-pathogen model (app. A) with $\Phi > 1$, only one round of pathogen infection each season. We analyze in detail version 3 of our model, in which host density dependence occurs on larvae at the end of the season after attack by the pathogen. We have chosen this version because it is directly analogous to May et al.'s (1981) model 3, in which host density dependence occurs after attack by a parasitoid. If we assume that only mortality due to the pathogen occurs during the host's susceptible window ($d_L = 0$), and that any loss of pathogen during the season is negligible ($d_p = 0$), then the equations in appendix A can be integrated over the season (with $P[t, \tau] = P[0, \tau]$) to give the following set of coupled difference equations:

$$\hat{L}(0, \tau + 1) = \hat{F}\hat{L}(0, \tau)\psi[\hat{P}(0, \tau)] \exp \{-\hat{\alpha}\hat{L}(0, \tau)\psi[\hat{P}(0, \tau)]\}; \quad (\text{B1})$$

$$\hat{P}(0, \tau + 1) = \sigma_p\hat{P}(0, \tau) + \sigma_p\hat{L}(0, \tau)\{1 - \psi[\hat{P}(0, \tau)]\}, \quad (\text{B2})$$

where $\psi[\hat{P}(0, \tau)] = \{1 + [\hat{P}(0, \tau)/\hat{k}]\}^{-\hat{k}}$ is the fraction of hosts escaping infection by the pathogen during the season, $\hat{L}(0, \tau) = \hat{v}\gamma L(0, \tau)$, $\hat{P}(0, \tau) = \hat{v}P(0, \tau)$, $\hat{\alpha} = (\alpha/\hat{v}\gamma)$, $\hat{k} = kT_L$, $\hat{F} = F\sigma_L$, and $\hat{v} = vT_L$. This reduction of parameters reveals that the dynamics of the system are controlled by four parameters: $\hat{\alpha}$ is the strength of host density dependence relative to the strength of mortality due to the pathogen (this term also includes the number of pathogen particles released from an infected host), \hat{F} is the effective host fecundity, \hat{k} is the nonlinearity in the pathogen transmission, and σ_p is the carryover of the pathogen between seasons. The parasitoid version of the model is obtained by setting $\sigma_p = 0$. In the absence of pathogens, the equilibrium density of the host is given by

$$\hat{L}^* = \frac{\ln(\hat{F})}{\hat{\alpha}}. \quad (\text{B3})$$

Here, the population dynamics are critically dependent on the host growth rate with the population showing stable equilibrium, limit cycles, or higher-order behaviors depending on particular values of \hat{F} . The nature and thresholds of these bifurcations are well documented (e.g., May 1974a, 1976). In the presence of the disease, the equilibrium density of the host is given by

$$\hat{L}_{(p)}^* = \frac{\ln[\hat{F}\psi(\hat{P}^*)]}{\hat{\alpha}\psi(\hat{P}^*)}, \quad (\text{B4})$$

and the equilibrium density of pathogen or parasitoids can be determined by solving the transcendental equation (B2)

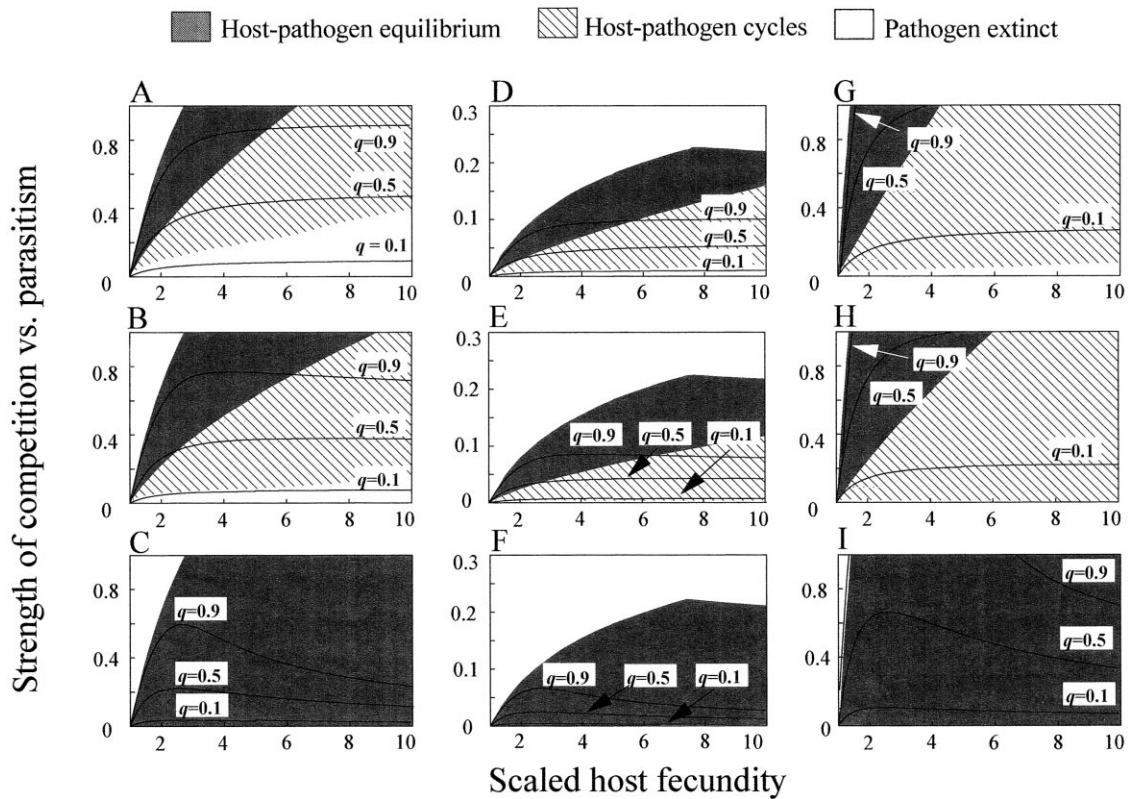


Figure B1: Regions of parameter space in which host-pathogen interaction shows limit cycles, a stable host-pathogen equilibrium, or extinction of the pathogen for the discrete-time limiting case of the seasonal age-structured model. A–C show the dynamical outcome for the parasitoid version (in which all parasitoids die by the end of the season after which they are produced) for different strengths of nonlinearity in the transmission function. A, $\hat{k} = \infty$ (linear transmission). B, $\hat{k} = 5$. C, $\hat{k} = 0.9$. D–I show the dynamical outcome for the pathogen version for different values of pathogen carryover σ_p and for different strengths of nonlinearity in pathogen transmission. D, $\hat{k} = \infty$, $\sigma_p = 0.1$. E, $\hat{k} = 5$, $\sigma_p = 0.1$. F, $\hat{k} = 0.9$, $\sigma_p = 0.1$. G, $\hat{k} = \infty$, $\sigma_p = 0.75$. H, $\hat{k} = 5$, $\sigma_p = 0.75$. I, $\hat{k} = 0.9$, $\sigma_p = 0.75$. Superimposed are contours showing the level of suppression (q) of the host equilibrium by the pathogen.

numerically for \hat{P}^* . The impact of the pathogens on the equilibrium abundance of the host can be measured by calculating q , the ratio of the host equilibrium in the presence of pathogens (\hat{L}_p^*) to the equilibrium density of hosts in the absence of pathogens (\hat{L}^*)

$$q = \frac{\hat{L}_{(p)}^*}{\hat{L}^*}. \quad (\text{B5})$$

In figure B1, we show for both the parasitoid and pathogen versions of the model, the regions of parameter space with four different dynamical outcomes: host-pathogen (or parasitoid) stable limit cycles, stable host-pathogen equilibria, extinction of the pathogen, with the host either cycling or persisting at its carrying capacity. On top of this, we have superimposed contours of q . In the absence of host density dependence, both the parasitoid and pathogen versions do not predict stable limit cycles (Briggs and Godfray 1996). The only two outcomes in this case are divergent oscillations if $\hat{k} > 1$ or a stable host-pathogen equilibrium if $\hat{k} < 1$. The addition of host density dependence can act to stabilize the otherwise divergent oscillations, allowing for persistent host-pathogen limit cycles in some regions of parameter space. In the parasitoid version of the model, with linear transmission (fig. B1A), the persistent limit cycles occur only when the pathogen has little impact on the host population ($q > 0.2$ – 0.4). Moderate nonlinearity in the transmission

rate ($\hat{k} = 5$, fig. B1B) allows persistent cycles to occur with slightly greater host suppression, in regions where q is greater than about 0.1. With $k < 1$, stable host-pathogen limit cycles do not occur, and the only possible outcomes are once again a stable host-pathogen equilibrium or the extinction of the pathogen.

In the pathogen version of the model (eqq. [B1] and [B2]), the presence of cycles and the impact on the host population depend crucially on the amount of carryover of the pathogen between years (fig. B1C, D). If much of the pathogen is lost between seasons, then the region of parameter space with persistent cycles is greatly reduced, and occurs in regions with only moderate host suppression (fig. B1D–F). If there is a large amount of pathogen carryover, persistent cycles occur for a wide range of values of \hat{F} and $\hat{\alpha}$ and can occur even when the host is driven to relatively low densities by the pathogen. As in the parasitoid version, host-pathogen limit cycles are possible only with $k > 1$.

What are realistic values for the transmission rate (ν), number of viral particles released (γ), and the strength of competition (α)? Dwyer (1992) estimates, for one forest insect-virus system (the Douglas fir tussock moth), a value for the transmission rate $\nu = 3.83 \times 10^{-10}$ m²/d, and literature estimates of the number of viral particles released from an infected cadaver are in the range of $\gamma = 10^7$ – 10^{10} particles per host (Dwyer and Elkington 1993). If we assume that hosts are vulnerable to the virus for about 50 d and that host density dependence is such that in the absence of the pathogen hosts at a density of 100/m² would experience a 50% mortality, then, depending on the value of γ , $\hat{\alpha}$ will be in the range of 0.00035–0.35, spanning the range of $\hat{\alpha}$ for which persistent host-pathogen cycles occur.

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