Delayed feedback and multiple attractors in a host–parasitoid system

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Abstract. Continuous-time, age structured, host–parasitoid models exhibit three types of cyclic dynamics: Lotka–Volterra-like consumer-resource cycles, discrete generation cycles, and "delayed feedback cycles" that occur if the gain to the parasitoid population (defined by the number of new female parasitoid offspring produced per host attacked) increases with the age of the host attacked. The delayed feedback comes about in the following way: an increase in the instantaneous density of searching female parasitoids increases the mortality rate on younger hosts, which reduces the density of future older and more productive hosts, and hence reduces the future per head recruitment rate of searching female parasitoids. Delayed feedback cycles have previously been found in studies that assume a step-function for the gain function. Here, we formulate a general host–parasitoid model with an arbitrary gain function, and show that stable, delayed feedback cycles are a general phenomenon, occurring with a wide range of gain functions, and strongest when the gain is an accelerating function of host age. We show by examples that locally stable, delayed feedback cycles commonly occur with parameter values that also yield a single, locally stable equilibrium, and hence their occurrence depends on initial conditions. A simplified model reveals that the mechanism responsible for the delayed feedback cycles in our host–parasitoid models is similar to that producing cycles and initial-condition-dependent dynamics in a single species model with age-dependent cannibalism.

Key words: Parasitoid – Age-structure – Delayed feedback – Population cycles – Multiple attractors

1 Introduction

Parasitoids are insects that lay their eggs on or in the body of an insect of another species (the host). The juvenile parasitoid uses the host for food as it develops, killing the host in the process. Theoretical studies of continuous-time, host–parasitoid systems have revealed that a wide variety of population cycles can result when stage-structure is included in the models. Murdoch et al. [1] took the first step towards classifying these cycles into three types. The first are the long-period cycles that are inherent in most consumer-resource models, for example the Lotka–Volterra model. Murdoch et al. [2] demonstrated that if the parasitoid attacks only juvenile hosts (as is the norm for most species), then a relatively long-lived invulnerable adult host stage can stabilize these long-period cycles. The second type of cycle has a period of approximately a single host generation, and may be found if the adult host is very short-lived, and if the consumer-resource cycles are stabilized by some other process such as density-dependence in the parasitoid attack rate (Godfray and Hassell [3, 4] and Gordon et al. [5]).

The third type of cycles observed in stage-structured host–parasitoid models are delayed feedback cycles that may occur when the "gain" to the searching female parasitoid increases with the age of the juvenile host attacked (Murdoch et al. [6, 1]). Table 1 lists a variety of parasitoid oviposition behaviors that lead to such age-dependent gain. In all of these behaviors, the parasitoid attacks a range of host ages, but attacks on older hosts result in a larger number of female parasitoid offspring than attacks on younger hosts. All of these behaviors have the same effect on host–parasitoid population dynamics, because they induce delayed density-dependence in the parasitoid recruitment rate. This delayed density-dependence comes about in the following way: an increase in the current density of searching female parasitoids increases the mortality rate on younger hosts, which reduces the density of future older and more productive hosts, and hence reduces the future per head recruitment rate of searching female parasitoids. If this delayed density-dependence is sufficiently strong, it can result in delayed feedback cycles that have a period of one to a few host generations.

The current paper focuses on the delayed feedback cycles, and has two aims. First, we explore the generality and robustness of delayed feedback cycles. Our description in the previous paragraph derives from models studied by Murdoch et al. [6, 1] that relied on a crude discretization of the parasitoid size-dependent response to hosts. The juvenile host population was divided into two or more discrete stages

Table 1. Host size dependent parasitoid behaviors. The juvenile host is usually the only source of nutrients that the juvenile parasitoid can use for development, so larger host individuals represent larger packets of resources for their offspring.

Host Size-dependent parasitoid behavior	Description
Sex allocation	Lay male egg in small host; lay female egg in large host (Parasitoids can determine sex of offspring at oviposition. unfertilized eggs = male, fertilized eggs = female)
Host feeding	Host feed on small host; parasitize in large host (Host-feeding is the behavior in which adult female parasitoids feed on the host tissues or fluids to gain nutrients for reproduction or maintenance, killing the host in the process)
Clutch size	Lay few eggs in small hosts; many eggs in large hosts
Juvenile survival	Higher juvenile parasitoid survival in larger hosts
Juvenile development	Faster parasitoid development in larger hosts
Offspring fecundity	Larger hosts yield larger and more fecund female parasitoids

with fixed durations, and the response of the parasitoids differed between the stages. This allowed the models to be formulated as systems of delay-differential equations. In some cases, these discrete stages can be interpreted as different host instars, between which there may in fact be abrupt changes in properties. However, in other systems, the response of the parasitoid may change in a more continuous fashion as the host grows. In the current paper, we formulate a general age-structured model in which the gain to the searching female parasitoid population can change continuously with host age, according to any arbitrary function. We then ask whether the delayed feedback cycles reported by Murdoch et al. [6, 1] are a general phenomenon or simply an artifact of the previous discretization, and we explore the effect of the shape of the host age-specific gain function on the observed dynamics.

The second aim of the paper is to develop a simpler model to act as a caricature of the general model, so that we can explore in detail the mechanism leading to delayed feedback cycles. The study of Hastings [7] and Hastings and Costantino [8], in a single-species stage-structured system, illustrates how developing such a 'model of a model' can be useful for understanding the dynamics that occur in limiting cases of complex models.

2 Continuous gain model

2.1 Model formulation

We consider insect species in which births and deaths can occur continuously, and there is the potential for all life stages to overlap. The equations are summarized in Table 2. Let $J(t, a)$ represent the density of juvenile hosts of age a at time t. All juvenile hosts are subject to mortality due to parasitism at a rate $fP(t)$, where f is the constant parasitoid attack rate and $P(t)$ is the density of adult parasitoids at time t. Mortality of juvenile hosts due to sources other than parasitism is assumed to be constant and density-independent at a per capita rate of δ_J. The dynamics of the juvenile host population can be described by the McKendrick–von Foerster equation:

$$\frac{\partial J(t, a)}{\partial t} + \frac{\partial J(t, a)}{\partial a} = -[fP(t) + \delta_J]J(t, a) \quad \text{for } a \leqq T_H \qquad (1)$$

Table 2. Host age-dependent parasitoid gain model

Variables	$J(t, a)$ = density of juvenile hosts of age a at time t
	$A(t)$ = density of adult hosts at time t
	$R_I(t)$ = recruitment rate into the immature parasitoid stage at time t
	$P(t)$ = density of adult parasitoids at time t
Parameters	T_H = duration of juvenile host stage
	T_P = duration of juvenile parasitoid stage
	ρ = host lifetime fecundity
	β = per capita adult host fecundity = $\frac{\rho}{T_A}$
	δ_J = juvenile host background death rate
	δ_A = adult host death rate
	T_A = average duration of adult host stage = $\frac{1}{\delta_A}$
	σ_I = juvenile parasitoid through-stage survival
	δ_P = adult parasitoid death rate
	f = parasitoid attack rate
Gain function	$g(a)$ = number of female parasitoid offspring of age 0 resulting from an attack on a host of age a
Equations	$\dfrac{\partial J}{\partial t} + \dfrac{\partial J}{\partial a} = -[fP(t) + \delta_J]J(t, a) \quad a \leqq T_H$
	$\dfrac{dA(t)}{dt} = J(t, T_H) - \delta_A A(t)$
	$\dfrac{dP(t)}{dt} = R_I(t - T_P)\sigma_I - \delta_P P(t)$
	where $R_I(t) = fP(t)\int_0^{T_H} g(a)J(t, a)\,da$
Boundary condition	$J(t, 0) = \beta A(t)$

Juvenile hosts that survive to the age of T_H enter the adult host stage which is invulnerable to parasitism. All hosts in the adult stage have the same fecundity and mortality, regardless of age, so we define $A(t)$ to be the total density of adult, with dynamics described by the ordinary differential equation:

$$\frac{dA(t)}{dt} = J(t, T_H) - \delta_A A(t) \tag{2}$$

where δ_A is the constant per capita adult death rate, and the average duration of the adult host stage is $T_A = \frac{1}{\delta_A}$. The constant per capita adult host fecundity is β, so the boundary condition specifying the production of newborn juvenile hosts is:

$$J(t, 0) = \beta A(t) \tag{3}$$

For convenience in the analysis we define $\rho = \beta T_A$ as the expected total number of offspring that an adult female host will produce during her lifetime. By holding ρ constant while varying T_A this allows us to investigate the effects of changing the average duration of the adult host stage without also changing the resultant number of offspring produced per host [2].

   We assume that adult female parasitoids attack and kill juvenile hosts of all ages at a constant per capita rate, f, but that the gain, $g(a)$, to the female parasitoid population depends on the age, a, of the host attacked. Therefore the recruitment rate, $R_I(t)$, into the immature parasitoid stage is equal to the rate at which juvenile hosts are parasitized, weighted by their age-specific gain:

$$R_I(t) = f P(t) \int_0^{T_H} g(a) J(t, a) da \tag{4}$$

Immature parasitoids have a fixed development time (of duration T_P), and only a constant, age- and density-independent death rate occurs during the immature parasitoid stage, so the dynamics of this stage can be subsumed into a delay in the recruitment into the adult female parasitoid stage. The dynamics of the adult female parasitoid population is then described by the delay-differential equation:

$$\frac{dP(t)}{dt} = R_I(t - T_P)\sigma_I - \delta_P P(t) \tag{5}$$

where σ_I is through-stage survival of immature parasitoids.

   To complete the description of the model, we need only to supply a specific function for this host age-specific gain, $g(a)$. It is the effect of the shape of this function that we will investigate in the Sect. 2.3.

Our investigation of the model dynamics uses both analytic and numerical methods. Numerical solutions for the partial differential equations can be obtained using the Escalator Boxcar Train algorithm (de Roos et al. [9]; de Roos [10]), however for some gain functions, the model can be recast as a system of delay differential equations, and in these cases numerical solutions can be obtained more efficiently using any integration program that can handle time delays.[1]

2.2 Equilibria and stability analysis

In this section we give the formulae for equilibrium solutions of the model equations, and outlines the technique used to investigate local stability of the equilibria.

The equilibria (denoted by the * symbol) are obtained in the standard way by setting the time rate of change to zero in the equations in Table 2, yielding:

$$J^*(a) = J^*(0) \exp\left\{-[fP^* + \delta_J]a\right\} \tag{6}$$

with:

$$J^*(0) = \rho\delta_A A^* \tag{7}$$

$$P^* = \frac{\ln(\rho) - \delta_J T_H}{f\,T_H} \tag{8}$$

$$A^* = \frac{\delta_P}{\rho\delta_A f\sigma_I \Phi} \tag{9}$$

where:

$$\Phi = \int_0^{T_H} g(a) \exp\left\{-[fP^* + \delta_J]a\right\} da \tag{10}$$

which can be calculated for any specific functional form of $g(a)$ used.

Juvenile hosts of all ages die at the same rate (due to both attacks by the parasitoid and background causes). Therefore, the PDE describing the rate of change of the juvenile host population can be integrated to yield an expression for the age distribution of juvenile hosts (see [11] for a thorough review of this technique). The maturation rate of

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juvenile hosts to the adult stage at time t is equal to $J(t, T_H)$:

$$J(t, T_H) = \rho\delta_A A(t - T_H)\exp\left\{-\int_{t-T_H}^{t}\left[fP(x) + \delta_J\right]dx\right\} \quad (11)$$

which can be substituted into Eqn. (2).

To determine if the equilibrium is locally stable, we use the standard technique of assuming that the system has been at its equilibrium for all $t \leqq 0$, and asking whether a small perturbation from equilibrium increases or decays through time. If we define:

$$\hat{A} = A(t) - A^*, \qquad \hat{P} = P(t) - P^* \quad (12)$$

then, we can linearize Eqns. (2) and (5) about equilibrium to give:

$$\frac{d\hat{A}(t)}{dt} = \rho\delta_A\sigma_H^*\hat{A}(t - T_H) - \rho\delta_A\sigma_H^* f\int_{t-T_H}^{t}\hat{P}(x)dx - \delta_A\hat{A}(t) \quad (13)$$

$$\frac{d\hat{P}(t)}{dt} = \hat{R}_I(t - T_P)\sigma_I - \delta_P\hat{P}(t) \quad (14)$$

where $\sigma_H^* = \exp\{-(fP^* + \delta_J)T_H\}$, and we define:

$$\hat{R}_I(t) = R_I(t) - R_I^* \quad (15)$$

The next step is to obtain an expression for $\hat{R}_I(t)$. In Eqn. (4) we have already shown that:

$$R_I(t) = fP(t)\int_0^{T_H} g(a)J(t, a)da \quad (16)$$

If $S(t - a, t)$ is defined as the probability that a juvenile host individual born at time $t - a$ survives at least until time t, then:

$$S(t - a, t) = \exp\left\{-\int_{t-a}^{t}[\delta_J + fP(x)]dx\right\}, \quad (17)$$

and,

$$J(t, a) = \rho\delta_A A(t - a)S(t - a, t) \quad (18)$$

So, $R_I(t)$ can be rewritten as:

$$R_I(t) = f\rho\delta_A P(t)\int_0^{T_H} g(a)A(t - a)S(t - a, t)da \quad (19)$$

We can define the perturbation, $\Psi(t - a, t)$ such that:

$$S(t - a, t) = \exp(-ya)\Psi(t - a, t) \quad (20)$$

where for convenience we define $y = fP^* + \delta_J$. From Eqns. (17) and (20) and linearizing, we get:

$$\Psi(t-a, t) = \exp\left\{-f\int_{t-a}^t \hat{P}(x)dx\right\} \simeq 1 - f\int_{t-a}^t \hat{P}(x)dx \quad (21)$$

Thus, $\hat{R}_I(t)$ can be written as:

$$\hat{R}_I(t) = \rho\delta_A f\left\{P^*\hat{\Theta}_1 - P^*A^*\hat{\Theta}_2 + A^*\hat{P}(t)\int_0^{T_H}\exp(-ya)g(a)da\right\} \quad (22)$$

where:

$$\hat{\Theta}_1 = \int_0^{T_H}\exp(-ya)g(a)\hat{A}(t-a)da \quad (23)$$

$$\hat{\Theta}_2 = \int_0^{T_H}\int_{t-a}^t \exp(-ya)g(a)\hat{P}(x)dx\,da \quad (24)$$

Taking the Laplace transformation of linearized equations (13) and (14), and using Eqn. (22), gives us a matrix $\boldsymbol{B}(\lambda)$, such that:

$$\boldsymbol{B}(\lambda)\begin{pmatrix}\hat{A}(\lambda)\\\hat{P}(\lambda)\end{pmatrix} = \begin{pmatrix}0\\0\end{pmatrix} \quad (25)$$

The system is locally stable, with all small perturbations from equilibrium eventually decaying to zero, provided all roots of the following characteristic equation have negative real parts:

$$\det \boldsymbol{B}(\lambda) = 0. \quad (26)$$

Most of the Laplace Transforms involved in arriving at the matrix $\boldsymbol{B}(\lambda)$ are straight-forward, however a few tricks are necessary to derive the transforms of $\hat{\Theta}_1$ and $\hat{\Theta}_2$. If we let:

$$G(a) = \begin{cases}\exp(-ya)g(a) & a \leqq T_H\\0 & a > T_H\end{cases}. \quad (27)$$

then $\hat{\Theta}_1$ can be written as:

$$\hat{\Theta}_1 = \int_0^t G(a)\hat{A}(t-a)da \quad t > T_H \quad (28)$$

which is the standard form for the convolution $G * \hat{A}$, the Laplace transform of which is $G(\lambda)\hat{A}(\lambda)$.

If we let:

$$\Omega(t) = \int_0^t \hat{P}(x)dx \quad (29)$$

then $\hat{\Theta}_2$ becomes:

$$\hat{\Theta}_2 = \int_0^{T_H} \exp(-ya)g(a)\left[\Omega(t) - \Omega(t-a)\right]da \qquad (30)$$

which can be rewritten as:

$$\hat{\Theta}_2 = \Omega(t)\Phi - \int_0^t G(a)\Omega(t-a)da \quad t > T_H \qquad (31)$$

where Φ is simply a number defined in Eqn. (10) for any particular form of function $g(a)$. The second half of the expression is now the convolution $G * \Omega$, which has Laplace transform: $G(\lambda)\hat{P}(\lambda)/\lambda$. The characteristic matrix, $\boldsymbol{B}(\lambda)$ is then:

$$\begin{vmatrix} \lambda + \delta_A - \rho\delta_A\sigma_H e^{(-\lambda T_H)} & \rho\delta_A A^* \sigma_H f\left\{\dfrac{1-e^{(-\lambda T_H)}}{\lambda}\right\} \\[2ex] -\rho\delta_A f P^* \sigma_I e^{(-\lambda T_P)}G(\lambda) & \lambda + \delta_P - \rho\delta_A f A^* \sigma_I e^{(-\lambda T_P)} \times \\[2ex] & \left\{-\dfrac{P^* f\Phi}{\lambda} + \dfrac{P^* f G(\lambda)}{\lambda} + \Phi\right\} \end{vmatrix} \qquad (32)$$

where $\sigma_H = \exp(-yT_H)$.

Stability boundaries in Figs. 2, 3, and 6 were calculated by setting $\lambda = i\omega$ in Eqn. (32) and numerically solving simultaneously the two equations:

$$Re[\det \boldsymbol{B}(\lambda)] = 0; \qquad Im[\det \boldsymbol{B}(\lambda)] = 0 \qquad (33)$$

2.3 Effects of different gain functions

In this section, we investigate the effects on the host–parasitoid dynamics of the shape of the gain function (Fig. 1).

2.3.1 Constant gain
If the gain to the female parasitoid population is independent of the age of the juvenile host attacked, $g(a) = c$ for $0 \leqq a \leqq T_H$ (Fig. 1a), with $c = 1$ we regain the Murdoch et al. [2] model in which the female parasitoid lays a single female egg in a juvenile host of any age that she encounters. A typical local stability boundary for this model is shown in Fig. 2. In this model, the units of time, host density, and parasitoid density can be scaled such that there is no loss of generality in choosing $T_H = 1$, $c = 1$, and $f = 1$. Thus, in Fig. 2, and in all subsequent figures,
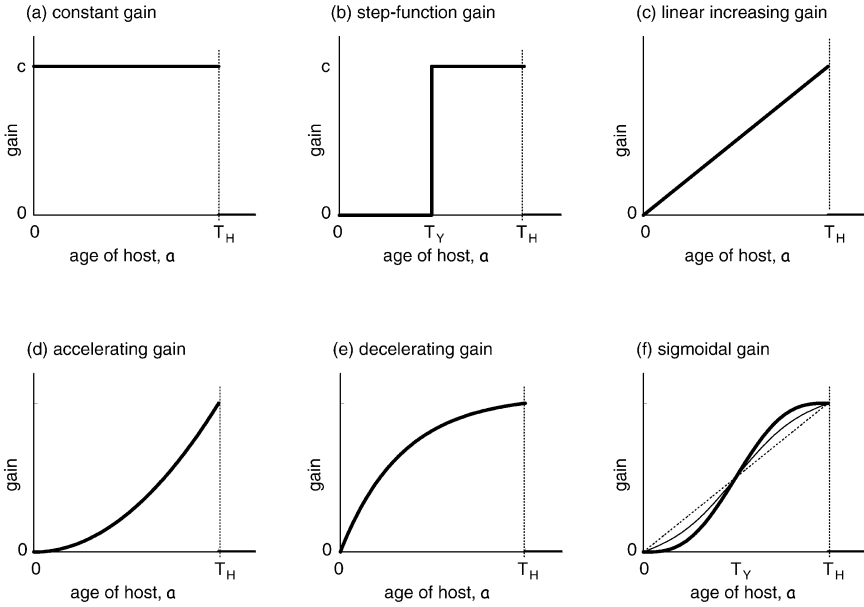
Fig. 1a–f. Potential functional forms for the host age-specific parasitoid gain, g (number of female offspring produced from attacks on host individuals of age a).
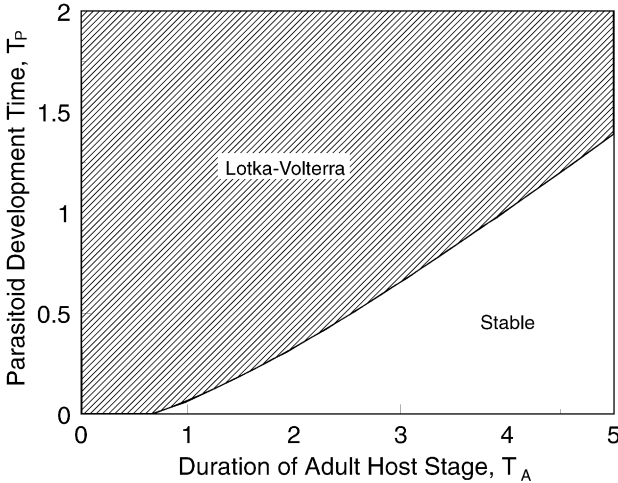

Fig. 2. Constant gain function: typical local stability boundary showing the region of local stability and Lotka–Volterra type cycles as a function of the duration of the invulnerable adult host stage, T_A, and the parasitoid development time, T_P. In this and all other figures, $f = 1$, $T_H = 1$, $\rho = 33$, $\delta_P = 8$, $\delta_J = 0$, and $\delta_I = 0$, except where otherwise noted.

all durations are scaled relative to the juvenile host development time, T_H which is set equal to 1. We will use the same values for the other parameters used by Murdoch et al. [2] as our default parameter set ($\rho = 33$, $\delta_P = 8$, $\delta_J = 0$, and $\sigma_I = 1$). Figure 2 shows that the equilibrium is locally stable when the duration of the invulnerable adult stage is long relative to the juvenile host stage, and when the parasitoid development time is relatively short. Numerical simulations suggest that in the unshaded region of Fig. 2, the equilibrium is globally stable as well, i.e. the system returns to the same stable equilibrium, following large perturbations as well as small ones. The magnitude of the gain, c (the number of female eggs laid on a juvenile host of any size), affects only the equilibrium densities and has no effect on the position of the boundary shown in Fig. 2.

Throughout most of the unstable region, stable limit cycles with a period of few to many host generations occur. These cycles are similar to those observed in many consumer-resource models, and therefore will be referred to as 'Lotka–Volterra cycles'. Numerical simulations show that the period of the cycles increases with T_P, but changes in T_A have little effect on the cycle period. However, stable limit cycles are not universal in this region of parameter space. Close to the vertical axis in Fig. 2 (i.e. when the adult stage is relatively short), two complications arise. First the amplitude of Lotka–Volterra cycles may never stabilize, so that the observed dynamics consist of divergent oscillations of ever increasing period. Second, also in regions of parameter space very close to the Y-axis, roots to the characteristic equation occur that would correspond to single host generation cycles, similar to those observed by Godfray and Hassell [3, 4]. However, these occur only in regions where the Lotka–Volterra cycles are unstable, and are never observed.

As reported in Murdoch et al. [2], other parameters have the effects of shifting the stability boundary shown in Fig. 2 to the right or left, and altering the period of the cycles, but do not change the qualitative results. For example, decreasing ρ or increasing δ_P shifts the boundary to the left. Increasing δ_P decreases the period of the cycles, but altering ρ does not change the cycle period significantly.

2.3.2 Gain increases as a step-function

If $g(a)$ is a step function, $g(a) = cu(a - T_Y)$ for $0 \leqq a \leqq T_H$ (Fig. 1b), we recapture the Murdoch et al. [6] model. In the above expression, and throughout, $u(a)$ represents the Heaviside unit step function defined as:

$$u(a) = \begin{cases} 0 & \text{if } a < 0 \\ 1 & \text{if } a > 0 \end{cases} \tag{34}$$

In this case, there is an abrupt transition with host age, such that attacks on young hosts contribute nothing to the future female parasitoid population, but attacks on old juvenile hosts contribute c new female parasitoid offspring. This could represent parasitoids that lay male eggs or host-feed on young hosts, and only lay female eggs on old hosts (as in Murdoch et al. [6]). This assumes that there are sufficient male parasitoids present that all female parasitoids are mated, and that eggs are not limiting to the parasitoid population. If male parasitoids produced from small hosts are limiting, or if the rate of production of new eggs from materials gained from host-feeding on small hosts limits the rate of oviposition on large hosts, then there can be a significant effective gain from attacks on small hosts. This version of the model assumes as a first approximation that the effective gain from small hosts is negligible.

As described in the introduction, delayed density-dependence in the parasitoid attack rate is induced when the searching parasitoid attacks young hosts, even though the potential gain, in terms of female offspring, is greater from old hosts. The strength of the delayed density-dependence is determined in part by the duration of the young host stage, T_Y from which the gain is 0. Typical local stability boundaries are shown in Fig. 3. If T_Y is relatively short, then the delayed density dependence is relatively weak. This weak density-dependence has a stabilizing effect on the host–parasitoid population dynamics, as evidenced by a shift to the left in the position of the boundary between the regions of a local stability and Lotka–Volterra cycles (comparing Fig. 3a in which $T_Y = 0.1$ with Fig. 2 in which $T_Y = 0$). In the unstable region to the left of the stability boundary in Fig. 3a, the dynamics are again dominated by Lotka–Volterra stable limit cycles.

The delayed density dependence is made stronger as T_Y is increased. This has the effect of further shifting the Lotka–Volterra boundary to the left, and thus can be thought of as stabilizing the Lotka–Volterra instability. However, it also introduces a second region of parameter space in which the equilibrium is unstable. In this region the dynamics are characterized by shorter-period delayed feedback cycles, that have a period of between 1 and a few host generations. Delayed feedback cycles can occur even when the invulnerable adult host stage is very long-lived. They occur when the parasitoid has a short development time relative to that of the host, and can also occur when the parasitoid development time is slightly greater than the host development time. For example, with $T_Y = 0.3$, there are two regions with delayed feedback cycles (Fig. 3b).

When the delayed density-dependence is relatively strong, numerical simulations show that there are multiple attractors in much of the
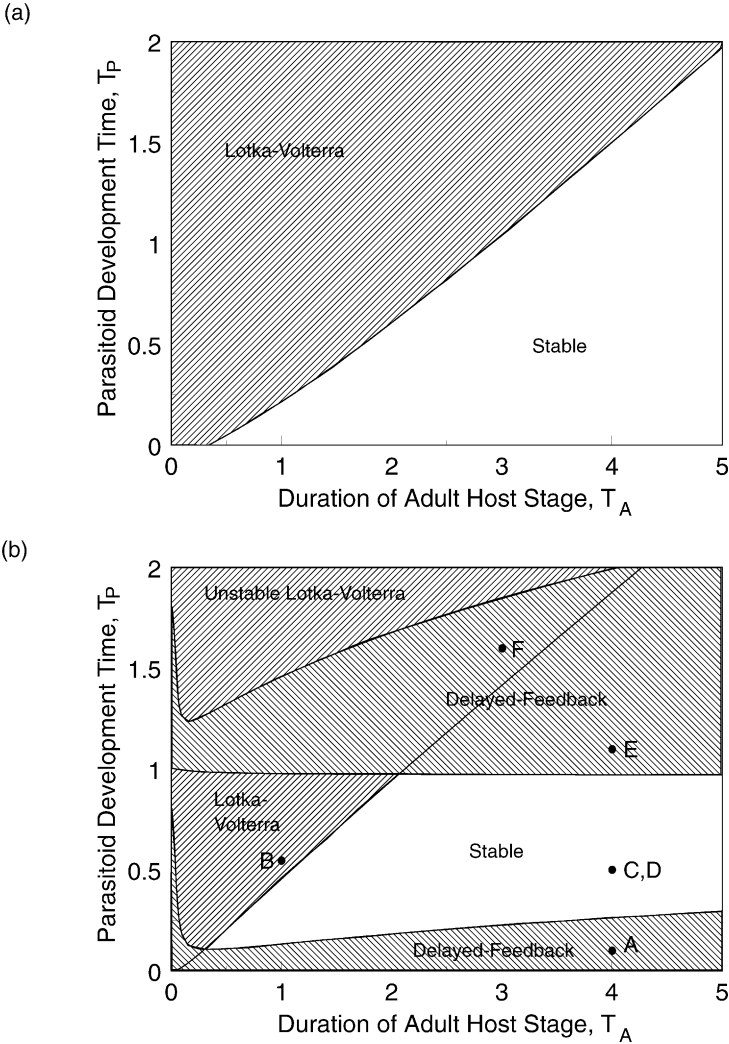
(a)

(b)

Fig. 3. Step-function gain: typical local stability boundaries showing the regions of parameter space with a locally stable equilibrium, Lotka–Volterra type cycles, and delayed feedback cycles. In (**a**) The duration of the young juvenile stage for which there is no gain to the female parasitoid population is very short, $T_Y = 0.1$. In (**b**) The duration of the young juvenile stage is increased to $T_Y = 0.3$, and the points labeled A–F are the parameter values for which the simulations are shown in Fig. 4.

parameters space marked as locally stable in Fig. 3b. In this region, the system returns to the stable equilibrium following a small perturbation from equilibrium, but after some larger perturbations, the system converges instead on a stable limit cycle of the same form as the delayed feedback cycles seen in the unstable region. The region with

multiple attractors may extend even into the region in which local stability analysis predicts that Lotka–Volterra cycles will occur. In this region, perturbations from the Lotka–Volterra-like limit cycle can result in the system settling instead on a delayed feedback limit cycle.

Figure 4 shows the range of dynamics that can be produced from this model, for the parameter values marked A–F in Fig. 3b. The simulation in Fig. 4A illustrates delayed feedback cycles, in a region of parameter space where the equilibrium is locally unstable. In B, the local stability analysis predicts Lotka–Volterra-like cycles. Following a small perturbation from equilibrium, the population fluctuations initially resemble Lotka–Volterra cycles. However, when the magnitude of these fluctuations get somewhat large, the population trajectory is captured by the delayed feedback attactor. Delayed feedback stable limit cycles result, in which the adult host density is always above its equilibrium level. This illustrates that the region of multiple attractors can extend into the area where Lotka–Volterra cycles are predicted. For the parameter values in C and D, the equilibrium is locally stable. Following a small perturbation, C, the population trajectories return to their equilibrium values. Following a large perturbation, D, the trajectory settles on the delayed feedback cycle attractor. Once again, the adult host density in the delayed feedback cycles is always above its equilibrium level. The simulation in Fig. 4E illustrates the second region of delayed feedback cycles, with $T_P > 1$. In Fig. 4F, both the Lotka–Volterra and delayed feedback roots have negative real parts. The population trajectory appears as delayed feedback cycles superimposed on Lotka–Volterra cycles. For very large values of T_P, the Lotka–Volterra cycles are unstable, and diverging oscillations result (top left of Fig. 3b, not shown in Fig. 4).

Figure 5 shows from simulations how the period of the delayed feedback cycles varies with the parasitoid development time, T_P, for different values of the average adult host stage duration, T_A (with $T_Y = 0.3$ as in Fig. 5b). Each point on the graph was obtained by measuring the dominant period from a numerical simulation of the model, following a large perturbation from equilibrium (after the transients had decayed). This figure shows that there are two distinct regions of delayed feedback cycles. The period of the cycles starts out close to 1 host development time for $T_P = 0$ and increases almost linearly with T_P up until $T_P = 1$. At this point, the period drops to 1 and again increases approximately linearly with T_P, but with a smaller slope. The duration of the invulnerable adult host stage (T_A) has little effect on the period of these cycles (Fig. 5). The one exception to this is that with a relatively short adult host stage, $T_A = 1$, the period of
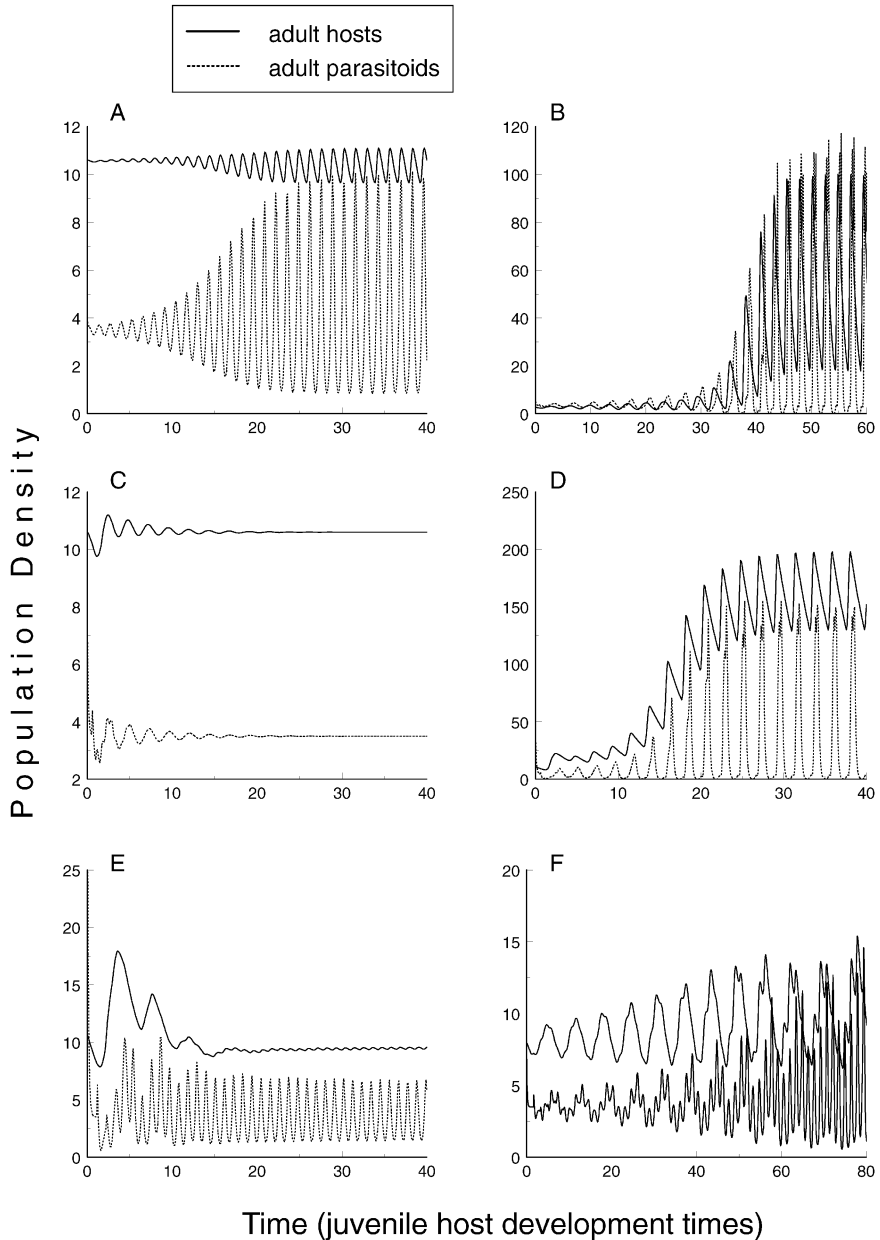
Fig. 4. Step-function gain: simulations of the model with $T_Y = 0.3$ for the points marked A–F in Fig. 3. In all simulations, the initial conditions had the host and juvenile parasitoid populations at to their equilibrium age distributions, and the adult parasitoid density set to a multiple of its equilibrium density, P^*. (**A**) $T_A = 4$, $T_P = 0.1$, $P_0 = 2 \times P^*$, (**B**) $T_A = 1$, $T_P = 0.55$, $P_0 = 1.5 \times P^*$, (**C**) $T_A = 4$, $T_P = 0.5$, $P_0 = 2 \times P^*$, (**D**) $T_A = 4$, $T_P = 0.5$, $P_0 = 10 \times P^*$, (**E**) $T_A = 4$, $T_P = 1.1$, $P_0 = 10 \times P^*$, and (**F**) $T_A = 3$, $T_P = 1.6$, $P_0 = 2 \times P^*$.
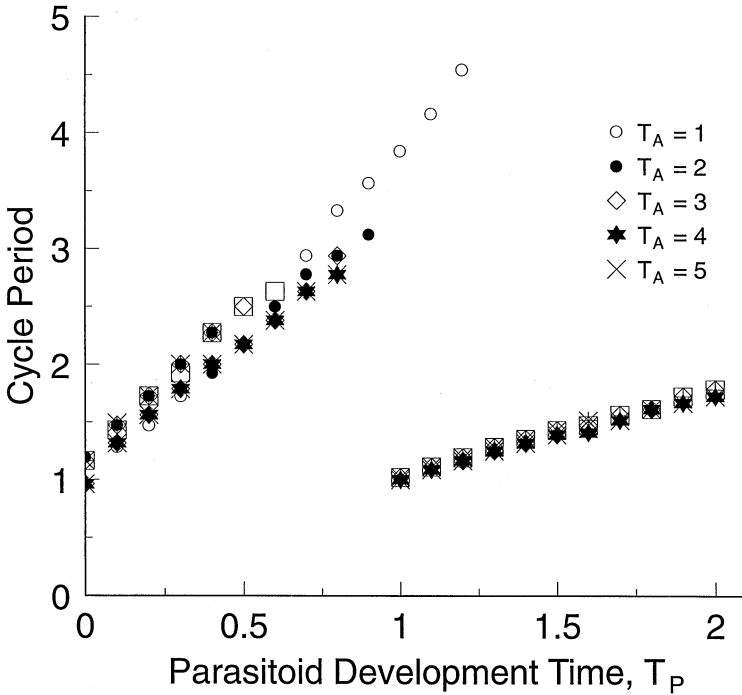
Fig. 5. Period of the delayed feedback cycles as a function of the parasitoid development time, T_P, measured from simulations of the model using 5 different values of the duration of the invulnerable adult host stage, T_A. The simulations were started with the host and juvenile parasitoid at their equilibrium densities and age structure, and with the adult parasitoid density at 100 times its equilibrium value, and with $T_Y = 0.3$.

the cycles increased approximately linearly until $T_P = 1.2$. Above this value of T_P, unstable divergent oscillations occur. Increasing the duration of the young juvenile stage, T_Y, to 0.5 leads to a slight increase in the period of the cycles for any given value of T_P, but the same pattern remains, with the period increasing almost linearly until $T_P = 1$, at which point the period dropped to 1 and increased linearly again. In real host–parasitoid systems, the development time of the parasitoid is usually shorter than the development time of the host ($T_P < 1$), so the upper region of delayed feedback cycles may never be observed.

   In the next sections we show that the delayed density-dependence induced by the larger gain to the future female parasitoid population from attacks on older hosts is not an artifact of the step-function.

2.3.3 Gain increases linearly with host age
The simplest function in which the gain to the future female parasitoid population increases smoothly with the age of the host is the linear

function, $g(a) = ma$, for $a \leqq T_H$ (Fig. 3c). A typical local stability
boundary for this case is shown in Fig. 6a. The slope of the gain
function, m, has no effect on the position of the stability boundary.
A linearly increasing gain function induces some degree of delayed-
density-dependence in the parasitoid attack rate (comparing Fig. 6a
with Fig. 3a). For our default parameters, with a linear increasing gain
the Lotka–Volterra boundary is shifted to the left compared to the
constant gain case, however the delayed density-dependence is not
strong enough to cause delayed feedback cycles. However, delayed
feedback cycles are possible with the linear gain function, if the delayed
density-dependence is made stronger through changes in the value of
other parameters in the model. For example, as the adult female
parasitoids are made shorter-lived (through increasing δ_P), delayed
feedback cycles are possible if the parasitoid also has a relatively short
development time.

2.3.4 Gain increases non-linearly with host age

Next we compare the results of the model with three different non-
linear gain functions to show that the degree of non-linearity in the
gain function affects the strength of the delayed density-dependence.
First, we look at the results when the parasitoid gain is an accelerating
function of the age of the host (e.g. $g(a) = a^n$ for $a \leqq T_H$, shown in
Fig. 1d for $n = 2$). The stability boundaries for $n = 2$ are shown in
Fig. 6b. The delayed density-dependence induced by the accelerating
gain is stronger than that produced by the linear case. The pattern is
the same as that seen for the step-function gain in Fig. 3b, with much of
the parameter space dominated by two large regions of delayed feed-
back cycles. Furthermore, multiple attractors occur in much of the
parameter space, with larger perturbations from equilibrium resulting
in delayed feedback cycles even when the equilibrium is locally stable,
just as in the step-function case.

   If on the other hand the gain to the female parasitoid population
is a decelerating function of the age of the host attacked, then the
strength of the delayed density dependence is reduced compared to the
linear increasing gain case. For example, with $g(a) = 1 - \frac{1 - \exp(-na)}{1 - \exp(-nT_H)}$
(shown in Fig. 1e), the local stability boundaries are shown in Fig. 6c
for the case where $n = 1$ and $n = 5$. The stability boundary is shifted to
the left compared to the case with a constant gain function, so the gain
function has lead to some degree of delayed density-dependence. How-
ever, the boundary is still to the right of the boundary for the linear
increasing gain case. The parameter n determines how fast the gain
reaches its asymptotic value of 1, with larger n making a faster ap-
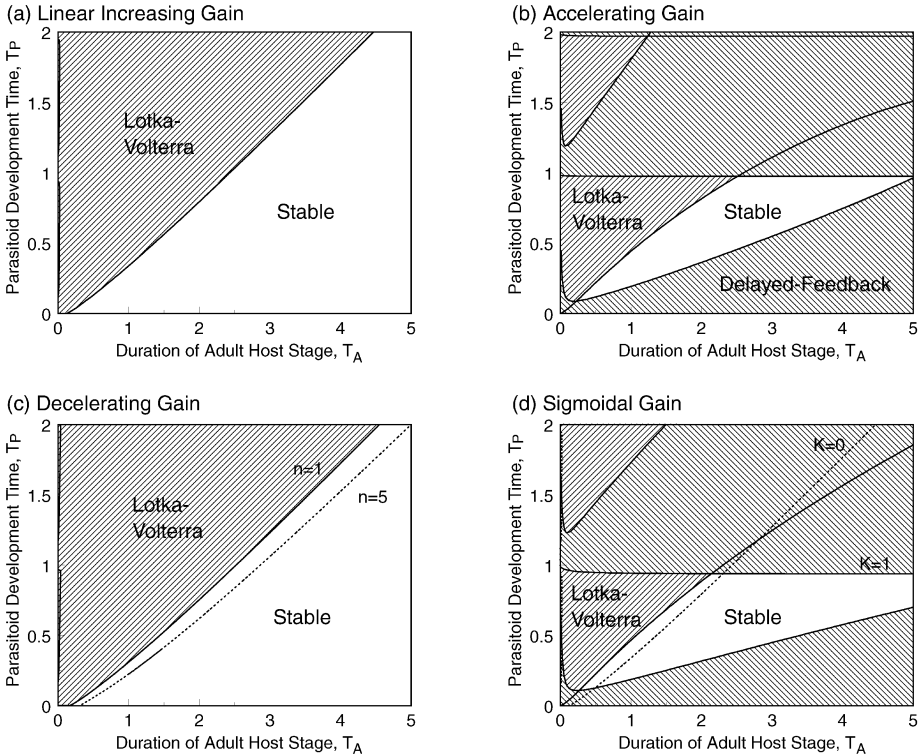proach. Comparison of the two boundaries in Fig. 6c shows that the

Fig. 6a–d. Typical local stability boundaries for the model with four different increasing host age-specific parasitoid gain functions. In (**a**) the gain increases linearly with host age. In (**b**) the gain is an accelerating function of host age: $g(a) = a^2$ for $a \leqq T_H$. In (**c**) the gain is a decelerating function of host age: $g(a) = \frac{1 - \exp(-na)}{1 - \exp(-nT_H)}$ for $a \leqq T_H$, with boundaries shown for $n = 1$, and $n = 5$ (dotted line). In (**d**) the gain is the sigmoidal function of host age: $g(a) = a - \frac{K}{2\pi} \sin(2\pi a)$ for $a \leqq T_H$, with trajectories shown for $K = 0$ (which is equivalent to a linear gain, shown with the dotted line), and $K = 1$.

boundary becomes more like the linear increasing gain case with small n, and more like the constant gain case with large n. As in the case with a linear increasing gain, delayed feedback cycles do not occur for our default parameter set, but are possible if other parameters which increase the strength of the delayed density dependence are changed (for example increasing δ_P).

   The third non-linear gain function that we look at is the sigmoidal function, $g(a) = a - K/2\pi \sin(2\pi a)$ for $a \leqq T_H$, $0 \leqq K \leqq 1$ (shown in Fig. 3f). The parameter K controls the degree of non-linearity in this function, with the linear increasing case being recaptured with $K = 0$, and the non-linearity increasing as K increases to 1. Figure 6d shows the stability boundary for $K = 0$ and $K = 1$. As the function becomes

more non-linear, it more closely resembles the step-function, and the strength of the delayed density-dependence increases. When the delayed density dependence is relatively strong, delayed feedback cycles and multiple attractors occur in much of the parameter space.

Thus, the model in which the gain to the future female parasitoid population increases continuously with host age exhibits all of the same dynamical features as the step-function model. The effect of an accelerating, non-linear, gain function (of which the step-function is an extreme example) is simply to strengthen the delayed density-dependence in the parasitoid attack rate, and thereby strengthen the potential for delayed feedback cycles.

3 Mechanism of delayed feedback cycles

In this section, we use a simplified model to investigate the mechanisms leading to the delayed feedback cycles. Figures 3b, 6b, and 6d show that delayed feedback cycles can occur when the adult host is long-lived relative to the juvenile host development time (i.e. T_A large). We therefore follow the example of Hastings [7] and Hastings and Costantino [8] and approximate the effects of a long-lived adult host stage by assuming a constant input of newborns into the juvenile host stage. This simplified model is particularly appropriate for the study of delayed feedback cycles, as Lotka–Volterra-like consumer-resource cycles are no longer possible, since the action of the parasitoids no longer determines the rate of recruitment into the host population. Delayed feedback cycles appear to result only from periodic variation in the survival of hosts through the juvenile age class, with cyclic recruitment into that class not being necessary. Here we explore further how this arises.

3.1 A simplified model

In this simplified model, we ignore the adult stage, and assume that there is a constant input, R, of newborns of age 0 into the juvenile host stage. Guided by our previous results, we restrict our discussion to the case of a step function representation of the host age-specific gain to the future female parasitoid population. We previously found that the model with a step function gain produces all of the same dynamical features as the model with a continuously increasing gain function.

In the model, H_1 and H_2 represent the densities of young and old juvenile hosts, respectively. Both host stages are attacked by the

parasitoid, but attacks on H_1 contribute nothing to the future female parasitoid population and each attack on H_2 contributes one new female juvenile parasitoid.

As before, we assume that the adult female parasitoids attack and kill juvenile hosts of all ages at a constant per capita rate, f, (with a type I functional response), and that the parasitoids have a constant development time of T_P. The young juvenile host stage has a duration of T_1 and the old juvenile host stage has a duration of T_2, with $T_H = T_1 + T_2$.

This model is formulated as a system of delayed-differential equations with balance equations describing the dynamics of young and old juvenile hosts and adult parasitoids:

$$\frac{dH_1(t)}{dt} = R - M_1(t) - [fP(t) + \delta_J]H_1(t) \tag{35}$$

$$\frac{dH_2(t)}{dt} = M_1(t) - M_2(t) - [fP(t) + \delta_J]H_2(t) \tag{36}$$

$$\frac{dP(t)}{dt} = fP(t - T_P)\, H_2(t - T_P)\sigma_I - \delta_P P(t) \tag{37}$$

where $M_1(t)$ and $M_2(t)$ are the maturation rates out of the young and old juvenile host stages, respectively, at time t:

$$M_1(t) = R \exp\left\{ -\int_{t-T_1}^{t} [fP(x) + \delta_J]dx \right\} \tag{38}$$

$$M_2(t) = M_1(t - T_2) \exp\left\{ -\int_{t-T_2}^{t} [fP(x) + \delta_J]dx \right\} \tag{39}$$

The equilibria for this model are:

$$H_1^* = \frac{R[1 - \exp(-yT_1)]}{y} \tag{40}$$

$$H_2^* = \frac{\delta_P}{f\sigma_I} \tag{41}$$

where for convenience $y = fP^* + \delta_J$. P^* can be found only by numerically solving the transcendental equation (derived from Eqn. (36)):

$$R \exp(-yT_1)\, [1 - \exp(-yT_2)] = y \frac{\delta_P}{f\sigma_I} \tag{42}$$

In the absence of the parasitoid, the density of hosts in each of the juvenile stages would reach a constant value set by the host recruitment rate, the background death rate, and the durations of the two juvenile host:

$$H_2^* = \frac{R \exp(-\delta_J T_1)\,(1 - \exp(-\delta_J T_2))}{\delta_J} \qquad (43)$$

If $\delta_J = 0$, this is equivalent to:

$$H_2^* = RT_2 \qquad (44)$$

A positive equilibrium for both the parasitoid and host occurs if the parasitoid can suppress the density of the old juvenile stage, H_2, below what it would have been in the absence of the parasitoid. If $\delta_J = 0$, this translates into:

$$RT_2 > \frac{\delta_P}{f\sigma_I} \qquad (45)$$

This simplified model reveals how the attacks by the parasitoid interact with the age-structure of the host population to produce delayed feedback cycles.

Using the technique described above, we calculated the typical local stability boundaries shown in Fig. 7 for the simplified model with both species present. In Fig. 7a, delayed feedback cycles occur in regions where the young juvenile host stage takes up an intermediate
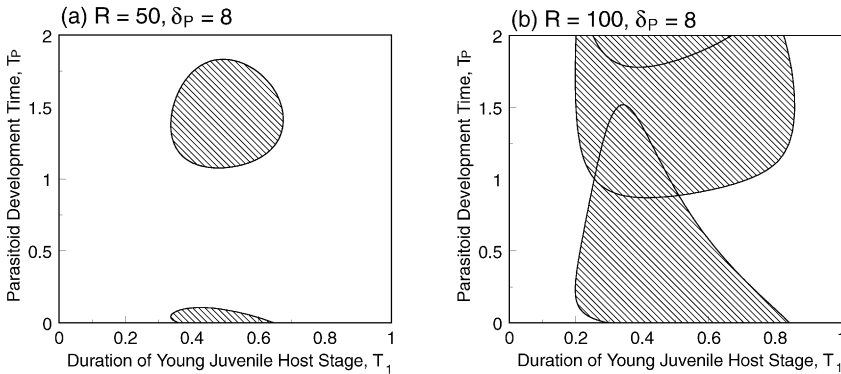

Fig. 7a, b. Typical local stability boundaries for the simplified model with a constant recruitment rate of hosts of age 0, showing the effect of changes in the host recruitment rate, with (**a**) $R = 50$, and (**b**) $R = 100$. In each case, the equilibrium is locally stable in the unshaded region, and local stability analysis predicts delayed feedback cycles in the shaded regions; however multiple attractors exist in the locally stable region such that following a large perturbation from equilibrium delayed-feedback cycles can occur in these regions as well. $\delta_P = 8$, $f = 1$, $\delta_J = 0$, and $\sigma_I = 1$.

fraction of the total host development time. Cycles occur in two areas: a lower region in which the parasitoid development time is short relative to the total host development time, and an upper region in which the parasitoid development time is greater than 1 host development time. Delayed feedback cycles occur over a greater range of parameters for higher values of the host recruitment rate (Fig. 7b). At the top of Fig. 7b, it can be seen that the pattern repeats for higher values of T_P, with a third region of delayed feedback cycles extending just below $T_P = 2$.

As in the full model, changing the values of other parameters can shift the position of the stability boundaries in Fig. 7. Increasing the value of the parasitoid attack rate, f, causes the parasitoid to suppress the density of juvenile hosts further below what it would have been in the absence of the parasitoid, and also has the effect of increasing the region of parameter space in which delayed feedback cycles occur.

In this simplified model, the same pattern of change in the cycle period with increasing T_P (Fig. 8) occurs as was seen in the full model. For the parameters used in Fig. 8, the minimum period of the delayed feedback cycles is approximately 1 host generation (however the minimum period can be affected by changes in other parameters). As T_P increases from 0, the period increases approximately linearly until $T_P = 1$. The period then drops to 1 and increases again at a smaller slope. Increases in the duration of the young juvenile host stage, T_1 also increases the period of the cycles for any given value of T_P (Fig. 8).

To understand the mechanism leading to delayed feedback cycles and the factors determining the period of the cycles it is instructive to look at the age structure within the juvenile host stage. Fig. 9 shows how the densities of hosts of different ages within the juvenile stage change over the course of a number of population cycles. Also shown are the trajectories of the birth rate of immature parasitoids per adult parasitoid ($=fH_2$), the total birth rate of immature parasitoids ($=fH_2P$), and the juvenile and adult parasitoid densities. For illustration, the juvenile host stage in Fig. 9 has been divided into 10 sub-stages of equal duration. Each line on the graph of host density shows the density of juvenile hosts in one of the sub-stages, with the line marked 1 representing the density of juvenile hosts aged 0–0.1, 2 representing hosts aged 0.1–0.2, etc. In Fig. 9, $T_1 = 0.6$, so only the 4 oldest subclasses, shown with solid lines, produce new parasitoids when attacked. Immature parasitoid take $T_P = 0.4$ time units to develop into adults.

Examining the structure of the cycles will show that there are four components to the delay that determines the time between successive
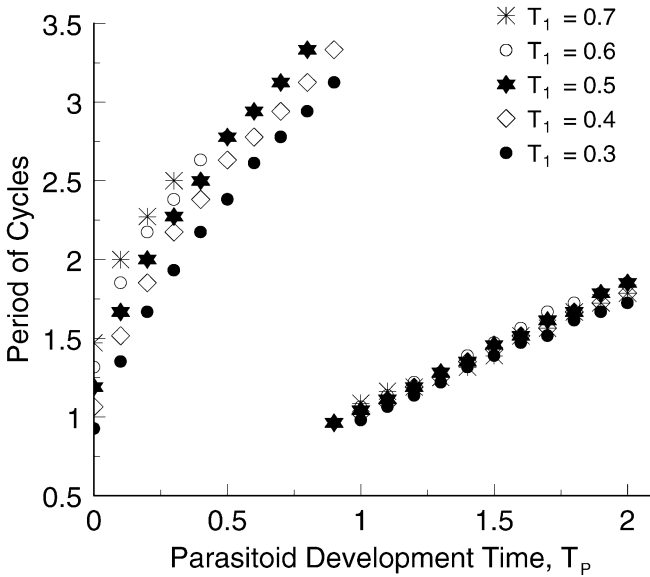
Fig. 8. Period of the delayed feedback cycles in the simplified model as a function of the parasitoid development time, T_P, measured from simulations using 5 different values of the duration of the young juvenile host stage, T_1. The simulations were started with the host and juvenile parasitoid at their equilibrium densities and age structure, and with the adult parasitoid density at 1.1 times its equilibrium value. All other parameters as in Fig. 7b.

peaks in adult parasitoid density (and hence determines the period of the delayed feedback cycles).

The simulation is started with all life stages of the host and the juvenile parasitoid at their equilibrium densities, but with the adult parasitoid density at $10 \times$ its equilibrium. The initial high density of adult parasitoids leads to an immediate reduction in the density of all host stages (point A in Fig. 9). Juvenile parasitoids resulting from these initial attacks develop into adult parasitoids at around time $= 0.5$ (point B). However, these parasitoids become adults at a time when the host densities have not yet recovered from the initial parasitoid attack, so few new parasitoids (point C) result from the offspring of the first peak. The densities of all host age classes remain suppressed until after the adult parasitoid density decays below its equilibrium value (point D). The time that it takes for this to occur is proportional to $\frac{1}{\delta_P}$.

There is always a high, constant, density of newborn hosts entering the first sub-stage. When the parasitoid density drops below equilibrium (point D), the young hosts have a greater than average survival rate, and begin to progress through the age classes. These hosts can be
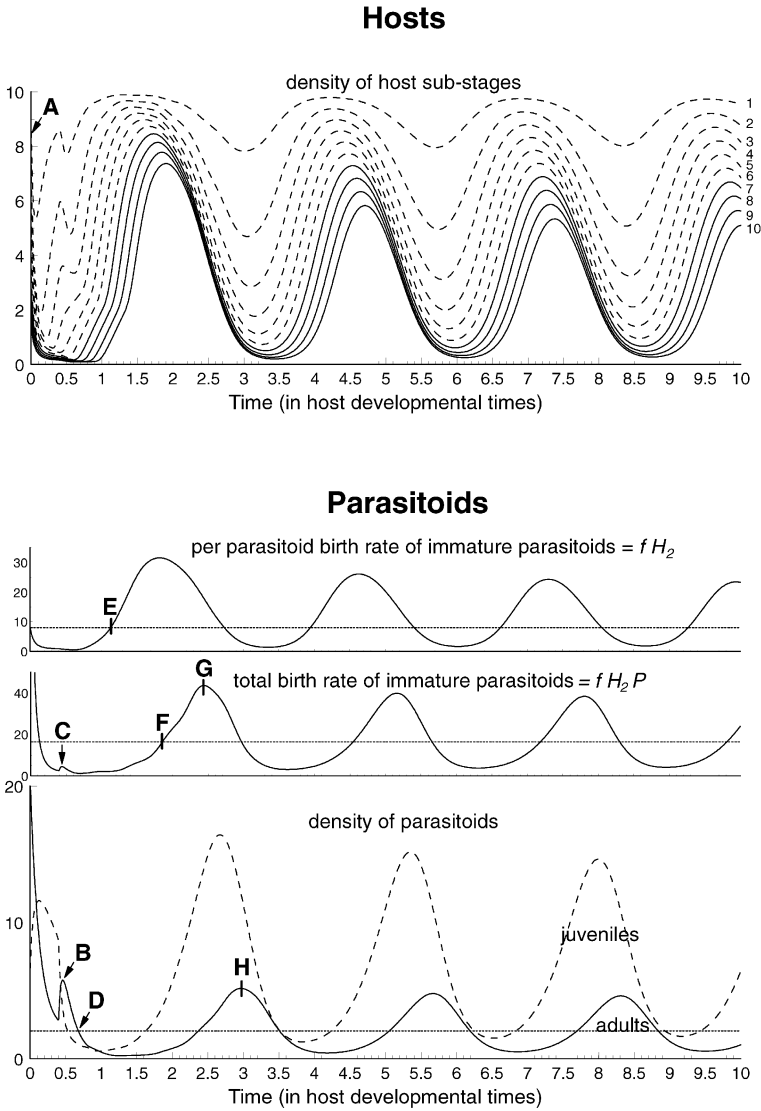
Hosts

Parasitoids

Fig. 9. Simulation of the simplified model, showing the age structure within the juvenile host stage, the per parasitoid birth rate of immature parasitoids, the total birth rate of immature parasitoids, and the density of parasitoids. The total juvenile development time is $T_H = 1$, with $T_1 = 0.6$ and $T_2 = 0.4$. For illustration, in this figure we have divided this stage into 10 sub-stages, each with a duration of 0.1, labeled 1–10 on the graph. Sub-stage 1 shows the density of host individuals of age 0–0.1, sub-stage 2 shows ages 0.1–0.2, etc. $f = 1$, $R = 100$, $T_P = 0.4$, $\delta_P = 8$, $\delta_J = 0$, and $\sigma_I = 1$. The simulation is started with H_1, H_2, and I all at their equilibrium age distributions, and with $P = 10P^*$. Lettered points are referred to in text.

used for the production of new parasitoids only after they reach the old juvenile host stage, H_2. This takes T_1 time units. Thus, a greater than average per parasitoid birth rate of immature parasitoids (point E) results when the density of individuals in the H_2 stage gets above its equilibrium, at approximately T_1 time units after point D in Fig. 9.

At point E the density of juvenile parasitoids starts to increase, however, the adult parasitoid density is still low and decreasing, with the first parasitoids becoming adults only after T_P time units. The total birth rate of immature parasitoids is fH_2P, therefore, even though the per capita parasitoid birth rate (fH_2) is high, there is a delay before the total parasitoid birth rate increases above its equilibrium value (point F). This delay is determined by the time that it takes the adult parasitoid density to start to increase, which depends directly on the parasitoid development time, T_P. This delay depends also on the parasitoid attack rate, f, with high values of f decreasing the delay. (This is not true in the complete model, where the densities of H_2 and P both scale directly with f.) The total birth rate of immature parasitoids, fH_2P, reaches its peak (point G) as H_2 is decreasing and P is increasing.

The maximum adult parasitoid density, and therefore the maximum per capita death rate on hosts, reaches its maximum T_P time units later (point H). As with point B, this peak in adult parasitoid density occurs at a time when the H_2 density is still suppressed, so few new parasitoid offspring result directly from this peak, and the cycle repeats.

Thus we can see that there are four components of the lag that determines the period of the delayed feed back cycles: (a) a term proportional to $\frac{1}{\delta_P}$, (b) T_1, (c) a term that depends on T_P along with a number of other factors including the parasitoid attack rate and the host recruitment rate, and (d) T_P. The parasitoid development time enters into the calculation of the cycle period twice: first it determines the time that it takes after hosts have entered the H_2 stage for the adult parasitoid density to start to increase, and second it determines the time between the peak in the total birth rate of immature parasitoids and in the subsequent peak in adult parasitoid density.

When the parasitoid development time is long relative to the lifespan of the adult parasitoid, i.e. in any of the upper regions of delayed feedback cycles in Fig. 7, a slightly different scenario occurs. Figure 10 shows a simulation in which $T_P = 1.5$, starting again with the host and juvenile parasitoids at their equilibrium densities and the adult parasitoid density at 10× equilibrium. As before, the high density of parasitoids immediately drives down the density of all host age classes (point A in Fig. 10). However in this case, these initial attacks result in new adult parasitoids after 1.5 time units (point B). By this time, the
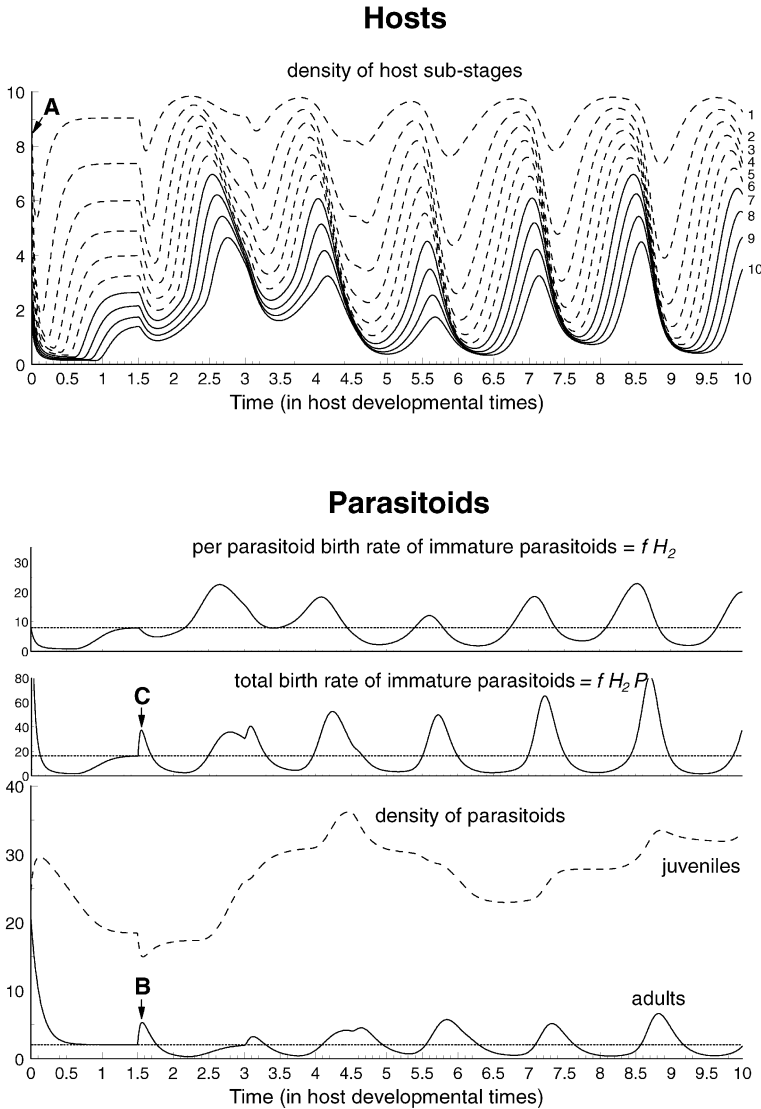
Hosts

density of host sub-stages

Parasitoids

per parasitoid birth rate of immature parasitoids = $f H_2$

total birth rate of immature parasitoids = $f H_2 P$

density of parasitoids

Fig. 10. As in Fig. 9, except $T_P = 1.5$.

host density has recovered from the initial attacks, and a peak in the birth rate of immature parasitoids results from the attacks by the offspring of the initial peak (point C). Thus, the offspring resulting from any peak in the density of parasitoids emerge as adults after the previous parasitoid peak has decayed, and after the host density (and in particular the density of old juvenile hosts, H_2) has been replenished through recruitment. The cycles seen at high values of T_P, and high δ_P, are true parasitoid generation cycles, with a period approximately equal to T_P.

4 Connection with single-species models

There is an obvious analogy between the host-parasitoid model presented here and the single species cannibalism model presented by Hastings [7] (see also Hastings and Costantino [8]). The Hastings model considers an insect system such as Tribolium, that has a long-lived adult stage, with a stage-structured juvenile stage, such that older juveniles (larvae) cause a substantial mortality on younger juveniles (eggs) through cannibalism. Our simplified model uses the same approximation as Hastings, replacing the long- lived adult stage with a constant input of juveniles.

In the cannibalism model, a large cohort of individuals entering the old juvenile stage causes a high mortality on young juveniles, suppressing their density. Because there is little mortality during the old juvenile stage, the suppressing effect lasts until the large age cohort matures out of the old juvenile stage. At that time, the youngest age cohorts can be replenished through recruitment. This results in cycles that have a period that is approximately equal to the insect's development time.

In our host-parasitoid model, instead of the high densities of old juveniles directly leading to heavy mortality on young juveniles, they lead to high densities of parasitoid after a developmental delay of T_P, which then leads to heavy mortality on young juveniles. This results in an increase in the period of the cycles that is proportional to T_P. Another important difference between the models is that in the single-species model, the suppressing effect of a large age cohort of old juvenile hosts on the density of young juvenile hosts lasts T_2 time units, because there is little mortality on the old juvenile stage. In the parasitoid-host model, the old juvenile hosts suffer mortality due to the parasitoids at the same rate as the young juvenile hosts, so the density of the older age cohorts in the H_2 stage are suppressed substantially. Therefore the suppressing effect on the density of the young juvenile stage lasts as long as the adult parasitoid density remains high, i.e. for a time inversely proportional to the parasitoid death rate, δ_P.

Another similarity between our parasitoid-host model and Hastings' cannibalism model is the presence of multiple attractors in both models. Hastings found that in much of the locally stable region of parameter space in his model, limit cycles with a period of one development time could result following a large perturbation. Hastings was able to prove for his single species model that the bifurcation leading to the unstable region was subcritical, using the technique of Diekmann and van Gils [12]. This analogous calculation is prohibitively difficult for our two-species model. However, the proof by Hastings enhances the plausibility of our numerical findings of multiple attractors.

5 Discussion

In this paper we developed a general model to investigate the dynamical effect of a common feature of many host age-dependent parasitoid oviposition behaviors: the gain to the parasitoid population (in terms of the number of female offspring produced) increases with the age of the host attacked. We find that a gain function that increases with host age leads to delayed density-dependence in the parasitoid birth rate which, if sufficiently strong, can induce delayed feedback cycles. These findings confirm the results of earlier models (Murdoch et al. [6, 1]) in which the gain increased as a step-function with host age. The range of dynamics produced, including delayed feedback cycles and multiple attractors, was the same for the model with a gain function that increased continuously with host age as for the step-function model, and therefore was not an artifact of the earlier step-function formulation. The shape of the gain function determines only the strength of the density dependence and the likelihood of observing delayed feedback cycles.

The delayed feedback cycles produced from host age-dependent parasitoid oviposition behavior in our model can occur when the adult hosts are very long-lived relative to their juvenile development time. This is also true of the cycles produced from age-dependent cannibalism in Hastings' [7] single-species model. The cycles in both of these models result from periodic variation in the survival of cohorts through the juvenile class caused by the age-dependent processes. Periodic variation in the recruitment into the juvenile stage is not required. This is in sharp contrast with the mechanism that can produce generation cycles only when the adult host stage is very short-lived in the host-parasitoid models of Godfray and Hassell [3, 4] and Gordon et al. [5]. The cycles in these models are produced by the action of a parasitoid whose development time is a fraction of that of its host. The host and parasitoid populations become synchronized in such a way that only hosts within a dominant age cohorts each generation are able to survive to reproduce. Apparently discrete host generations are maintained because the adult hosts live only for a short period of time, and most offspring are produced only within a short pulse. Long-lived adult hosts, on the other hand, would produce their offspring distributed over a relatively long period of time, smearing out the generational structure. Thus, in the Godfray and Hassell and the Gordon et al. models, periodic variation in recruitment as well as periodic variation in host juvenile survival are required for maintenance of the generation cycles.

Finally, we comment on the explanatory power of simplified 'models of models' for teasing out mechanisms. Hastings' [7] single-species

model exemplified how this approach could be used to understand the dynamics that occur on a time scale that is much shorter than the duration of the adult lifespan. In the current paper, we use a similar simplified model to investigate how host age-specific oviposition behavior by parasitoids can lead to delayed feedback cycles when host recruitment is constant and therefore when classic Lotka–Volterra consumer-resource cycles are not possible. These sharply defined limiting cases, made possible by simplified versions of models, provide insights that allow us to begin to understand the results of complex models.

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