Developmental variability and stability in continuous-time host–parasitoid models

Dashun Xu [a], John D. Reeve [b],[*], Xiuquan Wang [a], MingQing Xiao [a]

[a] *Department of Mathematics, Southern Illinois University, Carbondale, IL 62901, USA*
[b] *Department of Zoology, Southern Illinois University, Carbondale, IL 62901, USA*

ARTICLE INFO

ABSTRACT

Insect host–parasitoid systems are often modeled using delay-differential equations, with a fixed development time for the juvenile host and parasitoid stages. We explore here the effects of distributed development on the stability of these systems, for a random parasitism model incorporating an invulnerable host stage, and a negative binomial model that displays generation cycles. A shifted gamma distribution was used to model the distribution of development time for both host and parasitoid stages, using the range of parameter values suggested by a literature survey. For the random parasitism model, the addition of biologically plausible levels of developmental variability could potentially double the area of stable parameter space beyond that generated by the invulnerable host stage. Only variability in host development time was stabilizing in this model. For the negative binomial model, development variability reduced the likelihood of generation cycles, and variability in host and parasitoid was equally stabilizing. One source of stability in these models may be aggregation of risk, because hosts with varying development times have different vulnerabilities. High levels of variability in development time occur in many insects and so could be a common source of stability in host–parasitoid systems.

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1. Introduction

Insect host–parasitoid and predator–prey systems are often modeled using delay-differential equations, because this framework lends itself to the structure of insect life cycles and the time delays inherent in insect development. For example, a host–parasitoid system can often be represented by several host equations representing vulnerable and invulnerable stages as well as adult hosts with time delays associated with the development of these stages, while the parasitoid has equations for immature and adult parasitoids that also incorporate delays (e.g. Murdoch et al., 1987; Godfray and Hassell, 1989; Gordon et al., 1991; Reeve et al., 1994; Briggs et al., 2000; Wearing et al., 2004b). Among other findings, these models have shown that invulnerable host stages can be stabilizing and that parasitoids can induce generation cycles (cycles with a period of one host generation) in the host as well as longer period cycles. Also shown to be stabilizing are aggregation (or heterogeneity) in the risk of parasitism and interference among searching parasitoids.

A simplifying feature in many of these models is a fixed development time for the immature host and parasitoid stages, meaning that each individual takes the same time to complete development

in a particular stage. A few studies have examined scenarios where development time has a distribution. Smith and Mead (1974) simulated a model with two age classes for the host (prey), with host development modeled using a fixed time delay, or exponential and gamma distributions. Persistence was most likely for an exponential development time in combination with an invulnerable host stage. Hastings (1983, 1984) also found that exponential development times were more stable than fixed delays in predator–prey models with age structure and invulnerable periods. (In these models, if predator or prey develops into the next stage at a fixed rate this implies an exponential distribution of development time.) Briggs et al. (1993) found that variability in host development increased the likelihood of coexistence in a host–parasitoid model with two competing parasitoid species. Wearing et al. (2004a) found that generation cycles could be replaced by longer period cycles in a model of the *Plodia interpunctella–Venturia canescens* host–parasitoid system, as variability in host or parasitoid development time (modeled using a gamma distribution) was increased. The estimated values of the gamma shape parameter n were quite large, however, suggesting that natural variability in development time was low for this system (as $n \to \infty$ variability approaches zero). Eurich et al. (2005) examined the effect of distributed delays in prey development for Lotka–Volterra predator–prey systems and similar food chains and webs. They found that stability and persistence were strongly enhanced by distributed development, approaching the results found for systems without time

* Corresponding author.
E-mail address: jreeve@zoology.siu.edu (J.D. Reeve).

delays. Nakamichi et al. (2008) simulated the interaction between the stored grain pest *Callosobruchus maculatus* and its parasitoid *Heterospilus prosopidis*, and observed that persistence was enhanced by variability in host development. Variability was also relatively low in this system (*n* was large), but was still sufficient to influence the dynamics. These findings suggest that developmental variability could have substantial effects on persistence and stability in host–parasitoid models, but these effects also depend on the level of variability, which was only quantified in two of these studies. That these are predator—prey systems also appears important—in contrast, Blythe et al. (1984) observed little effect of distributed development on the stability of the single-species system.

We explore here the effects of distributed development in host and parasitoid stages on the stability of these systems, using as a starting point the host–parasitoid model developed by Godfray and Hassell (1989), for both random and negative binomial search in the parasitoid. This model includes age structure in both host and parasitoid as well as invulnerable host stages, with sufficient detail to be representative of many natural systems. Parameter values for this model were selected to be similar to those used in Godfray and Hassell (1989) or in Murdoch et al. (1987), which in turn were based on real host–parasitoid systems. A shifted gamma distribution was used to model the distribution of development time for both host and parasitoid stages. Previous theoretical work indicates that stability often depends on the amount of developmental variability, and so we also conducted a literature survey of insect development studies. The results of this survey provided a plausible range of parameter values for the gamma distribution. Our results indicate that stability is enhanced by distributed development in host or parasitoid, although the host effect appears stronger. Both generation and longer-period cycles are less likely when there is distributed development, relative to models with a fixed development time. These results occur for parameter values of the shifted gamma distribution that appear common in nature.

2. Survey of distributed development

The great majority of studies that have examined the distribution of development time in insects use the Weibull distribution, fitted by the method described in Wagner et al. (1984). The analysis first normalizes the data by dividing development times by the mean or median development time for each rearing temperature, thus allowing the data for different temperatures to be combined in a single analysis. A three-parameter version of the Weibull distribution is typically employed, involving shape, scale, and shift parameters (β, η, and γ, respectively), although some papers assume that $\gamma = 0$. The probability density for the Weibull is given by the equation

$$ef(t) = \frac{\beta}{\eta} \left(\frac{t - \gamma}{\eta} \right)^{\beta - 1} e^{-[(t-\gamma)/\eta]^{\beta}} \tag{1}$$

where $\beta > 0$, $\eta > 0$, and $t > \gamma$. Its corresponding distribution function is

$$F(t) = 1 - e^{-[(t-\gamma)/\eta]^{\beta}}, \tag{2}$$

(Johnson et al., 1994). Nonlinear regression is then used to fit the distribution function for the Weibull to the empirical distribution of development times. We located a total of 32 studies that used this methodology by searching for papers that cited Wagner et al. (1984). For modeling purposes, we were most interested in the amount of developmental variability in single immature stages, not for combined stages such as egg-adult development. After eliminating combined stages, we were left with Weibull parameter

estimates for 23 species and 65 stages (usually several per species), across 21 studies (Appendix A). The majority of the estimates were obtained from laboratory studies of development (59 of 65 stages). We note that only two species (5 stages) involved natural enemies, one predator (*Calosoma sycophanta*) and one parasitoid (*Cotesia melanoscela*) of the gypsy moth, *Lymantria dispar*. The remainder were typically pest species of some economic importance.

Continuous-time models that include developmental variability often use the gamma distribution to model development times, because it has a number of mathematical advantages. However, the gamma distribution is seldom used in empirical studies of development time, the only examples we could find being Blythe et al. (1984) and Nakamichi et al. (2008) (which examined total development time, not single stages), and Wearing et al. (2004a). We therefore developed a method of converting Weibull parameter values to the analogous ones of the gamma distribution. For each triplet of Weibull parameter values, we used the `rand` function in SAS 9.1 (SAS Institute, Inc., 2003) to generate a data set with $n = 5000$ observations from the Weibull distribution. We then fitted the gamma distribution to the simulated data using maximum likelihood as implemented in PROC UNIVARIATE (SAS Institute, Inc., 2003) The gamma distribution also has shape, scale, and shift parameters (n, c, and τ) and can take shapes similar to the Weibull distribution. The probability distribution of the gamma distribution is given by

$$f(t) = \frac{c^{n+1}}{\Gamma(n+1)} (t - \tau)^n e^{-c(t-\tau)} \tag{3}$$

where $n > -1$, $c > 0$, and $t > \tau$. Note that for $n = 0$ the gamma reduces to the exponential distribution. This parameterization of the distribution is the one we employed in the host–parasitoid models (see below). We assumed that the threshold parameter for the gamma distribution was equal to the Weibull parameter value to simplify the fitting process. The match between the gamma distribution and the simulated Weibull data was generally quite close except for very large values of β (the Weibull shape parameter), for which the Weibull distribution has a slightly left-skewed shape that cannot be matched by the gamma distribution. The net result was a triplet of gamma parameter values (n, c, and τ) corresponding to each triplet of Weibull parameter estimates.

The results of the literature survey suggest that small values of the gamma shape parameter n are quite common (Fig. 1(A)). The distribution had two peaks, with one at low values ($2 < n < 4$) and a second smaller peak at large values ($n > 18$). Previously published estimates of the gamma shape parameter (Wearing et al., 2004a) were clustered around the second peak (Fig. 1(A)). The shift parameter τ showed a broad spectrum of values (Fig. 1(B)) between 0 and 1. Note that τ in this case actually represents a **proportion** of the total development time because the Wagner et al. (1984) procedure uses normalized development time. Based on our results, we felt justified in using some relatively small n values, and a range of τ values, in the stability analyses discussed below. One drawback of our survey is that it examines developmental variability in general, not for particular host–parasitoid systems and stages, but these were the data available.

3. Models and analyses

As in Godfray and Hassell (1989), we consider an idealized host–parasitoid life cycle (Fig. 2). The life cycle of the host can be divided into three juvenile stages and an adult stage, with densities H_1, H_2, H_3, and H_4. Parasitism only occurs in the second host stage while the rest are invulnerable, although there may be density-independent mortality in all stages. The parasitoid life cycle is divided into juvenile and adult stages with densities P_1 and P_2. Parasitism by adult parasitoids converts juvenile hosts into juvenile parasitoids, which eventually mature into adult parasitoids (Fig. 2).
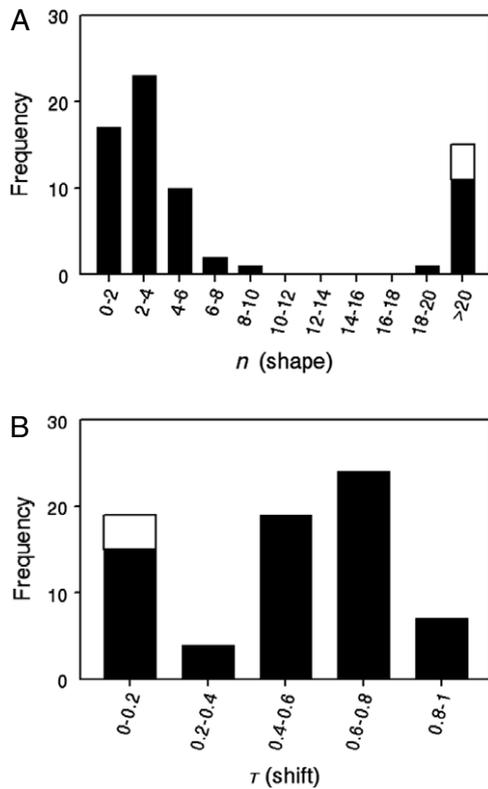
Fig. 1. Frequencies of the shape parameter n (A) and threshold τ (B) for the shifted gamma distribution, in a survey of papers examining variability in insect development time. Open bars are the results from Wearing et al. (2004a), which directly fitted the gamma distribution, while the remainder were obtained from studies using the Weibull distribution (see text for details).
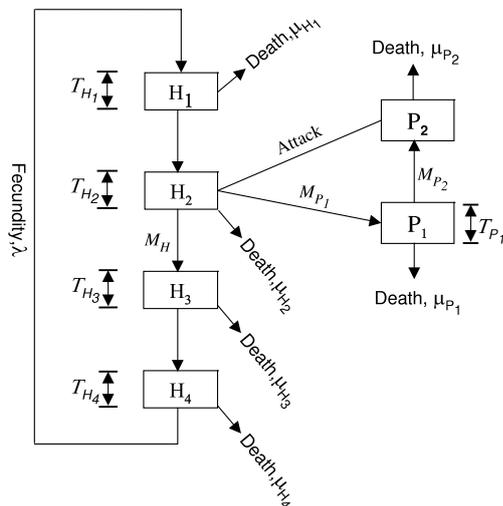

Fig. 2. Idealized host–parasitoid life cycles as described in Godfray and Hassell (1989). There are three juvenile stages in the host, with densities H_1, H_2, and H_3, while H_4 is the adult host stage. The parasitoids have two stages (juvenile and adult) with densities P_1 and P_2. Parasitoid attack converts hosts in the H_2 stage into juvenile parasitoids, while adult hosts reproduce at rate λ and generate new juvenile hosts. Density-independent mortality occurs in each host and parasitoid stage at rate μ_* ($*$ equals H_1, H_2, etc.)

Using techniques for modeling age-structured populations (see Nisbet and Gurney, 1983; Gurney et al., 1983; Murdoch et al., 1987; Godfray and Hassell, 1989), we obtain the following equations describing the dynamics of the system:

$$\frac{dH_2(t)}{dt} = \lambda H_4(t - T_{H_1})F_{H_1} - M_{P_1}(t) - M_H(t) - \mu_{H_2}H_2(t),$$

$$\frac{dH_4(t)}{dt} = M_H(t - T_{H_3})F_{H_3} - \mu_{H_4}H_4,$$

$$\frac{dP_1(t)}{dt} = M_{P_1}(t) - M_{P_2}(t) - \mu_{P_1}P_1, \tag{4}$$

$$\frac{dP_2(t)}{dt} = M_{P_2}(t) - \mu_{P_2}P_2.$$

Note that the host can be modeled using equations for only H_2 and H_4, because the host is assumed to pass through the H_1 and H_3 stages at fixed time periods during which there are no interactions with the parasitoid or reproduction. In this model, the instantaneous death rate for each stage μ_* ($*$ stands for H_1, H_2, etc.) is assumed to be constant. T_* represents the mean time period of the stage (see Fig. 2) and F_* is the probability of surviving until the end of the stage given by $F_* = e^{-\mu_* T_*}$. The instantaneous per capita birth rate of adult insects is λ, and hence the number of insects entering H_2 stage per unit time is the product of the rate of egg laying T_{H_1} time units ago, which is equal to $\lambda H_4(t - T_{H_1})$, and the survival probability F_{H_1} from H_1 stage. The insects entering H_2 may die due to the instantaneous mortality μ_{H_2}, be attacked by parasitoids at rate $M_{P_1}(t)$, or mature into the H_3 stage at rate $M_H(t)$. The recruitment rate of the H_4 stage (host adults) is the number of insects which enter H_3 stage T_{H_3} time units ago and survive until the end of the stage. These insects die at rate μ_{H_4}, whose reciprocal $1/\mu_{H_4}$ is assumed to be the mean lifespan T_{H_4} of adult hosts. The first term $M_{P_1}(t)$ in the equation for P_1 is the recruitment rate of parasitoids into the P_1 stage. The juvenile parasitoids die at rate μ_{P_1} or mature into adult parasitoids at rate $M_{P_2}(t)$. The adult parasitoids die at rate μ_{P_2}, with $T_{P_2} = 1/\mu_{P_2}$ their mean lifespan. Our formulation for adult lifespan differs in one detail from Godfray and Hassell (1989), who assumed a finite lifespan for adult hosts and parasitoids. All model parameters are listed in Table 1.

In this paper, we consider two variants of the above model with different types of parasitoid search and stabilizing mechanisms: (1) random search with a long and stabilizing invulnerable host adult stage (H_4), similar to Murdoch et al. (1987), and (2) parasitoid search according to the negative binomial model (May, 1978; Chesson and Murdoch, 1986), which is also stabilizing if the level of parasitoid aggregation is sufficiently high. Negative binomial search in combination with a ratio of parasitoid to host generation times of approximately 0.5 or 1.5 can also lead to generation cycles (Godfray and Hassell, 1989). For each variant, we study the effect of distributed development in both host and parasitoid on the stability of the system.

3.1. Random search and an invulnerable host stage

We begin by assuming that parasitoids search for hosts randomly and independently at rate a so that

$$M_{P_1}(t) = aP_2(t)H_2(t). \tag{5}$$

We now specify the forms of M_H and M_{P_2} for fixed development times in both host and parasitoid, and then gamma distributions of development time. An insect from H_1 stage will stay in H_2 stage for a fixed time period T_{H_2} and then enter H_3 stage. Therefore,

$$M_H(t) = \lambda H_4(t - T_{H_1} - T_{H_2})F_{H_1}e^{-\int_{t-T_{H_2}}^{t}(aP_2(x)+\mu_{H_2})dx}. \tag{6}$$

Similarly, $M_{P_2}(t)$ takes the following form:

$$M_{P_2}(t) = aP_2(t - T_{P_1})H_2(t - T_{P_1})F_{P_1}. \tag{7}$$

The model system thus consists of Eqs. (4)–(7). Under these classic assumptions, similar models have been well-studied (see Murdoch et al., 1987). Setting the right side of the equations in (4) equal to zero and solving for equilibrium yield

Table 1

Parameter values used in the stability analyses and numerical simulations. The values for the random search are similar to those used by Murdoch et al. (1987), including a long and stabilizing invulnerable adult host stage. Parameter values for the negative binomial search are patterned after Godfray and Hassell (1989). Note that C_H and C_P are functions of other parameters.

Parameter	Definition	Random search	Negative binomial search
T_{H_1}	Development time of H_1	3	3
T_{H_2}	Development time of H_2	varies	2
T_{H_3}	Development time of H_3	3	3
T_{H_4}	Longevity of H_4	$1/\mu_{H_4} = 16$	$1/\mu_{H_4} = 1$
T_{P_1}	Development time of P_1	varies	varies
T_{P_2}	Longevity of P_2	$1/\mu_{P_2} = 1$	$1/\mu_{P_2} = 0.4$
μ_{H_1}	Mortality rate for H_1	0	0
μ_{H_2}	Mortality rate for H_2	0	0
μ_{H_3}	Mortality rate for H_3	0	0
μ_{H_4}	Mortality rate for H_4	1/16	1
μ_{P_1}	Mortality rate for P_1	0	0
μ_{P_2}	Mortality rate for P_2	1	2.5
a	Parasitoid attack rate	1	1
λ	Host oviposition rate	33/16	15
k	Parasitoid aggregation	–	varies
n	Gamma shape (host)	varies	varies
m	Gamma shape (parasitoid)	varies	varies
τ_1	Gamma shift (host)	varies	varies
χ_1	Gamma shift (parasitoid)	varies	varies
C_H	$C_H = (n+1)/(T_{H_2} - \tau_1)$	varies	varies
C_P	$C_P = (m+1)/(T_{P_1} - \chi_1)$	varies	varies

$$H_2^* = \frac{\mu_{P_2}}{aF_{P_1}}, \qquad H_4^* = \frac{F_{H_3} H_2^* \ln\theta}{T_{H_2}\mu_{H_4}(\theta-1)},$$

$$P_1^* = aH_2^* P_2^* \frac{1 - F_{P_1}}{\mu_{P_1}}, \qquad P_2^* = \frac{1}{a}\left(\frac{\ln\theta}{T_{H_2}} - \mu_{H_2}\right), \tag{8}$$

where $\theta = \lambda F_{H_1} F_{H_3}/\mu_{H_4}$. If we set $\mathcal{R}_0 = \theta F_{H_2}$, then $\mathcal{R}_0$ represents the number of offspring produced by a typical adult host that successfully survived the H_1, H_2 and H_3 stages and was assumed to be greater than 1 to ensure the existence of the positive equilibrium. The equilibrium (8) is locally stable when all roots of the characteristic equation given in Box I (see Appendix B) have negative real parts. Murdoch et al. (1987) examined the stability properties of this model when $T_{H_1} = T_{H_3} = 0$. We use it here as a reference case for comparison with a model incorporating distributed delays in development.

3.1.1. Distributed development time

We incorporate developmental variability in the model by using a distribution function for development time in the H_2 and P_1 stages, because variability in these stages (as well as their mean length) seem likely to influence stability and persistence (e.g. Murdoch et al., 1987; Godfray and Hassell, 1989; Tuda and Shimada, 1995; Wearing et al., 2004a; Nakamichi et al., 2008). Assume that the maturation of insects in stage H_2 follows a distribution function $G(t)$. Therefore, the rate at which hosts survive parasitism and other mortality is given by

$$M_H(t) = \int_{-\infty}^{t} \lambda H_4(s - T_{H_1}) F_{H_1} G(t-s) e^{-\int_s^t (aP_2(x)+\mu_{H_2})dx}\,ds$$

$$= \int_0^{\infty} \lambda H_4(t - s - T_{H_1}) F_{H_1} G(s) e^{-\int_{t-s}^t (aP_2(x)+\mu_{H_2})dx}\,ds.$$

A natural choice of $G(t)$ is the gamma distribution:

$$g_n(t) = \frac{C_H^{n+1}}{n!} t^n e^{-C_H t}, \quad C_H \geq 0.$$

The gamma distribution reduces to the exponential if $n = 0$. As suggested by our literature survey, however, there should be a minimum period for which an insect must stay in H_2 stage before maturing into the next stage. To incorporate this minimum period, we consider a shift of the distribution:

$$G(t) = u(t - \tau_1) g_n(t - \tau_1),$$

where $u(r) = 0$ for $r < 0$ while $u(r) = 1$ for $r \geq 0$. Therefore the mean period of insects in H_2 is

$$T_{H_2} = \tau_1 + \tau_2, \quad \text{where } \tau_2 = \frac{n+1}{C_H},$$

and $M_H(t)$ has the form of

$$M_H(t) = \int_0^{\infty} \lambda H_4(t - s - T_{H_1}) F_{H_1} u(s - \tau_1)$$

$$\times g_n(s - \tau_1) e^{-\int_{t-s}^t (aP_2(x)+\mu_{H_2})dx}\,ds. \tag{9}$$

Taking the same assumptions on parasitoid maturation we have

$$M_{P_2}(t) = \int_0^{\infty} aP_2(t-s) H_2(t-s) u(s - \chi_1)$$

$$\times g_m(s - \chi_1) e^{-\mu_{P_1} s}\,ds, \tag{10}$$

where χ_1 is the minimum maturation period for parasitoids and $g_m(t)$ is the gamma distribution of order m with the mean time $\chi_2 = \frac{m+1}{C_P}$. Therefore, the mean juvenile period is

$$T_{P_1} = \chi_1 + \chi_2, \quad \text{where } \chi_2 = \frac{m+1}{C_P}.$$

The model for distributed development consists of Eqs. (4), (5), (9) and (10). Setting the right side of the model system equal to zero and solving for equilibrium yields

$$H_2^* = \frac{\mu_{P_2}}{a} e^{\mu_{P_1}\chi_1}\left(\frac{C_P + \mu_{P_1}}{C_P}\right)^{m+1},$$

$$H_4^* = \frac{F_{H_3}(aP_2^* + \mu_{H_2}) H_2^*}{\mu_{H_4}(\theta - 1)}, \tag{11}$$

$$P_1^* = \frac{aP_2^* H_2^*}{\mu_{P_1}}\left(1 - \left(\frac{C_P}{C_P + \mu_{P_1}}\right)^{m+1} e^{-\mu_{P_1}\chi_1}\right),$$

and $P_2^* = x^*/a$, where x^* is the unique solution of the equation:

$$(\theta e^{-\mu_{H_2}\tau_1})^{\frac{1}{n+1}} e^{-\frac{x\tau_1}{n+1}} = 1 + \frac{\mu_{H_2}}{C_H} + \frac{x}{C_H}. \tag{12}$$

Here $\theta = \lambda F_{H_1} F_{H_3}/\mu_{H_4}$. It was assumed that

$$(\theta e^{-\mu_{H_2}\tau_1})^{\frac{1}{n+1}} - \frac{\mu_{H_2}}{C_H} > 1$$

so that the Eq. (12) admits a unique positive solution and hence the equilibrium (11) is biologically meaningful. In the case of $\tau_1 = 0$,

i.e., no minimum development time in H_2 stage, Eq. (12) can be analytically solved and P_2^* is given by

$$P_2^* = (C_H \theta^{\frac{1}{n+1}} - C_H - \mu_{H_2})/a.$$

The local stability is governed by the roots of the characteristic equation in Box II given in Appendix B.

For both models (with fixed and distributed development times), we see that an increase in parasitoid search efficiency (a) reduces the equilibrium values of both adult host and parasitoid, but variation in a does not affect local stability. Moreover, the durations of host stages H_1 and H_3 are involved in the characteristic equations only through their sum $T_{H_1} + T_{H_3}$ in the case of no mortality in the stages (i.e., $\mu_{H_1} = \mu_{H_3} = 0$) (see also Godfray and Hassell, 1989).

3.1.2. Stability boundaries

The models described above are locally stable at the equilibrium points when all roots of the corresponding characteristic equations have negative real parts [see Box I or Box II in Appendix B]. Due to the complexity of these equations, we were forced to use numerical methods to locate the relevant roots of the equations, tracing the pure imaginary roots as the model parameters were varied. These roots determine the boundaries of stable vs. unstable areas in the parameter space, which were checked using numerical simulation of the model (4).

For the stability analysis, we used parameter values similar to Murdoch et al. (1987), who modeled the California red scale *Aonidiella aurantii* (Hemiptera: Diaspididae) and its parasitoid *Aphytis melinus* (Hymenoptera: Aphelinidae). We chose $T_{H_4} = 1/\mu_{H_4} = 16$ so that there was a long stabilizing invulnerable adult host stage relative to the duration of the other stages. We also chose λ so that the adult reproductive output ($\lambda T_{H_4} = 33/16 \times 1/16 = 33$) was the same as Murdoch et al. (1987) for their parameter ρ (Table 1). We then varied T_{H_2} and T_{P_1} because these parameters were important to stability in their model. The model structures are not identical, however, because we used four rather than two host stages as in Murdoch et al. (1987).

Fig. 3 shows the boundary of the stable area in the $T_{H_2} - T_{P_1}$ parameter plane for the random parasitism model with both fixed and distributed delays. Below each curve is the area where the corresponding model is stable, while periodically oscillating solutions occur above the curves. Fig. 3(A) indicates that the stable region for the distributed delay model is much larger than that for the fixed delay model, particularly for small (but still biologically plausible) values of the gamma shape parameters n and m. As the shape parameters n for the host and m for the parasitoid are increased the stability boundaries for the distributed delay model approach the fixed delay one. The inclusion of minimum development times τ_1 and χ_1 in both host and parasitoid reduces the area of stability, although it is still much larger than the fixed delay area (Fig. 3(B)). (Note that τ_1 and χ_1 are expressed as proportions of T_{H_2} and T_{P_1}, matching the way minimum development was expressed in the literature survey.) It also appears that variability in host, not parasitoid, development is important for stability. The stability boundaries change significantly for different n values, while m has virtually no effect on stability (Fig. 3(B)). Fig. 4 explores the effects of different minimum development times in the host and parasitoid. Increasing the minimum host development time τ_1 reduces the area of stability (Fig. 4(A)), while varying the minimum parasitoid development time χ_1 has no effect (Fig. 4(B)).

3.2. Negative binomial search and generation cycles

We now assume that the instantaneous risk of parasitism is of the form $k \ln(1 + aP_2/k)$, which is derived from the discrete-time negative binomial model (Godfray and Hassell, 1989). That is, the term M_{P_1} takes the form of

$$M_{P_1}(t) = k \ln \left(1 + \frac{aP_2(t)}{k} \right) H_2(t). \tag{13}$$

3.2.1. Fixed development time

Using this model for parasitoid search, Godfray and Hassell (1989) developed a fixed delay model where hosts and parasitoids in all stages mature into next stages at fixed time periods. From their work, the terms $M_H(t)$ and $M_{P_2}(t)$ take the following forms:

$$
\begin{aligned}
M_H(t) &= \lambda H_4(t - T_{H_1} - T_{H_2}) F_{H_1} \\
&\quad \times e^{-\int_{t-T_{H_2}}^{t} (k \ln(1 + aP_2(x)/k) + \mu_{H_2}) dx}, \\
M_{P_2}(t) &= k \ln \left(1 + \frac{aP_2(t - T_{P_1})}{k} \right) H_2(t - T_{P_1}) F_{P_1}.
\end{aligned} \tag{14}
$$

The equilibrium of the model consisting of Eqs. (4), (13) and (14) is given by

$$
\begin{aligned}
H_2^* &= \frac{\mu_{P_2} P_2^*}{F_{P_1} K^*}, \qquad H_4^* = \frac{F_{H_3} H_2^* (K^* + \mu_{H_2})}{\mu_{H_4} (\theta - 1)}, \\
P_1^* &= K^* H_2^* (1 - F_{P_1})/\mu_{P_1}, \\
P_2^* &= k \left[(\theta F_{H_2})^{\frac{1}{kT_{H_2}}} - 1 \right]/a,
\end{aligned} \tag{15}
$$

where $K^* = k \ln(1 + aP_2^*/k)$, $\theta = \lambda F_{H_1} F_{H_3}/\mu_{H_4}$. The local stability of the equilibrium is determined by its characteristic equation given in Box III in Appendix B.

Our model formulation differs slightly from that in Godfray and Hassell (1989), in that Eq. (4) implies that lifespans for adult hosts and parasitoids follow exponential distributions while they assumed fixed finite adult lifespans. However, the stability boundaries for the two models proved to be quite similar (see below).

3.2.2. Distributed development time

Similar to the case for random parasitism, we assume that the maturation periods in stage H_2 and P_1 follow a gamma distribution instead of a fixed development time. As before but in combination with negative binomial parasitism, the terms $M_H(t)$ and $M_{P_2}(t)$ have the following forms:

$$
\begin{aligned}
M_H(t) &= \int_0^\infty \lambda H_4(t - s - T_{H_1}) F_{H_1} u(s - \tau_1) g_n(s - \tau_1) \\
&\quad \times e^{-\int_{t-s}^{t} (k \ln(1 + aP_2(x)/k) + \mu_{H_2}) dx} ds, \\
M_{P_2}(t) &= \int_0^\infty k \ln \left(1 + \frac{aP_2(t - s)}{k} \right) H_2(t - s) u(s - \chi_1) \\
&\quad \times g_m(s - \chi_1) e^{-\mu_{P_1} s} ds.
\end{aligned} \tag{16}
$$

Then the mean period of hosts in stage H_2 is $T_{H_2} = \tau_1 + \tau_2$, where τ_1 is the minimum time period in the stage $\tau_2 = (n + 1)/C_H$, while the mean period of parasitoids in stage P_1 is $T_{P_1} = \chi_1 + \chi_2$ where χ_1 is the minimum time period in the stage $\chi_2 = (m + 1)/C_P$.

The equilibrium values of the variables in the model consisting of (4), (13) and (16) are

$$
\begin{aligned}
H_2^* &= \frac{\mu_{P_2} P_2^*}{K^*} e^{\mu_{P_1} \chi_1} \left(\frac{C_P + \mu_{P_1}}{C_P} \right)^{m+1}, \\
H_4^* &= \frac{F_{H_3} [K^* + \mu_{H_2}] H_2^*}{\mu_{H_4} (\theta - 1)}, \\
P_1^* &= \frac{1}{\mu_{P_1}} \left(1 - \left(\frac{C_P}{C_P + \mu_{P_1}} \right)^{m+1} E^{-\chi_1 \mu_{P_1}} \right) H_2^* K^*, \\
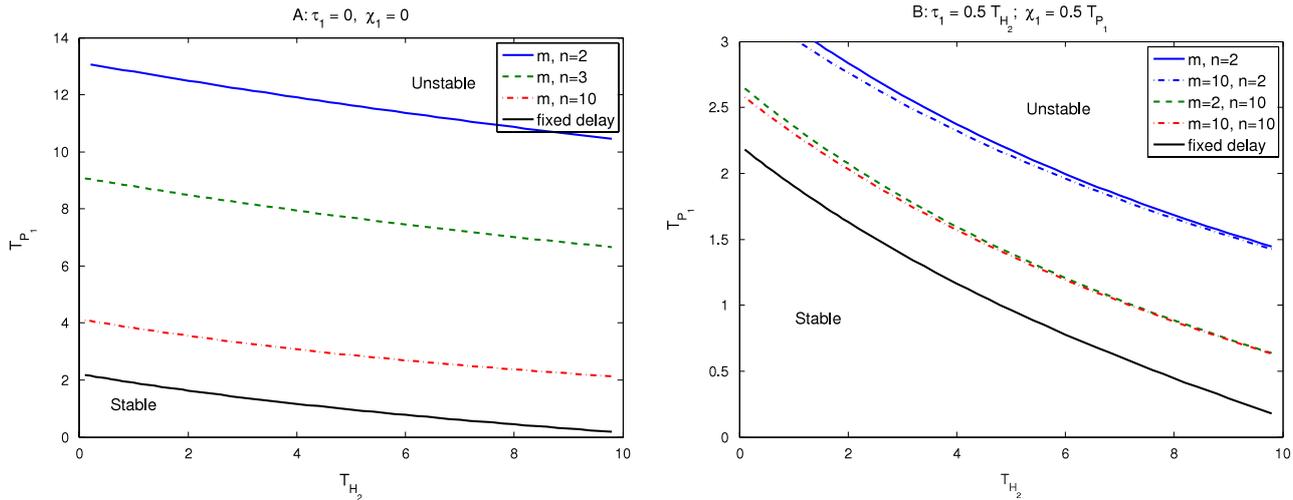P_2^* &= k(e^{x^*/k} - 1)/a,
\end{aligned} \tag{17}
$$

Fig. 3. Local stability boundaries for the random search plus invulnerable host stage model. The stability boundaries are plotted in the T_{H_2}–T_{P_1} plane, the mean development times of the vulnerable host (H_2) and parasitoid juvenile (P_1) stages, for either a fixed delay or a gamma distribution of development with different values of the shape parameters for the H_2 stage (n) and P_1 stage (m). (A) No minimum development time in H_2 or P_1 ($\tau_1 = 0$, $\chi_1 = 0$). (B) Minimum development times half of the development time in H_2 or P_1 ($\tau_1 = 0.5T_{H_2}$, $\chi_1 = 0.5T_{P_1}$). Other parameter values are listed in Table 1. Stable and unstable regions are labeled.
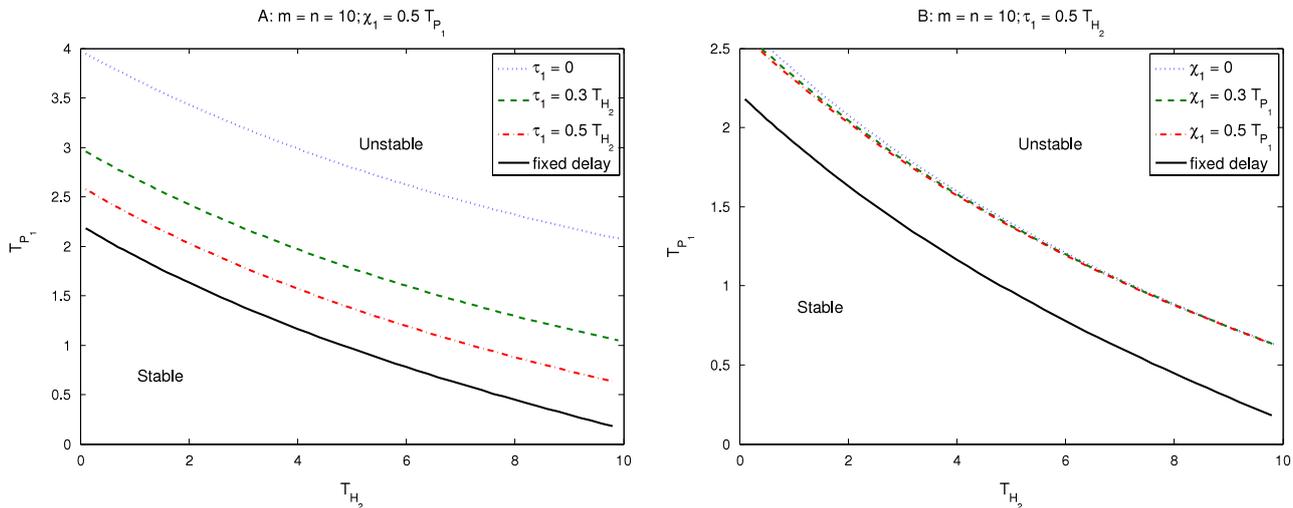

Fig. 4. Local stability boundaries for the random search plus invulnerable host stage model. The stability boundaries are plotted in T_{H_2}–T_{P_1} plane, for either a fixed delay or a gamma distribution of development in H_2 and P_1 across a range of τ_1 and χ_1 values, the minimum development times of the H_2 and P_1 stages. The gamma shape parameters for these stages were fixed at $m = n = 10$. (A) Minimum development time τ_1 varies while χ_1 is fixed. (B) Minimum development time χ_1 varies while τ_1 is fixed. Other parameter values are listed in Table 1. Stable and unstable regions are labeled.

where x^* is the unique solution of the Eq. (12) and $K^* = k \ln(1 + aP_2^*/k)$. In the case of $\tau_1 = 0$, i.e., no minimum time period for the H_2 stage, the Eq. (12) can be solved analytically and P_2^* is given by

$$P_2^* = k[e^{(C_H \theta^{\frac{1}{n+1}} - C_H - \mu_{H_2})/k} - 1]/a.$$

The characteristic equation for the equilibrium is given in Box IV in Appendix B.

3.2.3. Stability boundaries

As in previous models, we numerically determined the local stability boundaries for the fixed and distributed development versions of the negative binomial model. The boundaries are plotted in terms of the negative binomial parameter k and the ratio of parasitoid to host life cycle length, similar to the treatment in Godfray and Hassell (1989). In particular, we used the ratio of parasitoid to host generation time, namely $(T_{P_1} + T_{P_2})/(T_{H_1} + T_{H_2} + T_{H_3} + T_{H_4})$. The parameter values we used in the stability analysis were similar to Godfray and Hassell (1989), and so representative

of tropical host–parasitoid systems where the vulnerable H_2 stage is relatively brief compared to other stages, and P_1 can be quite short relative to the total host generation time, conducive to generation cycles (Table 1). We also selected a value of λ that gives nearly the same adult reproductive output ($\lambda T_{H_4} = 15$) as used by Godfray and Hassell (1989).

Fig. 5(A) shows the stability boundaries for the fixed delay version of the model, which are similar to those found by Godfray and Hassell (1989). In the figure, three "∪"-shaped curves near the ratios equal to 0.5, 1.5 and 2.5 are associated with stable population fluctuations of a period about a single generation of the host. The addition (Fig. 5(B)) of variability in development time for host and parasitoid not only significantly increases the overall stability of the system, but also eliminates single-generation cycles near the ratios equal to 1.5 and 2.5, which exist for the fixed delay version of the model. Even though there is a "∪"-shaped curve near the ratio equal to 0.5 in Fig. 5(A) and (B), representing population fluctuations, the periods of fluctuations are different (see Fig. 5(C)). Increasing the level of variability in development time for host and
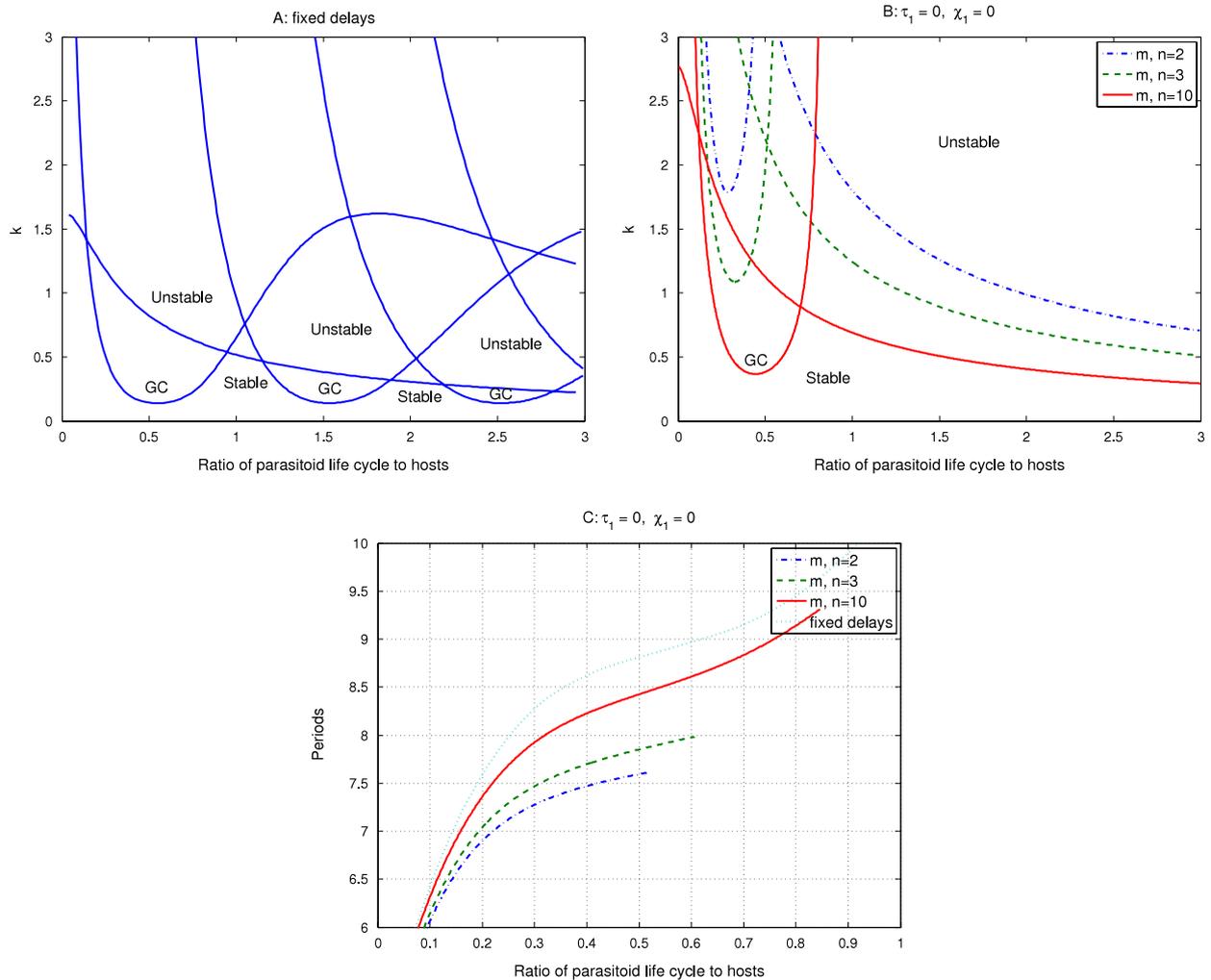
Fig. 5. Local stability boundaries for the negative binomial search plus generation cycles model with fixed or gamma distributed development times. The stability boundaries are plotted with respect to the ratio of parasitoid to host generation times (see text) and the search aggregation parameter k. (A) Fixed development times in H_2 and P_1. (B) Gamma distributed development in H_2 and P_1 for a range of values of the gamma shape parameters n and m, with no minimum development time ($\tau_1 = \chi_1 = 0$). Stable and unstable regions are labeled, with GC denoting parameter space that may produce stable population fluctuations on the order of one host generation. (C) The periods of the fluctuations represented by the "U" shaped stability boundaries in (A) and (B) for fixed and gamma distributed development times. Other parameter values are listed in Table 1.

parasitoid (i.e., decreasing values of m and n) decreases the period of single-generation cycles whenever they exist.

In contrast to the random parasitism model, variability in host and parasitoid development seem to contribute in roughly equal amounts to system stability (Fig. 6). The contribution decreases as the prescribed minimum development times increase (Fig. 6(B)). Fig. 7 shows that increasing the minimum development times τ_1 for the host and χ_1 for the parasitoid reduces stability.

4. Discussion

Our results suggest that developmental variability could be a significant source of stability in continuous-time, age-structured host–parasitoid systems. First, it appears that the level of developmental variability is quite high for some insects, even under the laboratory conditions for which most studies were conducted. Second, the area of stable parameter space was significantly increased by the addition of developmental variability to classic models and systems (Murdoch et al., 1987; Godfray and Hassell, 1989). For the random parasitism model with an invulnerable host stage, the addition of biologically plausible levels of developmental variability could potentially double the area of stable parameter space beyond that generated by an invulnerable adult host stage. Only variability in host development time was stabilizing in this

model. For the negative binomial model, however, developmental variability in both host and parasitoid was stabilizing to approximately the same degree. The most obvious effect in this model was to reduce the area of parameter space for which generation cycles occur relative to the fixed delay model (and eliminating them for ratios of the parasitoid to host life cycle of 1.5 or higher), but the overall area of stable parameter space was also increased.

What are the mechanisms underlying the enhanced stability seen in our models? This is difficult to directly ascertain from the stability analyses because of the complexity of the characteristic equations. However, it seems likely that two distinct stabilizing factors are operating in these models. One is that our models with distributed development are intermediate between models with fixed delays and ones where development time has an exponential distribution with no minimum development time, which are often more stable (Hastings, 1983, 1984; May, 1974; Eurich et al., 2005). We would therefore expect models with developmental variability to be more stable than their fixed delay counterparts, although not as stable as the exponential models. A second factor could be aggregation of risk (Chesson and Murdoch, 1986) generated by variability in host development time. To see this, consider the equation describing the host maturation rate for the random parasitism model:
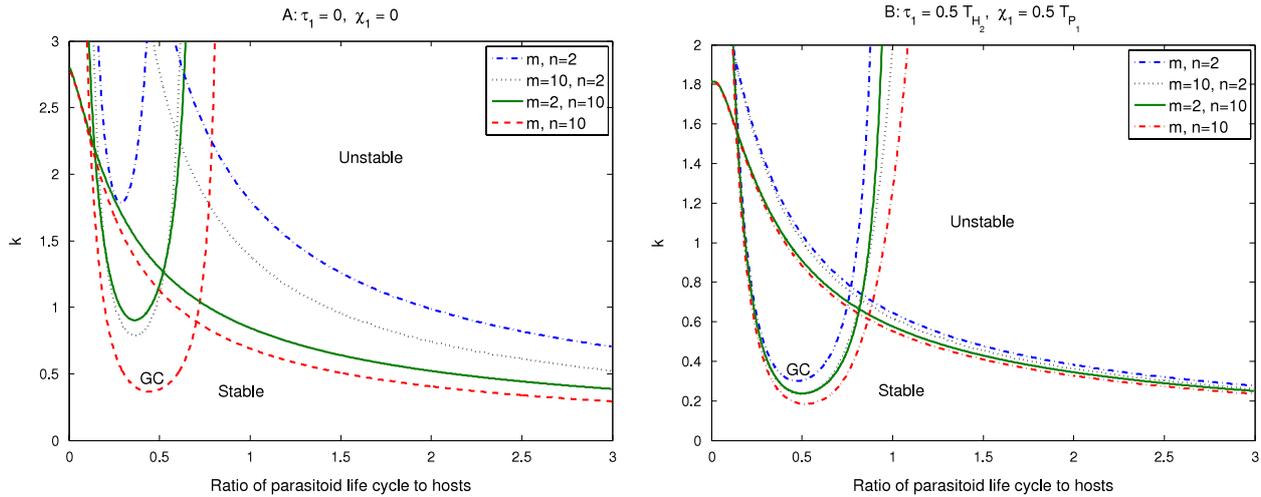
Fig. 6. Local stability boundaries for the negative binomial search plus generation cycles model for gamma distributed development times. The stability boundaries are plotted with respect to the ratio of parasitoid to host generation times (see text) and the search aggregation parameter k. (A) Effect of different values of the gamma shape parameter n for the H_2 stage vs. m for the P_1 stage, for no minimum development time ($\tau_1 = \chi_1 = 0$). (B) Effect of different values of the gamma shape parameter n for the H_2 stage vs. m for the P_1 stage, for $\tau_1 = 0.5T_{H_2}$ and $\chi_1 = 0.5T_{P_2}$. Stable and unstable regions are labeled, with GC denoting parameter space that may produce stable population fluctuations on the order of one host generation. Other parameter values are listed in Table 1.
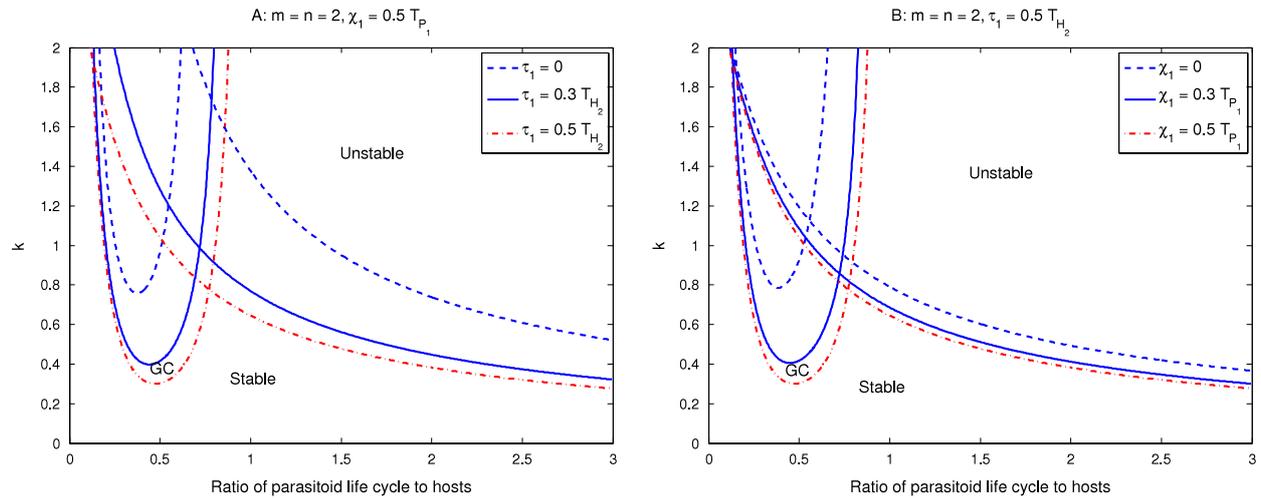
Fig. 7. Local stability boundaries for the negative binomial search plus generation cycles model for gamma distributed development times. The stability boundaries are plotted with respect to the ratio of parasitoid to host generation times (see text) and the search aggregation parameter k. (A) Effect of different values of the minimum development time τ_1 for the H_2 stage, for $m = n = 2$ and $\chi_1 = 0.5T_{P_1}$. (B) Effect of different values of the minimum development time χ_1 for the P_1 stage, for $m = n = 2$ and $\tau_1 = 0.5T_{H_2}$. Stable and unstable regions are labeled, with GC denoting parameter space that may produce stable population fluctuations on the order of one host generation. Other parameter values are listed in Table 1.

$$M_H(t) = \int_0^\infty \lambda H_4(t - s - T_{H_1}) F_{H_1} G(s) e^{-\int_{t-s}^t (aP_2(x) + \mu_{H_2}) dx} ds.$$

If we suppose that G has a gamma distribution with shape parameter n, $\mu_{H_2} = 0$, and P_2 and H_4 are constant over time, this equation reduces to

$$M_H = \lambda H_4 F_{H_1} (1 + aP_2/n)^{-n}.$$

The term in parentheses is the zero probability of the negative binomial distribution, i.e., the probability of surviving parasitism, and so the model reduces to a form of the negative binomial model under these conditions (May, 1978; Godfray and Hassell, 1989). This type of parasitoid search is stabilizing because it incorporates density-dependence in the overall attack rate of the parasitoid. The underlying mechanism usually cited for the negative binomial model is a spatially aggregated distribution of parasitoids, or differences in the vulnerability of individual hosts to parasitism such as refugia (Bailey et al., 1962; May, 1978; Chesson and Murdoch, 1986). In models with distributed host development,

however, hosts could differ in vulnerability because of natural variability in development time, which exposes them to parasitoid attack for different periods of time.

Our findings suggest some avenues for further research. One would be an examination of how host–parasitoid dynamics are influenced by developmental variability in other stages of the host life cycle, or variability in fecundity or mortality schedules with age. Another would be a comparison of the different methodologies used to quantify variability in development time. The most popular method uses nonlinear regression to fit the Weibull distribution function to the distribution of development times (Wagner et al., 1984), but maximum likelihood methods are available for both the Weibull and gamma distributions (such as PROC UNIVARIATE, SAS Institute, Inc. (2003)) and may have advantages. Our literature survey indicates that variability in the development time of natural enemies is seldom quantified, although this could potentially affect stability. There are also relatively few studies of developmental variability under field conditions. This would seem to require following the development of juveniles hosts and parasitoids in

$$
\begin{vmatrix}
\eta + \ln(\theta)/T_{H_2} & -\lambda F_{H_1} e^{-T_{H_1}\eta}(1 - e^{-T_{H_2}\eta}/\theta) & aH_2^* - \dfrac{a\mu_{H_4} H_4^* (1 - e^{-T_{H_2}\eta})}{F_{H_3}\eta} \\[2ex]
0 & \eta + \mu_{H_4}(1 - e^{-(T_{H_1}+T_{H_2}+T_{H_3})\eta}) & a\mu_{H_4} H_4^* e^{-T_{H_3}\eta} \dfrac{1 - e^{-T_{H_2}\eta}}{\eta} \\[2ex]
-aP_2^* F_{P_1} e^{-T_{P_1}\eta} & 0 & \eta + \mu_{P_2}(1 - e^{-T_{P_1}\eta})
\end{vmatrix} = 0
$$

Box I.

the field, but for systems where these stages are sessile this should be feasible. It seems likely that developmental variability would be greater under field conditions, where local differences in microclimate could operate. It would also be useful to follow the fate of individual hosts with respect to parasitism—do hosts with longer development times actually have a higher risk of parasitism? There are also laboratory systems where it may be possible to experimentally manipulate variability in development time and observe its effect on host–parasitoid dynamics. For example, Tuda (1996) manipulated the length of the vulnerable period in the bruchid beetle *C. maculatus* by rearing them on different bean species, and so mixtures of different bean species might be used to manipulate variability in development time. We further suggest that empirical investigations of host–parasitoid dynamics should take into account variability in development time, because this variability could significantly affect stability or persistence and is likely present to some extent in any such system.

Acknowledgments

Appendix A. Survey of distributed development

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Appendix B. Local stability analysis for the models

Let us first consider the model system (4)–(7) with random parasitism and fixed development times. Since the equations for the variables H_2, H_4, and P_2 do not involve the variable P_1 in the system, the local stability of the equilibrium (8) can be determined by the three-dimensional system involving H_2, H_4, and P_2. Let h_2, h_4, p_2 be the deviation of the variables H_2, H_4, P_2 from their equilibrium values, that is,

$$h_2(t) = H_2(t) - H_2^*, \qquad h_4(t) = H_4(t) - H_4^*,$$
$$p_2(t) = P_2(t) - P_2^*.$$

Substituting the deviations into the system (4)–(7) and linearizing the resulted system yields the following linear system, which governs the dynamics of the deviations to the first order.

where η is the variable of the equation and

$$\begin{vmatrix} \eta + aP_2^* + \mu_{H_2} & -\lambda F_{H_1}(1 - B_1)e^{-T_{H_1}\eta} & aH_2^* - a\lambda F_{H_1}H_4^*B_2 \\ 0 & \eta + \mu_{H_4} - \lambda F_{H_1}F_{H_3}B_1e^{-(T_{H_1}+T_{H_3})\eta} & a\lambda F_{H_1}F_{H_3}H_4^*e^{-T_{H_3}\eta}B_2 \\ -aP_2^*B_3 & 0 & \eta + \mu_{P_2} - aH_2^*B_3 \end{vmatrix} = 0$$

Box II.

$$\begin{vmatrix} \eta + \dfrac{\ln\theta}{T_{H_2}} & -\lambda F_{H_1}e^{-T_{H_1}\eta}(1 - e^{-T_{H_2}\eta}/\theta) & \dfrac{A_1^*}{F_{P_1}} - A_2^*\dfrac{1 - e^{-T_{H_2}\eta}}{F_{H_3}\eta} \\ 0 & \eta + \mu_{H_4}(1 - e^{-(T_{H_1}+T_{H_2}+T_{H_3})\eta}) & A_2^*\dfrac{1 - e^{-T_{H_2}\eta}}{\eta}e^{-T_{H_3}\eta} \\ -\dfrac{F_{P_1}\ln(\theta F_{H_2})e^{-T_{P_1}\eta}}{T_{H_2}} & 0 & \eta + \mu_{P_2} - A_1^*e^{-T_{P_1}\eta} \end{vmatrix} = 0$$

where

$$A_1^* = \frac{aF_{P_1}H_2^*}{1 + aP_2^*/k}, \qquad A_2^* = \frac{a\mu_{H_4}H_4^*}{1 + aP_2^*/k}$$

Box III.

$$\begin{vmatrix} \eta + K^* + \mu_{H_2} & -\lambda F_{H_1}(1 - C_1)e^{-T_{H_1}\eta} & A^*H_2^* - A^*H_4^*C_2 \\ 0 & \eta + \mu_{H_4} - \lambda F_{H_1}F_{H_3}C_1e^{-(T_{H_1}+T_{H_3})\eta} & A^*H_4^*F_{H_3}C_2e^{-T_{H_3}\eta} \\ -K^*C_3 & 0 & \eta + \mu_{P_2} - A^*H_2^*C_3 \end{vmatrix} = 0,$$

where

$$A^* = \frac{ka}{k + aP_2^*}, \qquad C_1 = \frac{1}{\theta}\left(\frac{K^* + \mu_{H_2} + C_H}{\eta + K^* + \mu_{H_2} + C_H}\right)^{n+1}e^{-\tau_1\eta},$$

$$C_2 = \frac{\lambda F_{H_1}}{\theta\eta}\left[1 - \left(\frac{K^* + \mu_{H_2} + C_H}{\eta + K^* + \mu_{H_2} + C_H}\right)^{n+1}e^{-\tau_1\eta}\right],$$

$$C_3 = \left(\frac{C_P}{\eta + \mu_{P_1} + C_P}\right)^{m+1}e^{-(\eta+\mu_{P_1})\chi_1}$$

Box IV.

$$\frac{dh_2(t)}{dt} = \lambda h_4(t - T_{H_1})F_{H_1} - m_{P_1}(t) - m_H(t) - \mu_{H_2}h_2(t),$$

$$\frac{dh_4(t)}{dt} = m_H(t - T_{H_3})F_{H_3} - \mu_{H_4}h_4(t), \qquad (18)$$

$$\frac{dp_2(t)}{dt} = m_{P_2}(t) - \mu_{P_2}p_2(t).$$

Here, $m_{P_1}(t) = aP_2^*h_2(t) + aH_2^*p_2(t)$ and

$$m_H(t) = \lambda h_4(t - T_{H_1} - T_{H_2})D^* \\ - \lambda H_4^*D^*\int_{t-T_{H_2}}^t ap_2(x)dxds,$$

$$m_{P_2}(t) = m_{P_1}(t - T_{P_1})F_{P_1}, \qquad D^* = F_{H_1}e^{-(aP_2^*+\mu_{H_2})T_{H_2}}.$$

Taking the Laplace transforms of the Eqs. (18) yields a system of algebraic equations whose characteristic equation is given in Box I, where $\theta = \lambda F_{H_1}F_{H_3}/\mu_{H_4}$ and η is the variable of the equation. The roots of this characteristic equation must have negative real parts for the local stability of the equilibrium. We use this model as a reference case for comparison with a model incorporating distributed delays in development.

For the random parasitism model with distributed development times consisting of Eqs. (4), (5), (9) and (10), the stability of the equilibrium (11) is governed by a characteristic equation.

Similarly, consider the deviations of the variables H_2, H_4 and P_2 from their equilibrium values given by (11). The dynamics of these deviations is controlled by system (18) with

$$m_H(t) = \int_0^\infty \lambda h_4(t - s - T_{H_1})F_{H_1}u(s - \tau_1) \\ \times g_n(s - \tau_1)e^{-(aP_2^*+\mu_{H_2})s}ds \\ - \int_0^\infty \lambda H_4^*F_{H_1}u(s - \tau_1)g_n(s - \tau_1) \\ \times e^{-(aP_2^*+\mu_{H_2})s}\int_{t-s}^t ap_2(x)dxds,$$

$$m_{P_2}(t) = \int_0^\infty ap_2(t - s)H_2^*u(s - \chi_1)g_m(s - \chi_1)e^{-\mu_{P_1}s}ds \\ + \int_0^\infty aP_2^*h_2(t - s)u(s - \chi_1)g_m(s - \chi_1)e^{-\mu_{P_1}s}ds.$$

By taking the Laplace transforms of the linearized system, we can obtain the characteristic equation in this case, given in Box II,

$$B_1 = \left(\frac{C_H}{\eta + aP_2^* + \mu_{H_2} + C_H}\right)^{n+1}e^{-(aP_2^*+\mu_{H_2}+\eta)\tau_1} \\ = \frac{1}{\theta}\left(\frac{aP_2^* + \mu_{H_2} + C_H}{\eta + aP_2^* + \mu_{H_2} + C_H}\right)^{n+1}e^{-\tau_1\eta},$$

$$B_2 = \frac{1}{\eta}\left[\left(\frac{C_H}{aP_2^* + \mu_{H_2} + C_H}\right)^{n+1}\right.$$
$$\left. - \left(\frac{C_H}{\eta + aP_2^* + \mu_{H_2} + C_H}\right)^{n+1} e^{-\tau_1 \eta}\right] e^{-(aP_2^* + \mu_{H_2})\tau_1}$$
$$= \frac{1}{\theta\eta}\left[1 - \left(\frac{aP_2^* + \mu_{H_2} + C_H}{\eta + aP_2^* + \mu_{H_2} + C_H}\right)^{n+1} e^{-\tau_1 \eta}\right],$$
$$B_3 = \left(\frac{C_P}{\eta + \mu_{P_1} + C_P}\right)^{m+1} e^{-(\eta + \mu_{P_1})\chi_1}.$$

In the case of negative binomial parasitism, the equilibrium for the model with fixed development time (consisting of Eqs. (4), (13) and (14)) is given by (15). Following the same procedures as before, we can obtain the corresponding characteristic equation given in Box III. However, if the model system consists of Eqs. (4), (13) and (16) including distributed development times, the corresponding characteristic equation is given by the equation in Box IV. Here the equilibrium values are given by (17).

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