The Dynamical Consequences of Developmental Variability and Demographic Stochasticity for Host-Parasitoid Interactions

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ABSTRACT: Few age-structured models of species dynamics incorporate variability and uncertainty in population processes. Motivated by laboratory data for an insect and its parasitoid, we investigate whether such assumptions are appropriate when considering the population dynamics of a single species and its interaction with a natural enemy. Specifically, we examine the effects of developmental variability and demographic stochasticity on different types of cyclic dynamics predicted by traditional models. We show that predictions based on the deterministic fixed-development approach are differentially sensitive to variability and noise in key life stages. In particular, we find that the demonstration of half-generation cycles in the single-species model and the multigeneration cycles in the host-parasitoid model are sensitive to the introduction of developmental variability and noise, whereas generation cycles are robust to the intrinsic variability and uncertainty that may be found in nature.

Keywords: demographic noise, distributed maturation periods, integro-differential equations, *Plodia-Venturia*, generation cycles, stage-structured models.

Variability and uncertainty are ubiquitous in nature, yet many models designed to explore ecological dynamics remain largely rigid and deterministic. In part, this is due to the insights offered by analytical approaches, which are often tractable when investigating relatively simple models. The popularity of deterministic models may also be due to the widely held assumption that when population numbers are considered to be sufficiently large, demographic stochasticity can be ignored (Beverton and Holt 1956) because the fluctuations in population numbers introduced by demographic noise scale inversely with population size (May 1973). However, a recent body of work shows that incorporating stochasticity into a broad spectrum of models can have important dynamical consequences (Higgins et al. 1997; Henson et al. 1998; Bjørnstad and Grenfell 2001; Coulson et al. 2001; Rohani et al. 2002; Turchin 2003) as well as strongly influence population persistence (Wilson and Hassell 1997; Donalson and Nisbet 1999; Rosà et al. 2003). For example, in contrast to conventional wisdom, decaying oscillations in a deterministic model can be sustained in the presence of demographic noise, even for large population numbers (Bartlett 1960; Aparicio and Solari 2001). Clearly, establishing how and under what circumstances stochasticity can affect the conclusions drawn by purely deterministic models is essential if we are to use models to adequately examine empirical data and develop a sound understanding of patterns and processes in population ecology.

Age- and stage-structured population models are a particularly rich source of interesting dynamical behavior and have led to important insights into single-species population dynamics as well as those of host-pathogen and predator-prey interactions (Briggs and Godfray 1995; Murdoch et al. 1997; Bonsall and Eber 2001; Caswell 2001). A variety of formalisms have been used to model age-structured populations, but almost all of these approaches are characterized by the assumption that aging is a synchronous process; that is, individuals born at the same time mature at the same rate (McKendrick 1926; von Foerster 1959), even in a periodic environment (Oster and Takahashi 1974). Often the assumption is much stronger: aging is uniform (i.e., every individual matures at the same rate), which is generally the case for the lumped age- and stage-structured frameworks (Leslie 1945; Gurney et al.

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1983). We suspect that the introduction of variability and uncertainty into population age or stage structure may play a key role in determining population dynamics. Evidence for this comes from recent work on disease models, which has explored the effects of variation in host susceptibility (Dwyer et al. 1997) and infectious period (Lloyd 2001; Keeling and Grenfell 2002) and shown them to have important dynamical consequences. Furthermore, the study of individual-based, size-structured models has demonstrated that variability in individual life histories can lead to population-level effects (De Roos and Persson 2001). Surprisingly, few other population-level models have considered that an individual's rate of development, or the period of time an individual spends in a particular stage, is described by a distribution of values (Blythe et al. 1984; Plant and Wilson 1986; Briggs et al. 1993; Pfister and Stevens 2003).

In this article we consider the effects of developmental variability and demographic stochasticity on the dynamics of a host-parasitoid model. The model was initially developed to investigate the laboratory population dynamics of the Indian meal moth *Plodia interpunctella* (henceforth *Plodia*) and its ichneumonid parasitoid *Venturia canescens* (henceforth *Venturia*). We therefore begin by introducing our study system and summarizing previous modeling work that assumed that individuals mature in a uniform manner. We then present experimental data to motivate why we should be interested in incorporating developmental variability into our modeling approaches. However, our aim is not solely to consider the effects of variability and uncertainty on a specific system. The *Plodia-Venturia* interaction has a number of regulatory mechanisms that are common to many host-parasitoid interactions: competition for resources, cannibalism, and parasitism of the larval stages. Furthermore, the model exhibits a range of cyclic dynamics that are characteristic of stage-structured host-parasitoid models in general: host generation cycles, half-generation cycles, and multigeneration consumer-resource cycles. It is this last property that is essential to the core of our article: we would like to understand the sensitivity of certain types of host-parasitoid dynamics to the sequential introduction of variability in maturation times and demographic stochasticity.

Our approach is to take parameter values from representative examples of the dynamics of the original model that assumes fixed developmental times and investigate how the dynamical outcomes are qualitatively altered as we manipulate the degree of variability in each developmental stage, both in the presence and absence of demographic noise. Simultaneously, we explore the implications of these results for the study of our specific laboratory populations by relating them to variability estimates from experimental data. We are, in fact, comparing the outcomes of three model formulations: the original deterministic fixed-delay model (FDM; development times are fixed); the deterministic distributed-delay model (DDM; development times are distributed, but the proportion of individuals taking a particular time to develop is always the same); and the stochastic distributed-delay model (SDDM; on average, fecundity, development, and mortality processes are the same as the DDM, but they are discrete probabilistic events giving rise to integer-valued populations).

Using these comparisons, we show that predictions from the traditional FDM are differentially sensitive to variability and noise in key life stages. Specifically, we find that the demonstration of half-generation cycles in the single-species model and the multigeneration cycles in the host-parasitoid model are sensitive to the introduction of developmental variability and noise, whereas generation cycles are robust to the intrinsic variability and uncertainty that may be found in nature. We discuss phenomena associated with the interplay of developmental variability, stochasticity, and nonlinearity as well as with the representation of populations as integer entities.

Variability in Insect Development Times

Like many insect species, our study organism, *Plodia*, has a life cycle composed of distinct developmental stages: egg, larva (five instars), pupa, and adult. Its parasitoid natural enemy, *Venturia*, has a juvenile stage spent entirely within the host and an adult stage that preferentially attacks late instar larvae (Sait et al. 1997). Regardless of the instar attacked, *Venturia*'s own development is delayed until the host starts to pupate (Harvey et al. 1994). Published time series data characteristically exhibit host generation cycles (~42 days) in both the host-alone and host-parasitoid systems (Begon et al. 1995). Evidence from a number of empirical and modeling studies suggests that both asymmetrical larval competition (whereby small larvae are more susceptible to competition than large larvae and exert weaker competitive effects) and larval egg cannibalism are driving the *Plodia* dynamics (Richards and Thomson 1932; Bjørnstad et al. 1998; Reed 1998; Briggs et al. 2000) and that *Venturia*'s interaction reinforces these dynamics (Bjørnstad et al. 2001; Wearing et al. 2004).

This system has been modeled previously by Briggs et al. 2000 (*Plodia* alone), Wearing et al. 2004 (*Plodia-Venturia*), and Sait et al. 2000 (three-species interaction with *Plodia*'s granulovirus) using a delay-differential equation approach. (For the mathematical details of the *Plodia-Venturia* model, see app. B.) In addition, Bjørnstad and colleagues have adopted a statistical time-series approach, which takes into account the demographic noise present in the data (Bjørnstad et al. 1998, 2001). Both of these

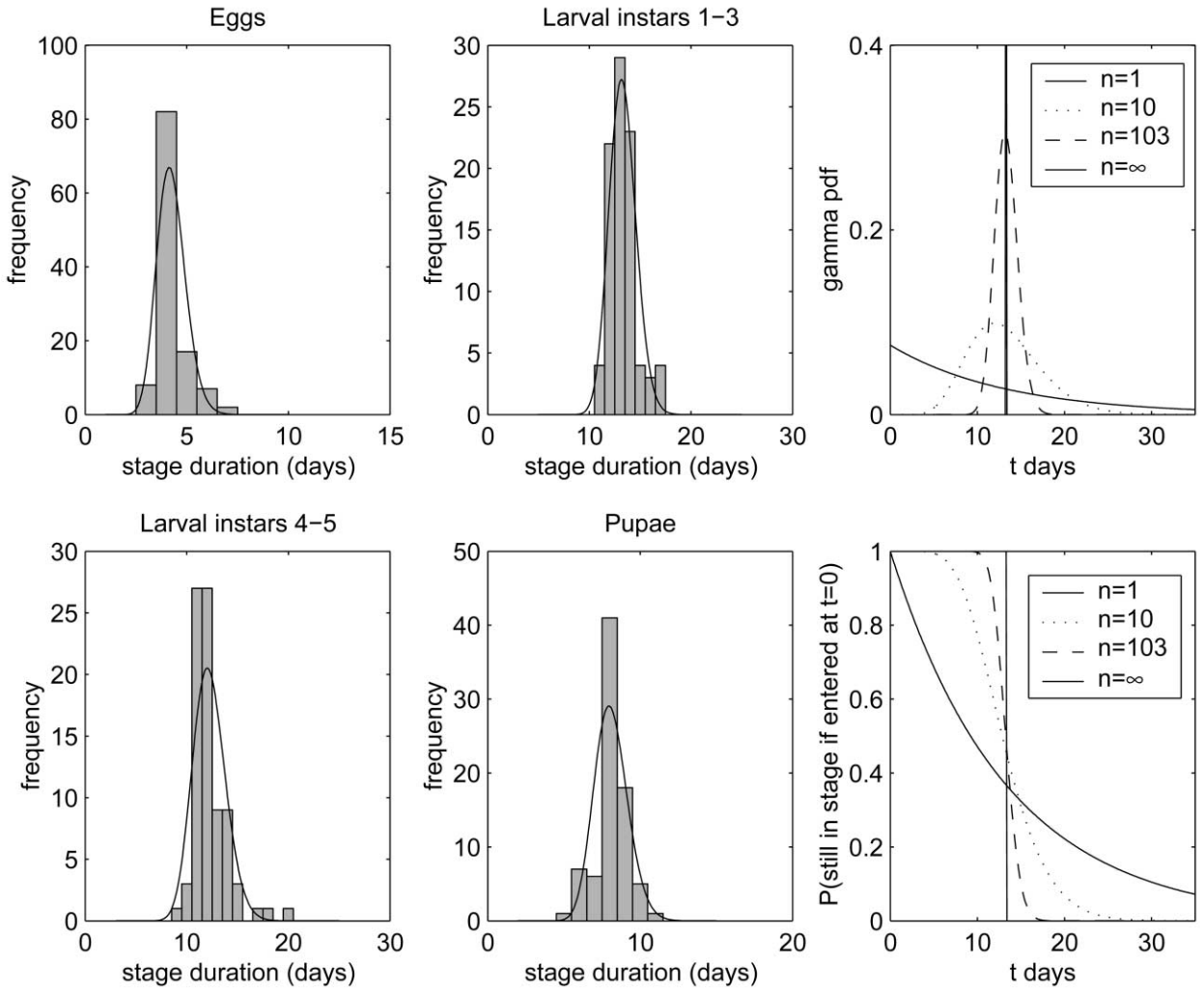

Figure 1: Histograms showing the distribution of maturation times for the egg, small larval (instars 1–3), large larval (instars 4–5), and pupal stages of *Plodia*. We divide the larval stages into two groups, as this reflects the dominant intra- and interspecific interactions with natural enemies in this system (see also Bjørnstad et al. 1998, 2001; Briggs et al. 2000). The solid curves show the best-fit gamma distribution using maximum likelihood techniques. The two far-right panels illustrate the probability density function, $f(t) = t^{n-1}e^{-t/\alpha}/\alpha^n(n-1)!$ (*top panels*) and the corresponding profiles of the probability of maturing out of the class at time t (in the absence of mortality) given that an individual entered at $t = 0$ (*bottom panels*). As n increases ($\alpha = 13.3/n$), the profile sharpens and approaches a step function, which is that represented by the fixed-delay models.

approaches assume a fixed stage duration even though it is known that individual maturation times in insect populations are variable and are often dependent on resource availability and quality (e.g., Benson 1973; Daumal and Pintureau 1985; Gordon and Stewart 1988; Johnson et al. 1992; Lane and Mills 2003). Furthermore, the mechanism by which half-generation and generation cycles propagate through the host population is dependent on the length of time during which the strongest competitive effects occur. For example, in the host-alone model with only asymmetric larval competition and no egg cannibalism, the strongest competitive effects, that is, the effect of large larvae on small larvae, occur for a relatively short period. This allows two cohorts to propagate through the population every generation, giving rise to half-generation cycles (Briggs et al. 2000). We might predict that increased variability in the large larval stage would sufficiently prolong the period of competition to suppress one of the cycles.

From our own laboratory experiments, based on ongoing population studies of *Plodia* and its natural enemies (Rohani et al. 2003; Wearing et al. 2004), we illustrate the distribution of maturation times for the egg, small larval, large larval, and pupal stages of *Plodia* (fig. 1; S. M. Sait,

unpublished data). Each histogram is characterized by a prominent mode within a left-hand skewed distribution. To link the data to our model, we fit a gamma distribution using maximum likelihood techniques. The choice of a gamma distribution is partly governed by the ease with which we can incorporate developmental variability into our modeling framework but also by the natural interpretation of one of the distribution's parameters, n. Specifically, n controls the shape of the gamma distribution (see the far-right panels of fig. 1): when $n = 1$, we obtain an exponential distribution; intermediate values give broadly bell-shaped distributions, and as $n \to \infty$, the gamma approaches a single peak (a delta distribution). The two extremes are equivalent to assuming that individuals mature out of a stage either at a fixed rate or after a fixed period (the fixed-delay formalism). For comparison, we also fit a normal distribution to each data set (see app. A for further details).

Incorporating Variability into a Stage-Structured Model

The most general way of incorporating variability in stage duration into the delay-differential equation models is to introduce a distributed delay. (For a review of methods applied to age-structured models—the McKendrick equation and Leslie matrices—see Plant and Wilson 1986.) The easiest way to illustrate this formalism is by example: let us consider the equation that describes the rate of change of the small larval class, $L_1(t)$,

$$\frac{dL_1}{dt} = \overbrace{R_{L1}(t)}^{\text{recruitment}} - \overbrace{f(L_1(t), L_2(t), P_A(t))L_1(t)}^{\text{mortality}} - \overbrace{M_{L1}(t)}^{\text{maturation}} \quad (1)$$

(where $L_2(t)$ denotes large larval density and $P_A(t)$ denotes adult parasitoid density), and focus on the rate of maturation out of this class. When the maturation time is a single fixed value, this term is just $R_{L1}(t - \tau_{L1})\mu(t, \tau_{L1})$, where τ_{L1} is the period spent as a small larva and μ denotes the through-stage survival probability ($\mu = \exp[-\int_{t-\tau_{L1}}^{t} f(x)dx]$). If we allow the maturation time to be drawn from a distribution, then

$$M_{L1}(t) = \int_0^\infty K(s)R_{L1}(t - s)\mu(t, s)ds, \quad (2)$$

resulting in an integro-differential equation where $K(s)$ specifies the distribution of maturation times (Blythe et al. 1984). In theory, we could take the function $K(s)$ directly from the data so that the integral becomes a discrete sum, and we obtain a delay-differential equation with considerably more delays than in the fixed-delay case (one for each bin of the distribution). However, if we wish to construct a general formulation that allows us to manipulate the degree of variability, then we assume that $K(s)$ is a continuous distribution that captures the qualitative features of the data. When we want to incorporate a specific data set, we fit the distribution to those data. If $K(s)$ is assumed to be a gamma distribution with the following notation,

$$K(s) = \frac{\rho^n s^{n-1} e^{-\rho s}}{(n-1)!}, \; n \geq 1, \; \rho > 0, \quad (3)$$

then we can use a property known as the linear chain trick (MacDonald 1978) to rewrite the integro-differential equation as a series of n coupled ordinary differential equations:

$$\begin{aligned}
\frac{dL_1^1}{dt} &= R_{L1}(t) - f(L_1, L_2, P_A)L_1^1 - \rho L_1^1, \\
\frac{dL_1^2}{dt} &= \rho L_1^1 - f(L_1, L_2, P_A)L_1^2 - \rho L_1^2, \\
&\vdots \\
\frac{dL_1^n}{dt} &= \rho L_1^{n-1} - f(L_1, L_2, P_A)L_1^n - \rho L_1^n. \quad (4)
\end{aligned}$$

The sum of these equations is equal to 1, specified by 2 and 3. If we fix the average time spent in a particular stage, say τ, then the parameter n determines the relative variation of the distribution (coefficient of variation [CV] $= 1/\sqrt{n}$) and the value of ρ ($= n/\tau$). Thus, changing n gives us a simple way of manipulating the variance of maturation times while keeping the average maturation time constant.

Under the assumption of a gamma distribution, we investigate how different degrees of variability affect the cyclic behavior in the host-alone and host-parasitoid systems by contrasting the DDMs with their FDM counterparts. In the host-alone FDM with asymmetric larval competition (large larvae exert a strong competitive pressure on small larvae), the dynamical behavior shifts from half-generation cycles to a stable equilibrium to generation cycles as the strength of egg cannibalism increases (Briggs et al. 2000). The level of egg cannibalism that occurs in the laboratory populations is an unknown quantity, so it is varied in the models to explore consistency with time series data. We choose two values of the egg cannibalism parameter (c_{E2}) away from bifurcation points: one that results in generation cycles and the other in half-generation cycles. For each value, we systematically alter the variance in the duration of each stage (the average time spent

in a stage is fixed) to investigate the stage-specific sensitivity of dynamics to variation.

In figure 2*A*, we show the amplitude of the DDM long-term dynamics as a fraction of the amplitude of the corresponding FDM dynamics with increasing n (decreasing variability). When the FDM exhibits generation cycles (fig. 2*A(i)*) it is clear that, unless variation is very high (n is small) and in all stages, the DDM produces the same cyclic dynamics as the FDM with a similar amplitude. In contrast, half-generation cycles (fig. 2*A(ii)*) are much more sensitive to variation; even with low variability, the amplitude of these cycles is much smaller than in the FDM. This is particularly the case when there is variability in either of the two larval classes (denoted by H_{L1} and H_{L2}).

In the host-parasitoid model, for fixed levels of parasitism, the FDM dynamics shift from multigeneration to

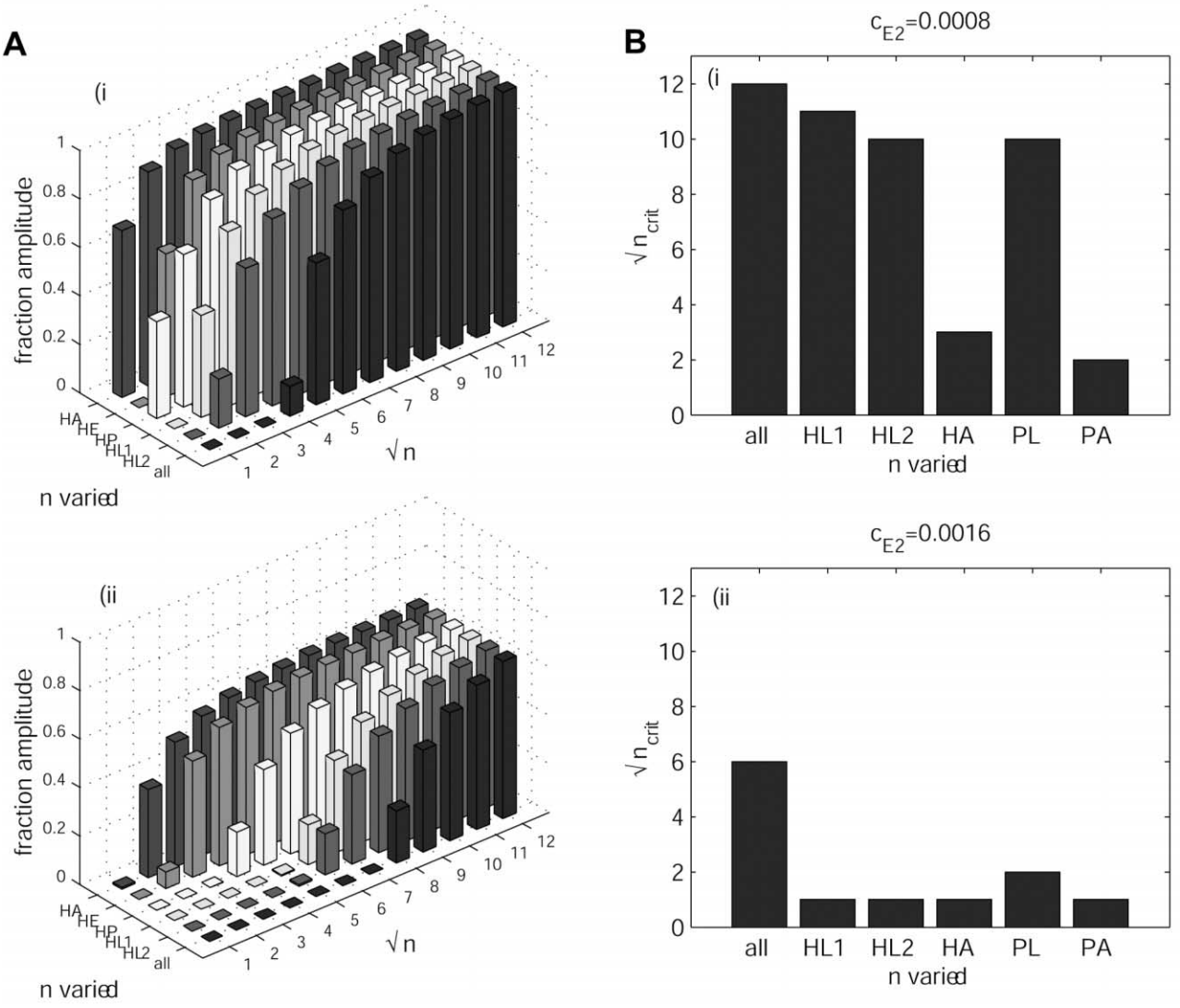

Figure 2: Sensitivity of host-alone and host-parasitoid model dynamics to variation in maturation times. In *A*, we display the amplitude of the long-term dynamics of the distributed-delay model as a fraction of the amplitude of the fixed-delay model (FDM) dynamics as we vary n, the degree of variability, for different stages of the host-alone model, when (*i*) the FDM generates generation cycles, and (*ii*) the FDM generates half-generation cycles. Zero amplitude indicates a stable equilibrium. Cycle periods are not shown because they scale with the average generation length in the same manner as in the FDM. In *B*, we record the critical value of $\sqrt{n}$ required to obtain the qualitative dynamics of the FDM for different stages of the host-parasitoid model. The parameter c_{E2} represents the strength of egg cannibalism by large larvae. H_E, H_{L1}, H_{L2}, H_P, and H_A corresponds respectively to the host stages: egg, small larval, large larval, pupal, and adult. Similarly, P_L and P_A correspond respectively to the parasitoid juvenile and adult stages. (For a color version of this figure, see the online edition of the *American Naturalist*.)

generation cycles as the level of egg cannibalism increases. The addition of variability in maturation times increases the prevalence of multigeneration cycles. Therefore, to investigate the relative effects of variation in different stages, we choose two values of the egg cannibalism parameter (c_{E2}) that give rise to generation cycles: one close to the bifurcation point in the FDM and the other away from it. In figure 2*B* we record the critical value of $\sqrt{n}$ required to obtain the qualitative dynamics of the FDM (i.e., generation cycles). The size of this value tells us the relative amount of variation ($1/\sqrt{n}$) in development rates that can be tolerated by the DDM before its dynamics differ qualitatively from those generated by the FDM. For the larger value of egg cannibalism (fig. 2*B(ii)*), we can introduce a substantial amount of variation into the DDM and still obtain the same dynamics as the FDM. As we decrease this parameter (e.g., fig. 2*B(ii)*), even small amounts of variation in the host larval and parasitoid juvenile stages give rise to multigeneration rather than generation cycles.

Thus far, we have shown that variability in host maturation times decreases the amplitude of cyclic behavior in the host-alone model and promotes multigeneration cycles in the host-parasitoid model. If we now take the level of variation from the data in figure 1, we can compare the FDM and DDM outcomes of the four cases investigated in figure 2 for our specific laboratory *Plodia-Venturia* system. The time series are shown in figure 3. In the host-alone cases (fig. 3*a*), the dynamics of the DDM are qualitatively the same as those of the FDM. However, the period and amplitude of generation cycles are slightly shorter (fig. 3*a(i)*) and the amplitude of half-generation cycles is severely reduced (fig. 3*a(ii)*). The cyclic dynamics predicted by the FDM are sustained because *Plodia* exhibits the least variability in one of the most sensitive stages—the small larval class. In the host-parasitoid simulations, we observe generation cycles for the robust case (fig. 3*b(i)*), that is, where a large amount of variation in most stages still gives rise to generation cycles and multigeneration cycles for the more sensitive one (fig. 3*b(ii)*). This is to be expected, because we know from inspection of the lower panel of figure 2*B* that the variation in the large larval class alone ($\sqrt{n} < 8$) is enough to qualitatively change the dynamics. Note that even when the host-parasitoid system exhibits generation cycles in the DDM, cycle amplitudes are considerably reduced. The time series illustrated in figure 3*a(i)* and figure 3*b(i)* (where the strength of egg cannibalism is given by $c_{E2} = 0.0016$) are the most consistent with published data (Begon et al. 1995).

Introducing Demographic Stochasticity

In the last section, we briefly explored the consequences of variability in development times and showed how some dynamical patterns are more sensitive than others. Here, we turn our attention to the consequences of allowing recruitment, mortality, and maturation to be stochastic processes; that is, we allow for random variation in the fate of individuals (demographic stochasticity). This means that, on average, probabilities determining reproduction and mortality are identical or, in the case of development times, drawn from an identical distribution, but chance events result in each individual realizing different fates. With the inclusion of demographic stochasticity, do we still see the persistence of these cyclic behaviors?

Creating a stochastic analogue of the DDM is straightforward if we assume that maturation times are described by a gamma distribution and use the ordinary differential equation formulation given by equation (4). A method developed by Gillespie (1977) provides an exact way of converting a set of ordinary differential equations into a Markov chain: the rates of the deterministic differential equation model are reinterpreted to give the probability of individuals reproducing, dying, or moving between classes in a given time interval. Specifically, we use Gillespie's direct method, which determines iteratively the time to the next event and the type of event that occurs next (further details are given in app. C in the online edition of the *American Naturalist*). The original algorithm is not computationally efficient for a large number of events (N), but if this is due to individuals passing through many states (as we have here), we can use an efficient modification by Gibson and Bruck (2000). With their method, the number of calculations per time step scales with $\log(N)$ rather than N.

Host Alone

Introducing demographic stochasticity into the *Plodia*-alone model (with distributed maturation times taken from the laboratory data in fig. 1) shows no significant difference from the deterministic equivalent (the DDM). This is most likely due to large population numbers, which average out the effects of demographic stochasticity, and it explains why laboratory populations of *Plodia* exhibit consistent dynamics in replicate experiments (Sait et al. 1994*b*; Begon et al. 1995; Bjørnstad et al. 1998). However, experimental host population dynamics are strongly influenced by resource quality, with patterns ranging from high-amplitude cycles on high-quality diets to damped oscillations on poor diets (e.g., Sait et al. 1994*b*; Knell et al. 1998; McVean et al. 2002). Here we can use the model to test whether the dynamics of populations under stress from increased competition on poorer resources are more susceptible to the effects of demographic noise. In the *Plodia*-alone model, it is possible to increase the overall strength of larval competition so that equilibrium values

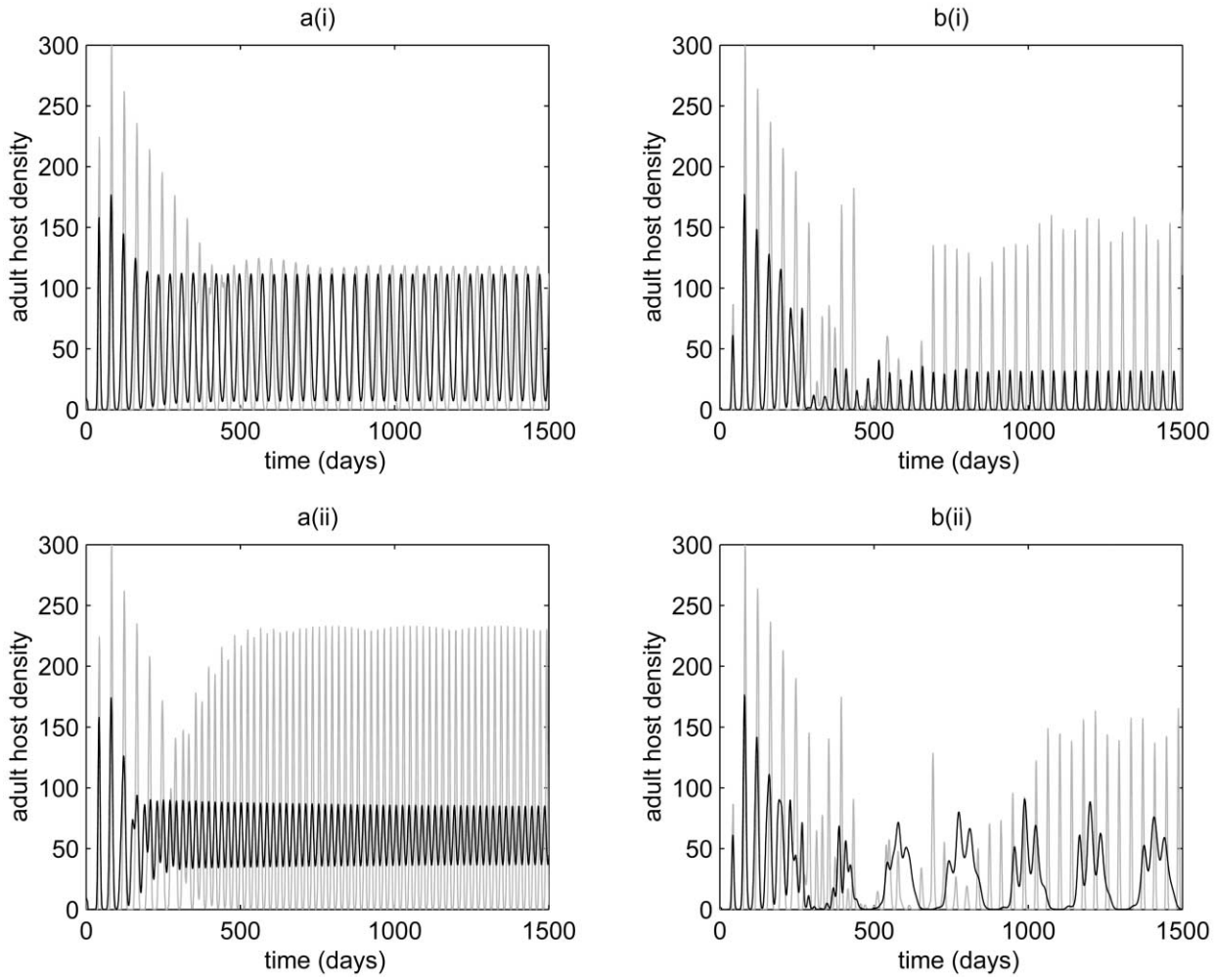

Figure 3: Comparison of *Plodia-Venturia* fixed-delay model (FDM; *light gray*) and distributed-delay model (*dark gray*) dynamics. The time series show adult host profiles from simulations with the model parameters used in figure 2 and average stage duration and stage variation from data shown in figure 1. The time series in *a(i)* and *a(ii)* illustrate host-alone dynamics for parameter values giving rise to generation cycles and half-generation cycles, respectively, in the FDM. The time series in *b(i)* and *b(ii)* illustrate host-parasitoid dynamics for parameter values giving rise to generation cycles in the FDM; adult parasitoids are introduced after 200 days. Parameter values for the gamma distributions are taken from the fits given in table A1 in appendix A (n is rounded to the nearest integer); all other stages are assumed to have $n = 150$ (small variation).

are smaller but stability and cyclic behavior remain unaffected. Under these circumstances, when persistence is still possible, the stochastic model often produces cyclic behavior with a dominant period around one generation while the deterministic analogue exhibits half-generation cycles (fig. 4*B*). Moreover, as figure 4*A* illustrates, this phenomenon occurs more frequently for larger values of n, when there is least variation in stage development.

Host with Parasitoid

For the host-parasitoid model, introducing stochasticity has dramatic consequences both in terms of host-parasitoid coexistence and in the pattern of dynamics observed. Multigeneration cycles such as those seen in figure 3*b(ii)* are inhibited, giving rise to either system (both host and parasitoid) or parasitoid extinction. Figure 5 shows the proportion of different outcomes from 50 replicate simulations for increasing levels of egg cannibalism. The deterministic system exhibits multigeneration cycles for $c_{E2} = 0$ and $c_{E2} = 0.0008$ and host generation cycles for $c_{E2} = 0.0016$. It is apparent that generation cycles are robust to the addition of demographic stochasticity, but multigeneration cycles give way to several possibilities. For no egg cannibalism, about 25% of simulations result in system extinction and the rest in parasitoid extinction. These

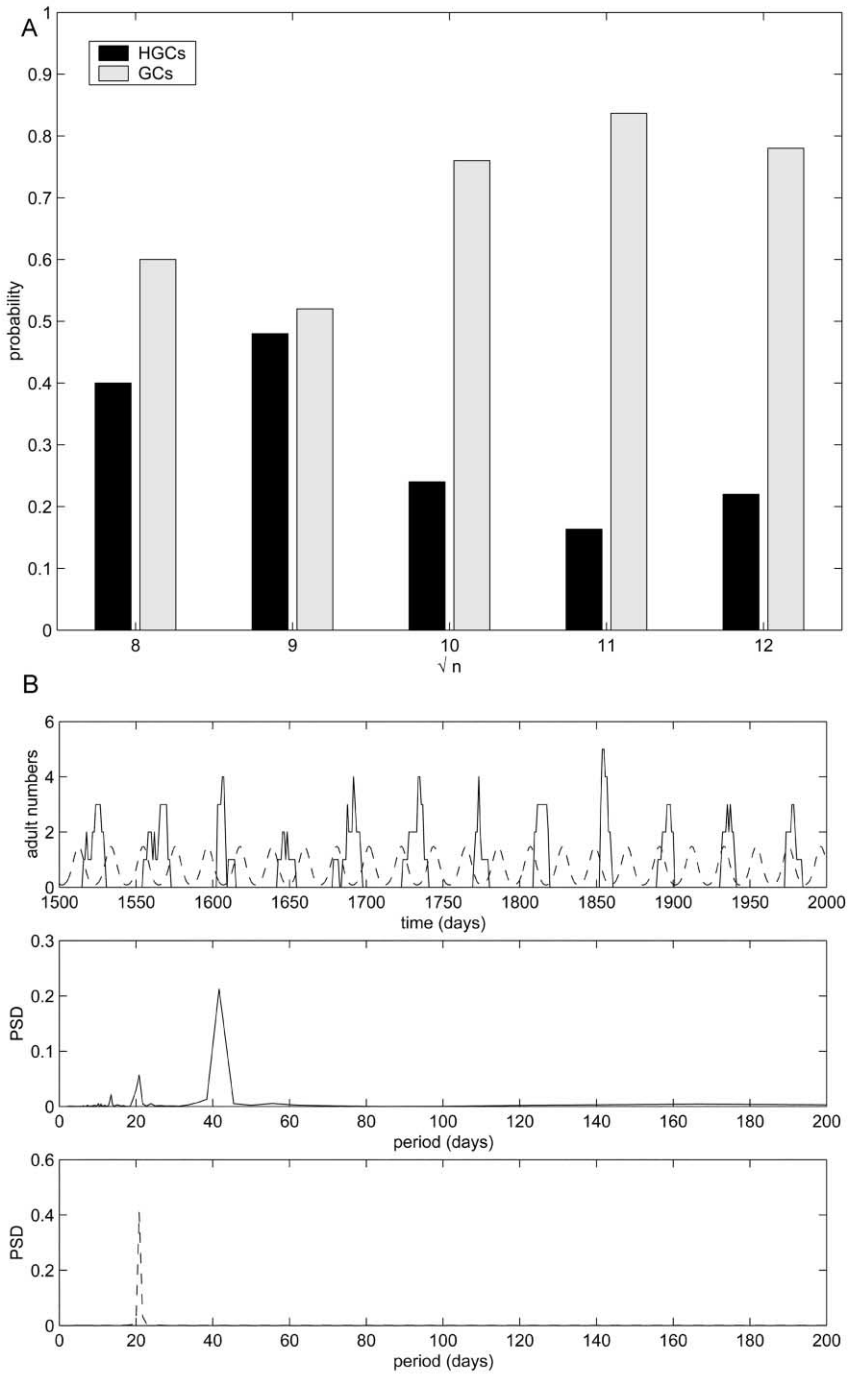

Figure 4: *A*, Dominant period from spectral analysis of realizations of the stochastic host-alone model with competition coefficient $c_{11} = 0.004$. For $\sqrt{n} > 8$, the deterministic analogue generates half-generation cycles (HGCs), whereas the majority of stochastic realizations have a dominant period closer to the average generation length (GCs). In each case, results are from 50 stochastic realizations. *B*, Deterministic simulation (*dashed line*) and a typical stochastic realization (*solid line*) of the host-alone model analyzed in *A* with variation in each stage specified by $\sqrt{n} = 12$. The lower panels show the spectral analysis of the deterministic and stochastic time series displayed in the top panel. The data are normalized before spectral analysis to allow direct comparison between any periodic signatures. The deterministic dynamics are clearly HGCs (around 20 days), whereas the stochastic realization exhibits a dominant period of a full generation.

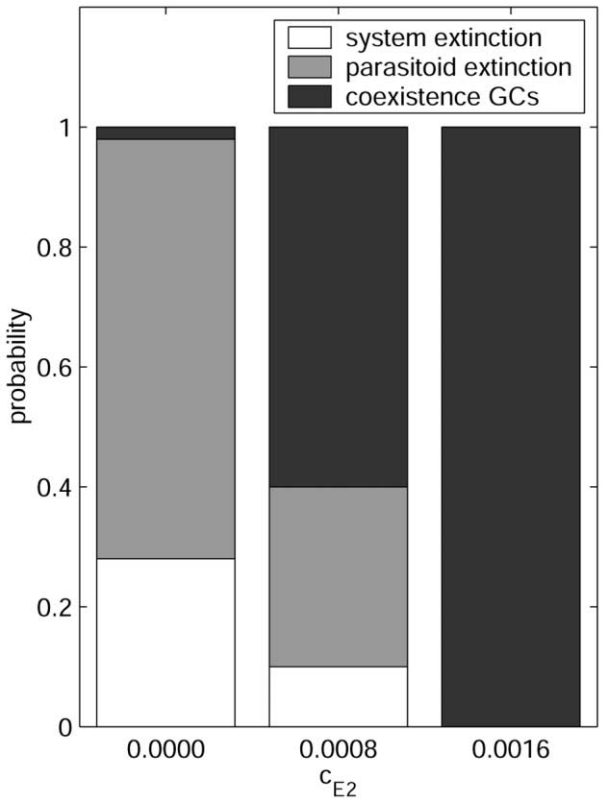

Figure 5: Introducing demographic stochasticity to the host-parasitoid model results in dynamical outcomes not predicted by the deterministic model. For 50 stochastic realizations, each run for a maximum of 2,000 days, the proportion of extinctions and existence behaviors is shown. In the deterministic analogues, the models exhibit multigeneration cycles for $c_{E2} = 0$ and $c_{E2} = 0.0008$ and generation cycles for $c_{E2} = 0.0016$.

probabilities decrease as egg cannibalism is increased, perhaps because this coincides with a reduction in the period of multigeneration cycles; hence, the duration of the "trough" is shortened. We should remark that time to extinction may be longer than experimental time series, so the probability of persistence on a laboratory time scale could be higher, on average, than is shown in figure 5. We also note that for $c_{E2} = 0.0008$, the deterministic host-alone model is stable, but there are significant periodic signatures in the stochastic simulations.

These results demonstrate that multigeneration cycles are extremely fragile in the presence of demographic stochasticity in our host-parasitoid model. In the deterministic system, these cycles show extensive periods with very low densities so that when population sizes are discrete, extinction is likely (atto fox phenomenon [Mollison 1991]). Is this still the case with multigeneration cycles whose population troughs are not so deep? As shown by Wearing et al. (2004), the *Plodia-Venturia* model can exhibit multigeneration cycles where minimum densities are not drastically low for extended periods of time. This is in the absence of egg cannibalism but with heightened competition from large larvae on all larvae. In figure 6, we alter the model parameters so that the deterministic system exhibits these smaller amplitude cycles and simulate the stochastic analogue. As in figure 5, some stochastic realizations still result in extinctions, but coexistence of host and parasitoid exhibiting multigeneration cycles is now possible.

Discussion

Our analyses indicate that the interaction between developmental variability and demographic stochasticity can have important qualitative and quantitative consequences for host-parasitoid population dynamics. We are led to speculate that certain population dynamics are more likely to be widespread in nature than others because they are more robust to variability and uncertainty. In particular, the addition of variability and stochasticity rarely inhibits generation cycles (the dynamics most often observed in the *Plodia-Venturia* populations [Begon et al. 1995]), whereas half- and multigeneration cycles are often sensitive to their presence. The manner in which this occurs may depend on both variability in maturation times and demographic noise. Indeed, the prevalence of multigeneration cycles in the host-parasitoid model increases with greater variability, but the addition of demographic stochasticity leads to extinctions or different coexistence dynamics. In the host-alone model, greater variability reduces the likelihood of half-generation cycles and shortens transient oscillations while the introduction of noise sustains this periodic signal.

With reference to our specific study system of *Plodia-Venturia*, the variability exhibited by *Plodia* is not sufficient to substantially change the qualitative predictions of the FDM. However, there are subtle changes in amplitude and period that could be important when we are using these dynamical measures to compare model outcome and data, especially with the aim of directly inferring mechanism. What is particularly interesting about *Plodia* development is that one of the stages most sensitive to variability (small larval) displays the least amount of variability. In the host-alone model, with all other stages having a small coefficient of variation, the egg and pupal stages can show a substantial amount of variability, more than twice the coefficient of variation, without the qualitative results being affected. In contrast, each larval stage is almost as sensitive to variability as are all stages combined, which is most likely due to the importance of larval competition in driving the host dynamics. Resource availability and quality are known to directly impact developmental rates in large

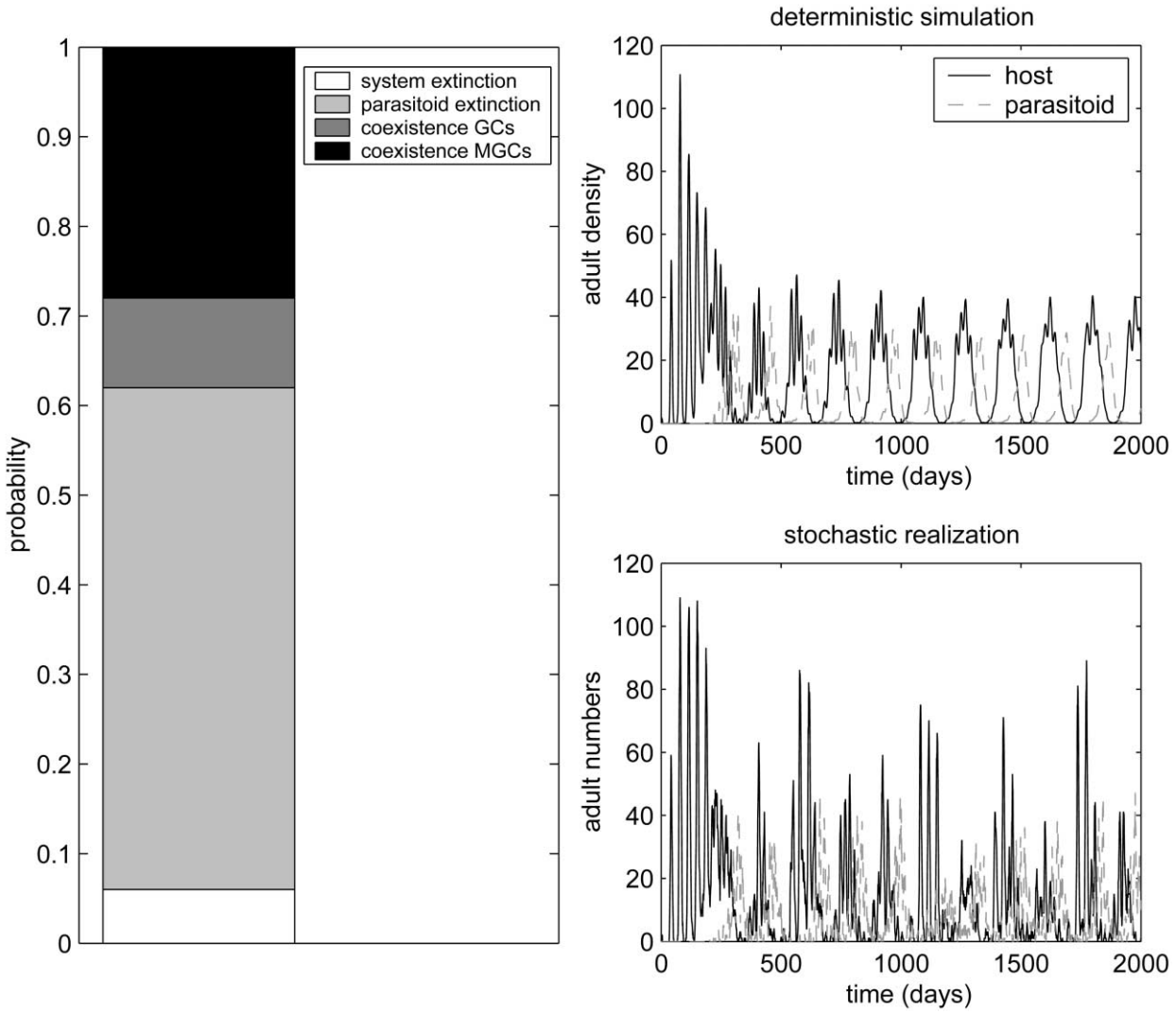

Figure 6: Outcome of stochastic realizations of the deterministic host-parasitoid model that produces multigeneration cycles with narrower troughs than in figure 5 (*top right panel*). Extinction still occurs, but coexistence via multigeneration cycles is now a possibility (*bottom right panel*). Adult parasitoids are introduced after a period of 200 days. The egg cannibalism parameter $c_{E2} = 0$, and the ratio of larval competitive effects $\chi = 40$. See appendix B for further explanation.

Plodia larvae (Reed 1998; S. M. Sait, unpublished data). Recent work by Pfister and Peacor (2003) has also suggested that population density can impact temporal correlations in the growth of individuals via changes in resource heterogeneity. Hence, one area of further work is to explore the consequences of density-dependent developmental variability.

When should stage-structured models incorporate variability in maturation times? Previous work on a model with a single developmental stage found that as long as the maturation times are not broadly distributed (the coefficient of variation is sufficiently small), the FDM adequately captures the dynamics of the DDM (Blythe et al. 1984). We have shown that when there is a series of developmental stages, differential sensitivity to variability can lead to novel predictions. Determining the stage that is the most dynamically sensitive is key. In our host-parasitoid model, developmental variability in the large larval stage, which is preferentially attacked by the parasitoid, has profound consequences for the patterns of abundance of the host and parasitoid. Crucially, in the presence of demographic noise, both parasitoid and system extinction seem more likely than coexistence as developmental variability increases. Any stochastic shift in host demographic

profile, perhaps as a result of host resource limitation or environmental changes, might be predicted to have a similar impact on the predator dynamics in tightly coupled predator-prey interactions where developmental synchrony is necessary for persistence (e.g., Godfray et al. 1994).

The introduction of demographic noise to our deterministic models results in two different phenomena: it results in qualitatively different dynamics to those observed in the deterministic system, and it introduces the possibility of population extinctions. The reasons for these phenomena may not be entirely due to uncertainty per se. Extinction is possible in part because the stochastic formulation allows the populations to be described as integer-valued variables. Indeed, extinctions occur predominantly when the deterministic system exhibits multigeneration cycles in which population densities are extremely low for extensive periods of time. The persistence of generation cycles rather than multigeneration cycles in the host-parasitoid system may be attributed to the presence of multiple stable attractors in the deterministic system (this has been shown in the FDM [Wearing et al. 2004]), whereby the addition of noise perturbs the system from one basin of attraction into another. However, the results from the host-alone model with small population numbers, in which the introduction of demographic stochasticity resulted in generation cycles rather than the half-generation cycles of the deterministic analogue, are less easy to explain; there are no multiple attractors in the host-alone model. There may well be an interesting interaction between integer-valued populations and stochasticity, but disentangling the effects of a discrete-valued population from stochasticity is not straightforward. A growing body of research has been looking at so-called lattice effects of discrete-state variables in discrete-time systems (Henson et al. 2001, 2003). Developing similar models to investigate discrete-state continuous-time populations may bring greater understanding to the results of stochastic realizations of the models presented in this article.

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APPENDIX A

Distribution Fits to Maturation Data

Table A1 lists the maximum likelihood estimates (along with 99% confidence intervals) for both gamma and normal distribution fits to the data shown in figure 1. If we examine the coefficient of variation (CV) for each stage, it is clear that the egg and pupal stages are subject to most variation, whereas the small larval stage has the least. Table A1 also gives the Kolmogorov-Smirnov coefficient, which is a measure of how far the cumulative distribution of the fitted distribution deviates from that of the data. None of the fits do well in this test, especially for the egg stage, but this is not surprising given the size of the discrete bins in the data. In general, the gamma distribution does slightly better because the data are skewed to the lower values for most of the stages.

Table A1: Maximum likelihood estimates of parameters for the gamma and normal distributions when fit to the data shown in figure 1

	Gamma distribution fit			Normal distribution fit		
Developmental stage	n	α	CV $(1/\sqrt{n})$	μ	σ	CV (σ/μ)
Egg	36.8	.116	.165	4.25	.745	.175
	(26.0, 47.6)	(.0841, .147)	K-S .4071	(4.07, 4.43)	(.636, .894)	K-S .4073
Small larval (instars 1–3)	103	.130	.0985	13.3	1.35	.102
	(62.6, 143)	(.0806, .178)	K-S .2227	(12.9, 13.7)	(1.13, 1.67)	K-S .2059
Large larval (instars 4–5)	57.9	.211	.131	12.2	1.72	.141
	(38.7, 77.1)	(.147, .276)	K-S .2440	(11.7, 12.7)	(1.43, 2.15)	K-S .2536
Pupal	54.7	.148	.135	8.10	1.07	.132
	(35.9, 73.5)	(.0946, .202)	K-S .2132	(7.78, 8.42)	(.885, 1.34)	K-S .2334

Note: Confidence intervals (99%) are given in parentheses. The coefficient of variation (CV) is calculated for each distribution to compare the relative variation between each stage. The normal distribution fit gives the sample mean (μ) and variance (σ^2). K-S represents the Kolmogorov-Smirnov coefficient, the maximum deviation of the cumulative fitted distribution from the cumulative distribution of the data. Gamma pdf: $f(x) = x^{n-1}e^{-x/\alpha}/\alpha^n(n-1)!$. Normal PDF: $f(x) = (1/\sigma\sqrt{2\pi})\exp[-(x-\mu)^2/2\sigma^2]$.

APPENDIX B

Host-Parasitoid FDM Equations and Parameters

Host-Parasitoid Fixed-Delay Model

This is the host-parasitoid model without a delay in the development of the parasitoid, formulated and investigated by Wearing et al. (2004). (The host-alone model [Briggs et al. 2000] is obtained by setting the adult parasitoid variable to 0 in the host equations.) The regulation of each stage in the life cycle of the host or parasitoid can be divided into three components: recruitment, mortality (both density-independent and density-dependent), and maturation. If there is density-dependent mortality from competition, cannibalism, or parasitism, then the through-stage survival is an integral equation. As is common practice when solving delay-differential equations, we then take the survival probability to be a variable and solve for the derivative of the integral equation at the same time as the other equations.

Host Equations. For the host population, we only need to solve explicitly for eggs, $H_E(t)$, small larvae, $H_{L1}(t)$, large larvae, $H_{L2}(t)$, and adults, $H_A(t)$. The delay-differential equations describing these stages are:

$$\frac{dH_E(t)}{dt} = \overbrace{R_E(t)}^{\text{recruitment}} - (\overbrace{c_{E2}H_{L2}(t)}^{\text{cannibalism by large larvae}} + \overbrace{d_E}^{\text{background death rate}})H_E(t) - \overbrace{M_E(t)}^{\text{maturation}}, \tag{B1}$$

$$\frac{dH_{L1}(t)}{dt} = R_{L1}(t) - (\overbrace{P_{f1}(P_A(t))}^{\text{parasitism}} + \overbrace{c_{11}H_{L1}(t)}^{\text{competition from small larvae}} + \overbrace{c_{12}H_{L2}(t)}^{\text{competition from large larvae}} + d_{L1})H_{L1}(t) - M_{L1}(t), \tag{B2}$$

$$\frac{dH_{L2}(t)}{dt} = R_{L2}(t) - (P_{f2}(P_A(t)) + c_{21}H_{L1}(t) + c_{22}H_{L2}(t) + d_{L2})H_{L2}(t) - M_{L2}(t), \tag{B3}$$

$$\frac{dH_A(t)}{dt} = R_A(t) - d_A H_A(t) - M_A(t), \tag{B4}$$

where the recruitment rates, $R_i(t)$, and maturation rates, $M_i(t)$, are given by

$$\begin{aligned} R_E(t) &= rH_A(t), \\ M_E(t) &= R_E(t-\tau_E)\sigma_E S_{HE}(t), \end{aligned} \tag{B5}$$

$$\begin{aligned} R_{L1}(t) &= M_E(t), \\ M_{L1}(t) &= R_{L1}(t-\tau_{L1})\sigma_{L1}S_{HL1}(t), \end{aligned} \tag{B6}$$

$$\begin{aligned} R_{L2}(t) &= M_{L1}(t), \\ M_{L2}(t) &= R_{L2}(t-\tau_{L2})\sigma_{L2}S_{HL2}(t), \end{aligned} \tag{B7}$$

$$\begin{aligned} R_A(t) &= M_{L2}(t-\tau_P)\sigma_P + I_{HA}(t), \\ M_A(t) &= R_A(t-\tau_A)\sigma_A, \end{aligned} \tag{B8}$$

$\sigma_i = \exp(-d_i\tau_i)$, and the time-dependent survival probabilities are

$$S_{\mathrm{HE}}(t) = \exp\left(\int_{t-\tau_{\mathrm{E}}}^{t} -c_{\mathrm{E2}}H_{\mathrm{L2}}(x)dx\right), \tag{B9}$$

$$S_{\mathrm{HL1}}(t) = \exp\left(\int_{t-\tau_{\mathrm{L1}}}^{t} -[c_{11}H_{\mathrm{L1}}(x) + c_{12}H_{\mathrm{L2}}(x) + P_{\mathrm{f1}}(P_{\mathrm{A}}(x))]dx\right), \tag{B10}$$

$$S_{\mathrm{HL2}}(t) = \exp\left(\int_{t-\tau_{\mathrm{L2}}}^{t} -[c_{21}H_{\mathrm{L1}}(x) + c_{22}H_{\mathrm{L2}}(x) + P_{\mathrm{f2}}(P_{\mathrm{A}}(x))]dx\right). \tag{B11}$$

The probabilities are evaluated by differentiating equations (B9)–(B11) with respect to t and solving the resulting delay-differential equations with those for the other variables. For example, from equation (B9) we obtain

$$\frac{dS_{\mathrm{HE}}(t)}{dt} = -c_{\mathrm{E2}}[H_{\mathrm{L2}}(t) - H_{\mathrm{L2}}(t-\tau_{\mathrm{E}})]S_{\mathrm{HE}}(t). \tag{B12}$$

The inoculation of adult hosts, $I_{\mathrm{HA}}(t)$, is defined as a constant i_{HA} over the time interval $(0, 1]$ and 0 elsewhere.

Parasitoid Equations. For the parasitoid population, we only need to solve explicitly for adults, $P_{\mathrm{A}}(t)$:

$$\frac{dP_{\mathrm{A}}(t)}{dt} = R_{\mathrm{PA}}(t) - d_{\mathrm{PA}}P_{\mathrm{A}}(t) - M_{\mathrm{PA}}(t), \tag{B13}$$

where

$$R_{\mathrm{PA}}(t) = P_{\mathrm{f1}}[P_{\mathrm{A}}(t-\tau_{\mathrm{PL}})]H_{\mathrm{L1}}(t-\tau_{\mathrm{PL}})\sigma_{\mathrm{PL}} + P_{\mathrm{f2}}[P_{\mathrm{A}}(t-\tau_{\mathrm{PL}})]H_{\mathrm{L2}}(t-\tau_{\mathrm{PL}})\sigma_{\mathrm{PL}} + I_{\mathrm{PA}}(t), \tag{B14}$$

$$M_{\mathrm{PA}}(t) = R_{\mathrm{PA}}(t-\tau_{\mathrm{PA}})\sigma_{\mathrm{PA}}. \tag{B15}$$

The parasitism function is defined as

$$P_{\mathrm{f}i}(P_{\mathrm{A}}) = k\ln\left(1 + \frac{a_i P_{\mathrm{A}}}{k}\right), \quad i = 1, 2. \tag{B16}$$

The inoculation of adult parasitoids, $I_{\mathrm{PA}}(t)$, is defined as a constant i_{PA} over the time interval $(i_{\mathrm{PA}}\mathrm{start}, i_{\mathrm{PA}}\mathrm{start} + 1]$ and 0 elsewhere.

Parameter Values. Tables B1 and B2 list the parameter values used in the simulations, unless otherwise specified in the main text.

Table B1: Host parameters

Host parameter	Description	Value
τ_{E}	Duration (in days) of egg stage	4.3[a]
τ_{L1}	Duration (in days) of early larval stage	13.3[a]
τ_{L2}	Duration (in days) of late larval stage	12.2[a]
τ_{P}	Duration (in days) of pupal stage	8.1[a]
τ_{A}	Duration (in days) of adult stage	5.5[b]
r	Daily adult fecundity (female eggs)	21[b]
d_{E}	Background mortality of eggs	.017[b]
d_{L1}	Background mortality of early larvae	0[b]

Table B1 (*Continued*)

Host parameter	Description	Value
d_{L2}	Background mortality of late larvae	0[b]
d_P	Background mortality of pupae	0[b]
d_A	Background mortality of adults	.1
c_{E2}	Rate of egg cannibalism by late larvae	Varied (see main text)
c_{11}	Mortality of H_{L1} from competition by H_{L1}	.00004
c_{12}	Mortality of H_{L1} from competition by H_{L2}	$\chi \times c_{11}$
c_{21}	Mortality of H_{L2} from competition by H_{L1}	$1/\psi \times c_{11}$
c_{22}	Mortality of H_{L2} from competition by H_{L2}	$\chi/\psi \times c_{11}$
χ	Ratio of competitive effects	20
ψ	Ratio of competitive sensitivities	10
i_{HA}	Inoculation of adults	2

[a] Data from table A1.

[b] Data from Sait et al. 1994*a*.

Table B2: Parasitoid parameters

Parasitoid parameter	Description	Value
τ_{PL}	Duration (in days) of juvenile stage	20[a]
τ_{PA}	Duration (in days) of adult stage	2[a]
d_{PL}	Background mortality of juveniles	.1
d_{PA}	Background mortality of adults	.1
a_1	Attack rate on early larvae	.005
a_2	Attack rate on late larvae	.025
k	Interference parameter	1
i_{PA}	Inoculation of adults	2
i_{PA}start	Timing of inoculation of adults	200

[a] Data from Harvey et al. 1994.

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