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Stage-structured competition and the cyclic dynamics of host–parasitoid populations

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Summary

1. Cyclic dynamics of various periods are pervasive in many insect populations where interactions with natural enemies are known to be important. How stage-structured processes within the host population, such as competition and cannibalism, affect these interactions has received little attention so far.
2. Using the well-studied laboratory host–parasitoid system of *Plodia interpunctella* and *Venturia canescens*, we explore a series of host–parasitoid models of increasing complexity. Specifically, we identify the circumstances under which stage-structured processes both within the host and parasitoid populations generate dynamical behaviour ranging from generation to true consumer–resource (multi-generation) cycles.
3. We find that both within-host interactions (strong competition and egg cannibalism by late instar larvae) and parasitoid recruitment structure (a developmental lag in the parasitism of larvae) can suppress long period cycles and promote host generation cycles. In short, we show that simple stage-structured processes in both host and parasitoid can modulate the strength of the consumer–resource interaction.
4. For some parameters we find more than one stable cyclic attractor. The presence of multiple attractors means that the same mechanism can give rise to cycles of different periods depending on initial population numbers. Because the host-alone system may exhibit transient dynamics for a substantial period, the timing of a parasitoid invasion can be crucial to the dynamical outcome.
5. We discuss the consequences of using a single descriptor of an ecological time series, the cycle period, to infer properties about the underlying system and its food web interactions.

Key-words: cannibalism, multiple attractors, *Plodia–Venturia*, population cycles, stage-structured models

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Introduction

A variety of population fluctuations are exhibited by a diverse range of organisms. Cyclic dynamics are particularly interesting because they provide evidence of potentially important processes involving nonlinear feedback that may be intrinsic to many species. As such, the pursuit of the underlying mechanisms that can account for such population cycles has driven a large body of ecological research (Kendall *et al.* 1999; Turchin & Hanski 2001; Berryman 2002). Much of this work has involved the formulation of a number of different mathematical models, often in tandem with detailed empirical studies, and it has emerged that processes such as predation and parasitism are thought to be key regulators of cyclic host populations. How natural enemies interact with and mould their prey populations and how this feeds back into enemy dynamics has been a subject of considerable debate in ecology (Hanski *et al.* 1993; Krebs *et al.* 1995.; Hudson, Dobson & Newborn 1998; Albon *et al.* 2002). In particular, the intimate interaction between insect hosts and their natural enemies has been the focus of many modelling studies (see Hassell 1978; Crawley 1992; Berryman 1999; Hassell 2000), because host–parasitoid or host–pathogen systems are often amenable to laboratory experiments under

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controlled conditions. These systems also provide examples of populations where stage structure plays an influential role in the dynamical outcome. In this paper, we are interested in understanding how the subtleties of stage-specific processes in the host can affect the types of cyclic dynamics that we might expect to see in the presence of a natural enemy. Our motivation stems from recent theoretical syntheses and, while we focus on a specific host–parasitoid system, many of the properties that we incorporate are common to predator–prey interactions.

The model system that we use centres on the interaction between the Indian meal moth, *Plodia interpunctella* (Hübner) (Lepidoptera; Pyralidae) (henceforth *Plodia*), and its parasitoid, *Venturia canescens* (Gravenhorst) (Hymenoptera; Ichneumonidae) (henceforth *Venturia*). Both the host and the parasitoid have distinct life stages with overlapping generations. The host life cycle is comprised of egg, larval, pupal and adult stages. The parasitoid life cycle is divided into a juvenile (within-host) and adult (free-living) stage.

In the next section, we briefly review the different cyclic dynamics that emerge from previous host–parasitoid models with this basic structure, before detailing the experimental and theoretical work on *Plodia–Venturia.*

CYCLIC DYNAMICS AND CONTINUOUS-TIME STAGE-STRUCTURED MODELS

Historically, host–parasitoid systems have been modelled within a discrete-time framework. This approach assumes that the parasitoid feeds on univoltine hosts with distinct, non-overlapping generations and, in the simplest case, the parasitoid's generation is perfectly synchronized with that of the host. Most of the models using this approach have evolved from generalizations (Hassell 1978) of the work of Nicholson & Bailey (1935). However, multivoltine hosts, such as *Plodia*, are best modelled using a continuous-time framework, which takes into account both within- and between-generation dynamics. Over the past 20 years, the importance of age/stage structure in host and parasitoid populations has led to the development of continuous-time models that incorporate lumped age/stage classes using delay-differential equations (Gurney, Nisbet & Lawton 1983). We adopt this formalism because it allows a flexible approach to examining the role of population structure in host–parasitoid dynamics.

The cyclic dynamics that result from stage-structured, continuous-time host–parasitoid interactions can be broadly classified as either: (i) 'generation' cycles with a period close to the generation length of the host; (ii) 'delayed feedback' cycles with a period generally between two and four generation lengths of the natural enemy; or (iii) longer period 'consumer–resource' cycles, such as those generated by the Lotka–Volterra predator–prey model (Lotka 1925; Volterra 1926). Each type of cyclic behaviour may be caused by different mechanisms. However, cycles that scale with the consumer (parasitoid) generation length are only possible if recruitment to the host population is approximately constant. This may be the case if the adult (reproductive) host stage is relatively long-lived and/or there is a refuge from predation. Delayed feedback cycles are then produced when there is some delayed density dependence in the parasitoid recruitment rate, which may be due to host-size dependent parasitoid feeding and oviposition (Murdoch, Briggs & Nisbet 1997; Briggs, Nisbet & Murdoch 1999). Parasitoid generation cycles can occur instead of the delayed feedback cycles under the somewhat restrictive condition that the parasitoid has a longer developmental stage than the host (Briggs *et al*. 1999). In the absence of any structure in the parasitoid recruitment rate, constant recruitment to the host population ensures that equilibrium behaviour is the only outcome.

On the other hand, host generation cycles result from pulses in host reproduction driven by preceding pulses in host survival. Generation cycles in eggs and larvae were first demonstrated in experimental studies of flour beetles (*Tribolium confusum*) (Chapman 1928), but more recently they have been shown to arise from host–parasitoid interactions as well as the within-host processes of competition and cannibalism (for a review see Knell 1998). In the simplest host–parasitoid models, host generation cycles are possible when the reproductive stages of both host and parasitoid are relatively short and when the parasitoid's developmental duration is approximately 1/2, 3/2, etc., that of the host's (Godfray & Hassell 1989). The restriction of a parasitoid development time of approximately 1/2 that of the host's can be relaxed when the parasitoid development is delayed within the host (koinobiont lifestyle) until the host has matured (Gordon *et al*. 1991). In a similar model developed to understand host–pathogen dynamics, Briggs & Godfray (1995) noted that at least part of the juvenile host stage needs to be invulnerable to attack for persistent generation cycles (implicit in the Godfray and Hassell model) and that a nonlinear transmission/parasitism function increases the likelihood of observing such cycles. With only a short-lived invulnerable host adult stage and linear density dependence in the parasitoid attack rate, longer period consumer–resource cycles are prevalent (Murdoch *et al*. 1987).

All these host–parasitoid models consider the role of host stage structure in terms of the parasitoid recruitment rate alone. The relative importance of within–host interactions, such as competition and cannibalism, has been somewhat overlooked in host–parasitoid models of multivoltine species, despite their ability to drive single-species cyclic dynamics (Gurney & Nisbet 1985) and their affect on stability in discrete-time (Bernstein 1986) and seasonally structured (Bonsall & Eber 2001) host–parasitoid systems. In this paper, we expand on recent work (Rohani *et al*. 2003) that has briefly addressed how interactions between host stages can modify the types of cyclic behaviour that we expect to observe, based on previous theory, in a host–parasitoid system. Our interest in exploring this issue was partly due to a

recent paper by Murdoch *et al*. (2002) in which they gave considerable empirical evidence for a general rule for cyclic univoltine species; generalist consumers should exhibit single-species dynamics whereas most specialist consumers should follow longer period consumer–resource dynamics. The basic ideas underlying these predictions date back to Holling's work on non-structured populations (Holling 1965), but Murdoch *et al*.'s synthesis is founded partly on theory from stage-structured populations. In the well-studied laboratory *Plodia*-*Venturia* community (see below), with complex stage-structured processes in both host life cycle and parasitoid attack, the introduction of the specialist parasitoid does not induce multi-generation cycles, although it affects quantitatively the host dynamics. We are therefore interested in investigating the general conditions under which we observe generation or multi-generation cycles in a specialist natural enemy system. We will show that simple stage-structured processes in both host and parasitoid can modulate the strength of the consumer–resource interaction.

PLODIA–*VENTURIA*: POPULATION BIOLOGY

Plodia has five larval instars, the first of which is invulnerable to attack from the parasitic wasp because it is too small (Sait *et al*. 1995), while a preference for larger larval stages is exhibited by the parasitoid (Sait *et al*. 1997). Regardless of the instar attacked, *Venturia* development is delayed until the host starts to pupate (Harvey, Harvey & Thompson 1994). Time-series data, illustrated in Fig. 1, show characteristic host generation cycles (period of approximately 42 days) in both the host-alone and host–parasitoid systems (Begon, Sait & Thompson 1995). Despite no qualitative change in the dynamics, the parasitoid severely suppresses host density, and, in particular, substantially deepens host troughs. Bjornstad *et al*. (2001) used nonlinear time-series analysis to show that the addition of the parasitoid actually increased the strength of coupling of the system, as evidenced by an increase in the embedding dimension (the number of density-dependent time lags required to best fit the data) (Takens 1981). This was in contrast to another natural enemy, the *Plodia interpunctella* granulovirus (PiGV), whose interaction also results in host generation cycles (Sait, Begon & Thompson 1994b) but is simply equivalent to modulating *Plodia*'s vital rates (with no increase in the embedding dimension).

Previous modelling work by Briggs *et al*. (2000) has examined the type of stage structure required to obtain the correct cycle period observed in the *Plodia*-alone populations; in the absence of natural enemies, *Plodia* populations fluctuate with a period approximately equal to the length of a generation. Briggs *et al*. (2000) found that it was necessary to include both asymmetrical larval competition (small larvae are more susceptible to competition than large larvae and exert a weaker competitive effect) and larval egg cannibalism in their model to reproduce the observed dynamics. Similar conclusions were reached by Bjornstad *et al*. (1998) who fitted mechanistic time-series models to these data. They found that incorporating egg cannibalism added another significant lag to their model, which more accurately captured the patterns in the data.

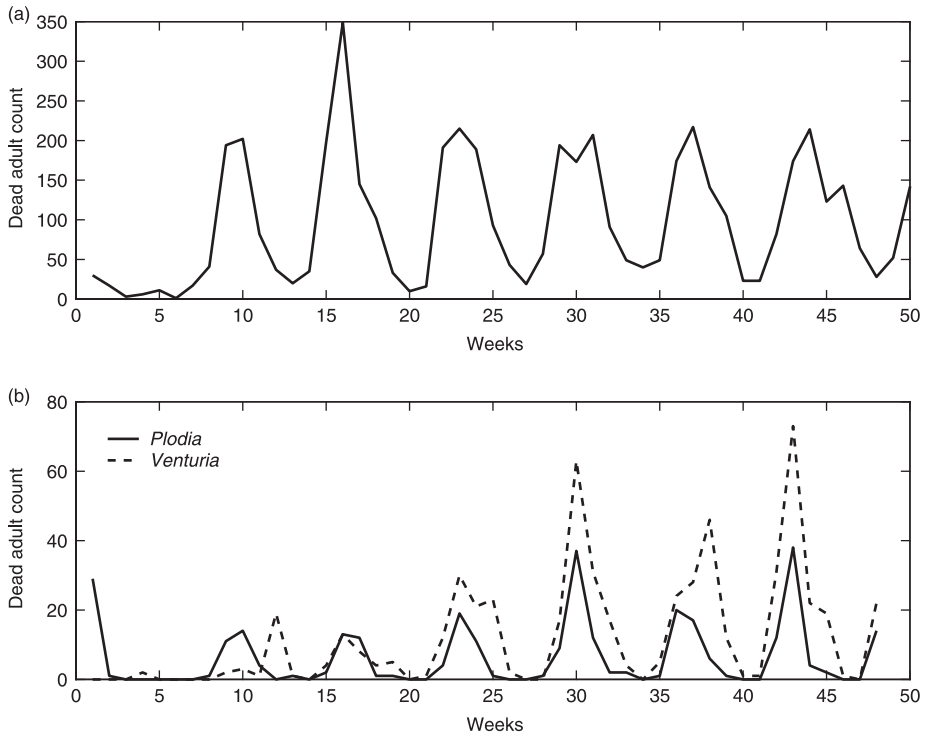

Fig. 1. Weekly data from (a) *Plodia*-alone and (b) *Plodia*–*Venturia* laboratory populations both show cycles with a period of approximately one host generation (around 42 days) in *Plodia* and *Venturia* populations.

In this paper we systematically explore the role of host stage structure, in driving different types of cycle in the host–parasitoid system. As in Briggs *et al*. (2000), this is done in a step-by-step manner, starting with the simplest model and successively adding more stage-structured processes to the host population. We identify the processes within the host population that lead to true consumer–resource (multi-generation) cycles and explore how structure in the parasitoid recruitment rate can alter these results. We also investigate the presence of multiple cyclic attractors and discuss their dynamical implications for the timing of a parasitoid invasion.

Modelling approach

We investigate host–parasitoid dynamics using the delay-differential equation framework pioneered by Gurney *et al*. (1983). Due to model complexity, we rely primarily on numerical simulation of the equations and power spectrum analysis of the resulting time series to examine dynamics (see Appendix for details). The simulations broadly follow the protocol of the *Plodia–Venturia* laboratory experiments that motivate our work; *Plodia* populations are established before *Venturia* is allowed to invade.

In our basic host–parasitoid model, the host population has four distinct stages: egg, larval, pupal and adult, each with a fixed maximum life span. Initially, we lump the five larval instars into a single class, but in the more complex models that we consider we will divide the class into small and large larvae, which allows us to examine the effects of differential competition and parasitism within the larval stage. The parasitoid population has two distinct stages: juvenile and adult, each also with a fixed maximum life span. Thus, reproduction by both adult hosts and parasitoids is limited to a fixed period of time. Each stage of both host and parasitoid is subject to a constant density-independent mortality rate. As we investigate the effects of host competition, we add density-dependent mortality to some of the host stages. This is done in a simple linear manner to minimize the number of extra parameters that we need to estimate or explore. A key feature of the parasitoid attack, as is the case in many host–parasitoid systems, is that the host is only vulnerable to parasitism during the larval stage. Parasitism is modelled via a nonlinear attack function with a measure of density dependence (Godfray & Hassell 1989; Hughes, Harvey & Hubbard 1994; Rohani, Godfray & Hassell 1994a). A schematic diagram of the model is given in Fig. 2; the equations and parameter values are stated in the Appendix.

Host stage structure is added to the model systematically as we include the following key features of many insect life cycles with a limited food resource: within-stage competition, between-stage competition and egg cannibalism. To assess the importance of the host population on the host–parasitoid dynamics we compare the host period of the host–parasitoid system with the host period of the host-alone system.

Host–parasitoid models

UNIFORM LARVAL COMPETITION

We begin by introducing host larval density-dependent mortality, $-cH_L^2$, where H_L denotes the density of the host larval class and the constant c is an indicator of the strength of competition for resources. In Fig. 3, we show how the cycle period of the host and parasitoid populations alters as we vary host fecundity and larval competition strength for a fixed rate of parasitism. In the host-alone case, the system exhibits generation cycles for all but small values of host fecundity when the population is at a stable equilibrium (white region). As discussed in Briggs *et al*. (2000), these cycles are slightly longer than the host generation length because the peak in adult numbers is not produced by adults of the previous peak but rather by adults in the tail of the previous peak; intense larval competition experienced by the offspring of adults in the peak allows the offspring of adults in the tail to suffer less mortality and produce the next peak in adult numbers.

In the host–parasitoid case, the change in qualitative behaviour occurs as the competition strength is increased. An illustration of the different types of behaviour is given in Fig. 4. For low levels of intraspecific larval competition the parasitoid is able to disrupt the host cycles, although the host dynamics still exhibit a strong generational signature. By attacking the larvae produced by the peak in host adult numbers, adult parasitoids produce a large offspring cohort, which attacks those larvae that would have formed the following peak in host adult numbers. Thus, as parasitoids deplete their resource, their numbers fall until the host population

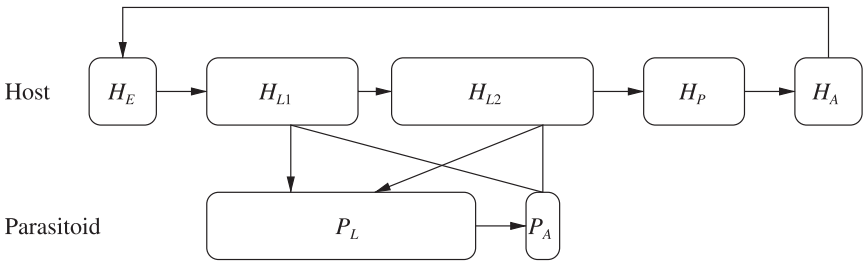

Fig. 2. A schematic interpretation of the life cycle of the basic host–parasitoid model. The host stages are egg (H_E), larval (H_{L1}, instars 1–3; H_{L2}, instars 4–5), pupal (H_P) and adult (H_A). The parasitoid stages are juvenile (P_L) and adult (P_A).

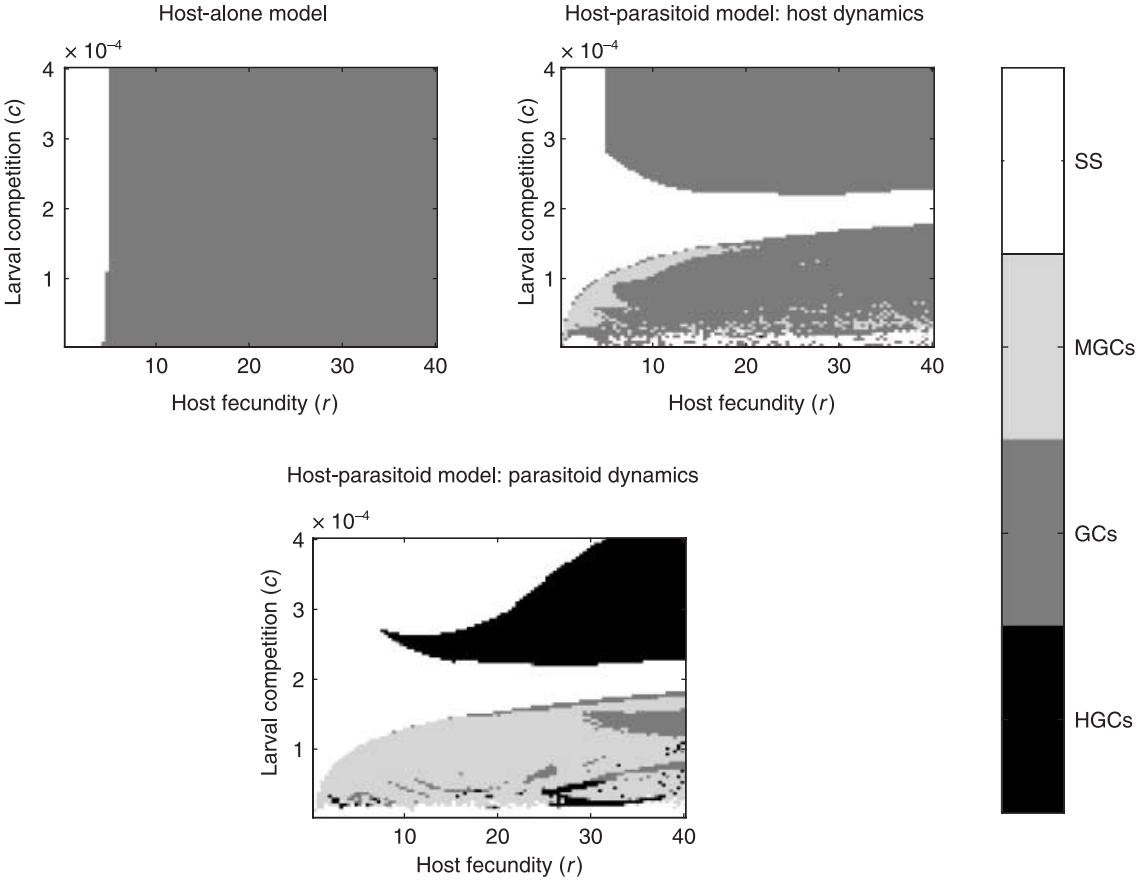

Fig. 3. Behaviour of the host-alone and host–parasitoid models with uniform larval competition. The shaded diagrams show the period of host or parasitoid cycles scaled with the host generation length as both the fecundity (r) and strength of competition (c) are increased. The most significant changes in host and parasitoid dynamics occur with increasing host larval competition. Multi-generation cycles are only possible for intermediate levels of the competition parameter. Simulations are initialized with 10 adult hosts. In the host–parasitoid model, two adult parasitoids are added after 200 days. Parameter values are given in the Appendix (ULCM values). Key: HGCs, half-generation cycles; GCs, generation cycles; MGCs, multi-generation cycles; SS, steady-state.

can increase again, creating the longer period irregular cycles as shown in Fig. 4(a). As intraspecific competition is increased, the parasitoid is unable to have the same numerical effect on the host dynamics and simply suppresses the cycles, which gives rise to constant population levels (Fig. 4b). If the strength of competition is increased further, the parasitoid becomes entrained on the dynamics of the host, although, as Fig. 4(c) suggests, both populations appear to show quasi-periodic behaviour (Rohani, Miramontes & Hassell 1994b). We also note that the parasitoid population exhibits two peaks for every single peak in the host population. This is due to the same mechanism that gives rise to multi-generation cycles when there is less competition (more larvae to attack); a cohort of parasitoid adults and their offspring are able to parasitize the same generation of hosts. In Fig. 3, the bifurcation diagram of parasitoid population dynamics indicates half host-generation cycles for these parameter values because the length of a parasitoid generation, which corresponds to the dominant peak of the power spectrum, is approximately half the length of a host generation. Eventually, larval competition is so strong that the parasitoid is unable to sustain itself and the system returns to host-alone dynamics (Fig. 4d). In summary, longer period cycles are only possible for moderate levels of within-stage host larval competition. Note that, for a fixed strength of competition, longer cycles can be obtained by increasing the rate of parasitism, thereby demonstrating the ability of the parasitoid to modulate the host dynamics (results not shown).

ASYMMETRIC LARVAL COMPETITION

We now divide the host larval class into two groups: small larvae (instars 1–3; denoted by H_{L1}) and large larvae (instars 4–5; denoted by H_{L2}). We then assume that both larval classes are subject to within-stage and between-stage competition, arising from both competition for resources and cannibalism, and that competition is asymmetric between the two classes. Thus, instead of a single competition parameter, c, we now have four, c_{11}, c_{12}, c_{21} and c_{22}, where c_{ij} represents the competitive effect of class H_{Lj} on class H_{Li}. As in the *Plodia* model of Briggs *et al.* (2000), we rewrite three of the parameters as scaled multiples of the fourth in order to focus on the relative competitive effects of each class. Specifically, we denote $c_{12} = \chi c_{11}$, $c_{21} = c_{11}/\psi$ and $c_{22} = \chi c_{11}/\psi$. This

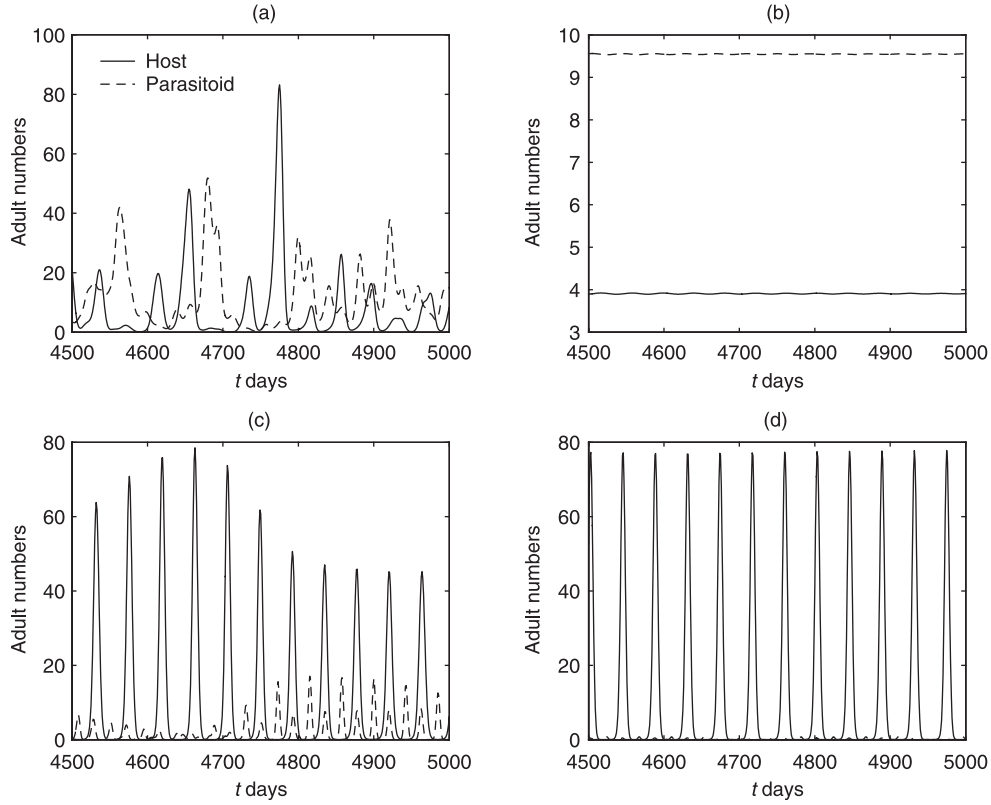

Fig. 4. Typical time series generated by the uniform larval competition model as the competition parameter, c, is increased for a fixed reproduction rate, $r = 21$. From (a)–(d) the parameters are on a vertical transect through the host–parasitoid bifurcation diagrams shown in Fig. 3. This shows the progression from multi-generation cycles to steady-state dynamics to host generation cycles to virtual extinction of the parasitoid population with increasing host larval competition. The values of the competition strengths are (a) $c = 0{\cdot}0001$; (b) $c = 0{\cdot}0002$; (c) $c = 0{\cdot}00025$; (d) $c = 0{\cdot}0003$; all other parameter values are given in the Appendix.

implies that χ ($= c_{i2}/c_{i1}$) represents the ratio of the competitive effects, and ψ ($= c_{1j}/c_{2j}$) represents the ratio of the competitive sensitivities. For example, if we assume that $\chi > 1$ then the effects of the large larvae are greater than those of the small larvae, and if $\psi > 1$ then small larvae are more sensitive to competition than large larvae. Because this is a qualitative feature of the *Plodia–Venturia* system we focus on these cases. We assume that the rate of parasitism is the same for both classes to avoid imposing any differential effects from the parasitoid attack. Later, we will discuss what happens when this assumption is relaxed.

In the host-alone model, the system exhibits host generation cycles or steady population levels when there is little asymmetric competition, otherwise the population fluctuates with a period equal to half the host generation length (Fig. 5). Two cycles can propagate through the population for each generation because the strongest competitive effects, i.e. the effect of large larvae on small larvae, occur for a relatively short period. This is in contrast to the previous model with uniform competition, where the effects are the same throughout the entire larval period. When the parasitoid is added the populations fluctuate with longer period cycles, except when the ratio of the competitive effects is very high (Fig. 5). However, as is illustrated in Fig. 6, the nature of these multi-generation cycles changes as χ is increased. For small χ, there is greater host larval survival resulting in very high parasitism, which severely depletes host numbers and subsequently reduces the parasitoid population to very low densities. During these troughs in parasitoid numbers, the host population begins to cycle with periods of half its generation length, as it would if the parasitoid was absent (Fig. 6a). As χ is increased and the effects of large larvae become significantly greater than those of small larvae, the parasitoid engages in true consumer–resource dynamics (Fig. 6b).

EGG CANNIBALISM

In the previous section, we showed that asymmetric competition, and in particular large larvae having a dominant competitive effect over small larvae, is conducive to generating multi-generation cycles in the host–parasitoid model. However, we know from time-series data of *Plodia–Venturia* that neither the host-alone nor host–parasitoid systems exhibit such dynamics (Begon *et al*. 1995). A key feature of the *Plodia* populations on limited resources that is so far missing from the model is egg cannibalism (Richards & Thomson 1932). Indeed, egg cannibalism is a key force in the life cycle of a number of organisms, especially in insects (Hastings & Costantino 1987; Dickinson 1992), snails

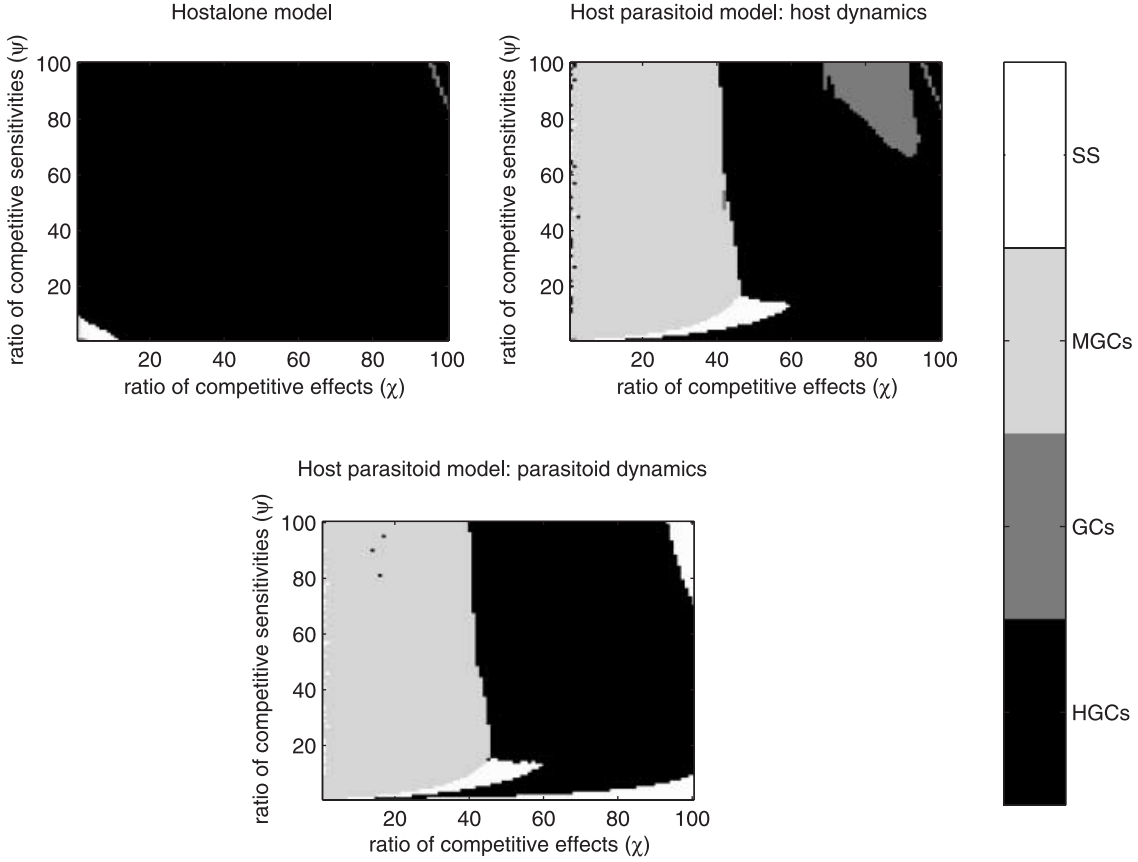

Fig. 5. Behaviour of the host-alone and host–parasitoid models with asymmetric larval competition. The shaded diagrams show the period of host or parasitoid cycles scaled with the host generation length as both the ratio of competitive effects (χ) and sensitivities (ψ) are increased. Host and parasitoid populations cycle with long periods when χ is below a certain threshold and with half-generation cycles when χ is above it. Simulations are initialized as in Fig. 3. Parameter values are given in the Appendix (ALCM values). Key: HGCs, half-generation cycles; GCs, generation cycles; MGCs, multi-generation cycles; SS, steady-state.

(Baur 1988) and some fish (Vinyoles, Cote & de Sostoa 1999). Both Briggs *et al.* (2000) and Bjornstad *et al.* (1998) found egg cannibalism to be crucial in obtaining the observed dynamics in the host-alone model. In Fig. 7 we demonstrate the effects of egg cannibalism on both the host-alone and host–parasitoid system. To make comparisons with Fig. 5, we show the dynamical behaviour as we vary both the strength of egg cannibalism (c_{E2}) and the ratio of the competitive effects of each stage (χ). We fix the ratio, ψ, such that smaller larvae are much more sensitive to competition than large larvae. With $\chi > 1$ the greatest competitive effect is that of large larvae on small larvae.

In the absence of the parasitoid, the host exhibits half-generation cycles for highly asymmetric competition and small values of egg cannibalism. As egg cannibalism is initially increased, the half-generation cycles are damped giving rise to steady-state dynamics, while very high levels of egg cannibalism give rise to generation cycles. The inclusion of egg cannibalism results in generation cycles because it acts to prevent the second pulse in host numbers propagating through the host population. These generation cycles are, however, different from those in the first model of uniform competition, since they are almost exactly a host generation in length. Briggs *et al.* (2000) point out that it is the combination of asymmetric competition and egg cannibalism that cause these cycles.

In the presence of the parasitoid there is a clear bifurcation from multi-generation to generation cycles as c_{E2} is increased, and from steady populations to half-generation cycles as χ is increased. When egg cannibalism is low, the parasitoids can attack a significant proportion of the larvae, which leads to multi-generation cycles (Fig. 7a) in a similar manner to the previous model. Increasing the level of egg cannibalism reduces egg survival and subsequently the number of larvae in the current cohort. This results in reduced cannibalism and large larval numbers in the next cohort, giving rise to generation cycles (Fig. 7b) in which the parasitoid plays a more limited role (see also Rohani *et al.* 2003). We note, however, that the bifurcation in the host–parasitoid system does not coincide with a change in behaviour in the host-alone case. On further inspection, the host population is often exhibiting transient behaviour at the time of the parasitoid introduction. A preliminary investigation revealed that the timing of the parasitoid invasion affects the long-term behaviour of the host–parasitoid model for parameters in the vicinity of the bifurcation. This led us to explore more generally whether multiple attractors exist in the model and we present these results in a later section.

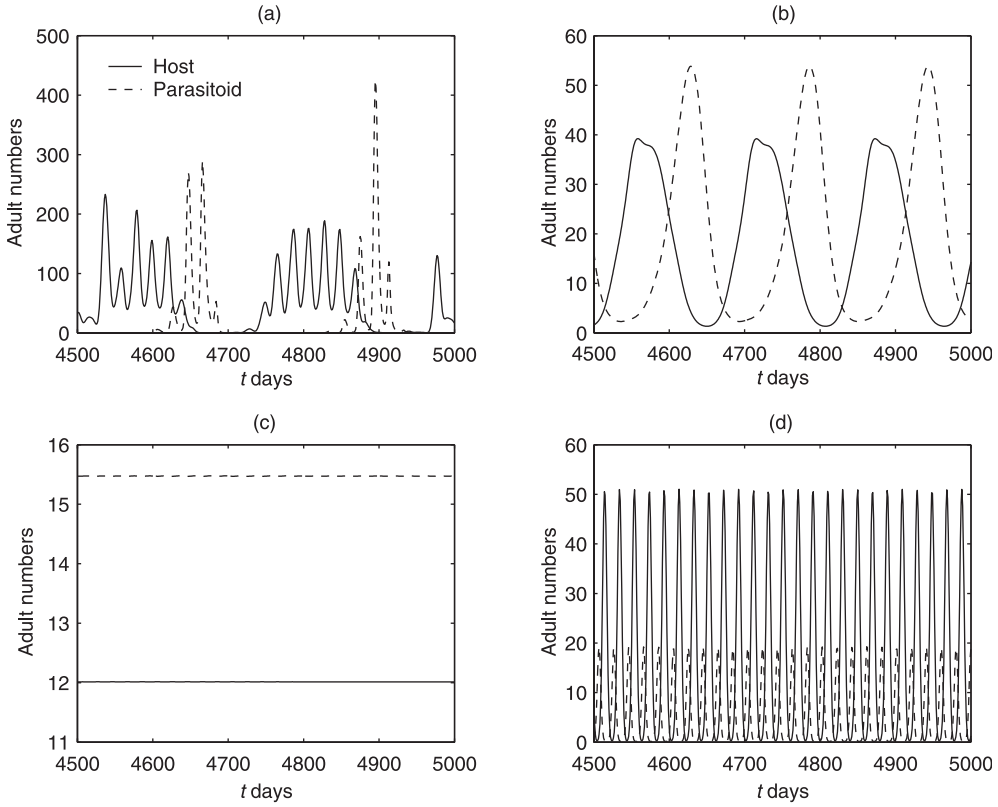

Fig. 6. Typical time series generated by the asymmetric larval competition model as the ratio of the competitive effects, χ, is increased for a fixed value of the ratio of the sensitivities, $\psi = 10$. From (a)–(d) the parameters are on a horizontal transect through the host–parasitoid bifurcation diagrams shown in Fig. 5. This shows the progression from multi-generation cycles to steady-state dynamics to host half-generation cycles as the competitive pressure of large larvae on all larvae is increased. Note the very different appearance of the cycles in graphs (a) and (b) although they can both be described as having a multi-generational period. The values of the ratio of competitive effects are (a) $\chi = 10$; (b) $\chi = 30$; (c) $\chi = 50$. (d) $\chi = 80$; all other parameter values are given in the Appendix.

THE EFFECTS OF PARASITOID ATTACK STRUCTURE

Here we briefly show what happens when we add parasitoid attack structure to the previous models, which now incorporates two key features of the *Plodia–Venturia* interaction. First, the parasitoid preferentially attacks large larvae (Sait *et al*. 1997) so that the rate of parasitism is higher in the large larval class than in the small larval class. Second, parasitoid juvenile development is delayed when an adult attacks an early instar larva (Harvey *et al*. 1994). This means that the parasitized host continues its development into the large larval class before the parasitoid egg begins its own development. We are interested in how these properties influence our conclusions about the impact of host stage structure and we begin by increasing the attack rate on the large larval class. For asymmetric competition, the system can only exhibit true consumer–resource cycles (multi-generation cycles) if the parasitoid is sufficiently effective at attacking late instar larvae to counteract the competitive effect of the large larvae on the small larvae (Fig. 8a). When egg cannibalism is incorporated, the system can only exhibit true consumer–resource cycles if the level of cannibalism is low (Fig. 8c). If the parasitoid development in small larvae is delayed then the populations fluctuate with a period of about one host generation length for most parameter values (Fig. 8b,d). This result holds for a range of juvenile parasitoid development times. The developmental lag synchronizes the development of the parasitoid with that of the host, which is typical of koinobiont parasitoids. It prevents the boom–bust scenario characteristic of multi-generation, consumer–resource cycles because when the parasitoids attack a peak in small larvae numbers their offspring do not all emerge as adult parasitoids at the same time. This effect is reinforced when the developmental delay is applied to the parasitism of all larvae, regardless of instar (results not shown). Gordon *et al*. (1991) found that adding a developmental lag to their basic model, which lacked host competition and cannibalism, also increased the region of parameter space that gave rise to generation cycles.

The presence of multiple attractors

In this section, we take the egg cannibalism model with differential parasitism and systematically explore whether different initial conditions give rise to different dynamical outcomes. Each of the host–parasitoid models has

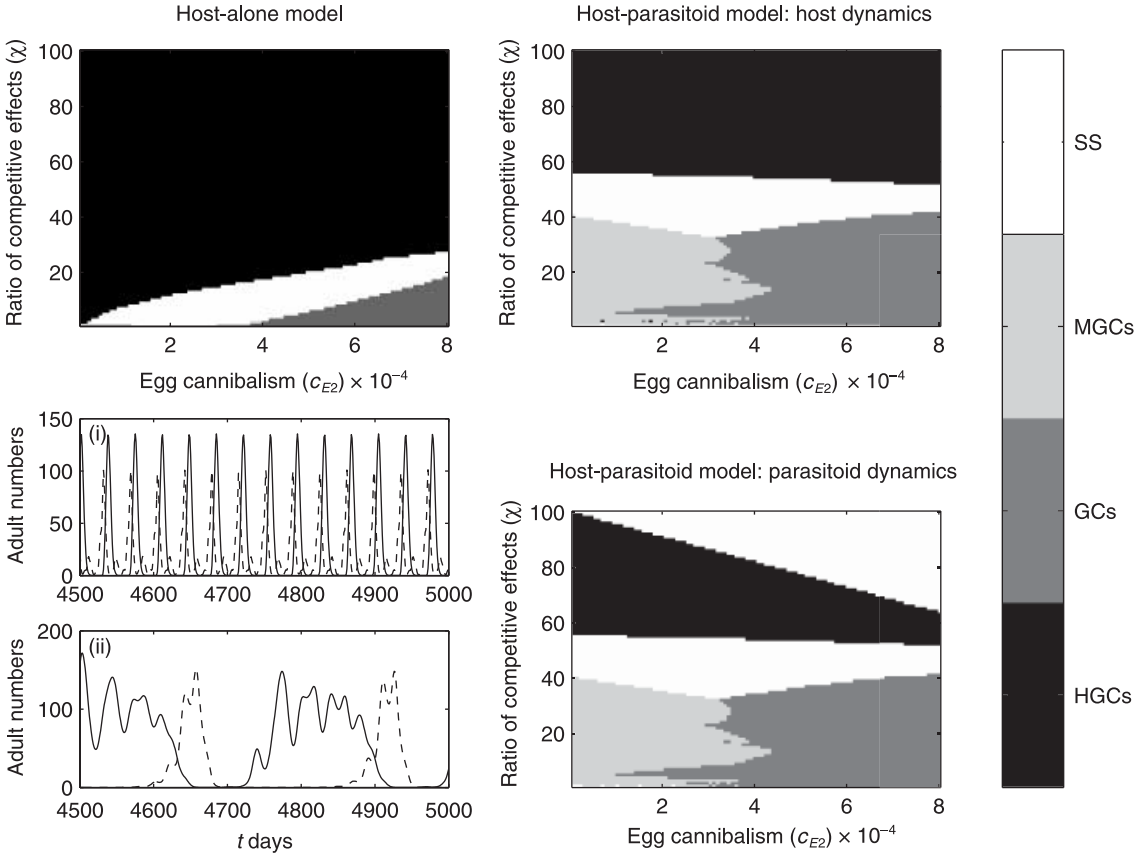

Fig. 7. Behaviour of the host-alone and host–parasitoid models with larval competition and egg cannibalism. The shaded diagrams show the period of host or parasitoid cycles scaled with the host generation length as both the strength of egg cannibalism (c_{E2}) and the ratio of competitive effects (χ) are increased. If χ is not too high then the populations cycle with long periods for small amounts of egg cannibalism, but with generational periods for higher levels of egg cannibalism. Graphs (a) and (b) illustrate typical time series generated by the model as c_{E2} is increased for a fixed value of $\chi = 20$ (hosts are denoted by the solid line, parasitoids by the dashed line). Simulations are initialized as in Fig. 3. The values of the egg cannibalism parameter are (i) $c_{E2} = 0{\cdot}0002$; (ii) $c_{E2} = 0{\cdot}0006$; all other parameter values are given in the Appendix (ECM values). Key: HGCs, half-generation cycles; GCs, generation cycles; MGCs, multi-generation cycles; SS, steady-state.

three equilibria: the trivial equilibrium with no hosts or parasitoids; the host-alone equilibrium when no parasitoids are present; and the coexistence equilibrium when both hosts and parasitoids are present. Although with the inclusion of egg cannibalism, we cannot explicitly solve the equations for the non-trivial equilibria, it is straightforward to find them numerically. In our simulations we start from one of these equilibria and then perturb it by adding or removing adult hosts and parasitoids. We do this using the inoculation procedure of Gurney *et al.* (1983). In Fig. 9(a–c), we illustrate the long-term behaviour of the system as we vary both the egg cannibalism parameter (c_{E2}) and the initial perturbation of adult hosts (the initial perturbation of parasitoids is kept fixed). To verify that these are not transient dynamics we have run simulations for more than 50 000 host generations and the same dynamics persist in each case for the entire period. Starting close to the trivial equilibrium, the bifurcation from multi-generation cycles to host generation cycles shifts as the size of the perturbation increases. Specifically, generation cycles are seen for lower levels of egg cannibalism when more host adults are initially added to the system. Although the shift in the bifurcation is less dramatic starting from the other (non-trivial) equilibria – indeed multi-generation cycles are more prevalent – it still exists.

If we fix egg cannibalism at some intermediate level close to the bifurcation, we can explore how perturbing host and parasitoid numbers simultaneously alters the dynamical outcome. The results of this are shown in Fig. 9(d) for the coexistence equilibrium. The multi-generation cyclic attractor is reached if either adult hosts or adult parasitoids are removed from the system. Otherwise the populations exhibit generation cycles. All this evidence implies that for at least part of the parameter space, both generation and multi-generation cycles are locally stable. In particular, perturbations of the trivial equilibrium, which is the set-up closest to many experimental protocols, lead to different outcomes for a greater range of the egg cannibalism parameter. We note that for the range of egg cannibalism parameters shown, the host-alone system does not exhibit generation cycles. However, in the host-alone model, the host will often exhibit transient generation cycles before settling to its long-term equilibrium behaviour. We speculate that if the parasitoid is added while the host

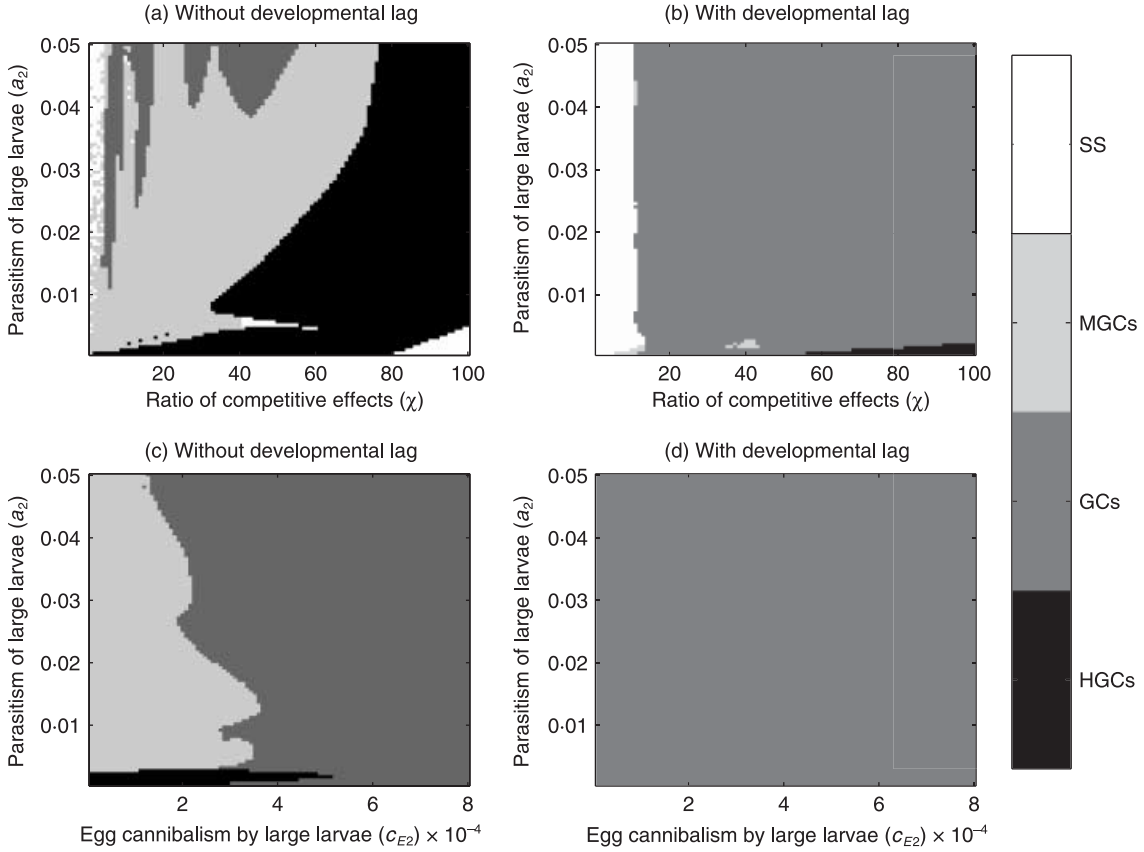

Fig. 8. Behaviour of the asymmetric competition and egg cannibalism host–parasitoid models with differential parasitism and a developmental lag in the parasitism of small larvae. The shaded diagrams show the period of parasitoid cycles scaled with the host generation length (the period of host cycles follows the same pattern). In (a) and (c), varying the parasitism rate of large larvae while keeping that of small larvae fixed only has the effect of shifting the bifurcation point between multi-generation and generation/half-generation cycles. However, as shown in (b) and (d), if the parasitoid also delays its development when parasitizing small larvae then both host and parasitoid fluctuate with a period of one host generation for most parameter values. Simulations are initialized as in Fig. 3. Parameter values are given in the Appendix. Key: HGCs, half-generation cycles; GCs, generation cycles; MGCs, multigeneration cycles; SS, steady-state.

population is still in the transient phase then the interaction with the parasitoid is able to lock the system onto generation cycles. Furthermore, preliminary investigation suggests that delayed parasitoid development in early instar larvae results in generation cycles for a far wider range of initial conditions.

Discussion

It is often assumed that multi-generation cycles in population dynamics are a signature of strong coupling between predator and prey, consumer and resource. We have asked whether host stage structure can disrupt this signature. Both within host interactions (strong competition and egg cannibalism by late instar larvae) and parasitoid recruitment structure (a developmental lag in the parasitism of small larvae) can suppress multi-generation cycles and promote host generation cycles. Of course, host competition and cannibalism are strong intraspecific processes and their disruption of multi-generation cycles is not necessarily structure-specific. However, the ability of the host or parasitoid to induce host generation cycles is dependent on having invulnerable and vulnerable stages in the host population. Interestingly, knowing the period of the host-alone dynamics does not necessarily allow us to make predictions about the host–parasitoid system. Indeed, the presence of multiple attractors dictates that for some parameter values either multi-generation or generation cycles are possible depending on initial conditions. This suggests that in our host–parasitoid system we cannot automatically deduce the type of natural enemy interaction by looking at the cycle period of either the host or the parasitoid. However, it does highlight how the timing of the natural enemy invasion may fundamentally alter the long-term dynamics of the system.

The results of our modelling demonstrate that coupling between host stages can dominate host–parasitoid dynamics even when the parasitoid interaction is also highly structured; when the host-alone model exhibits generation cycles, the host–parasitoid model usually has the same qualitative behaviour (see Summary of EC models in Table 1). However, the fact that the parasitoid population cycles with a period close to the host

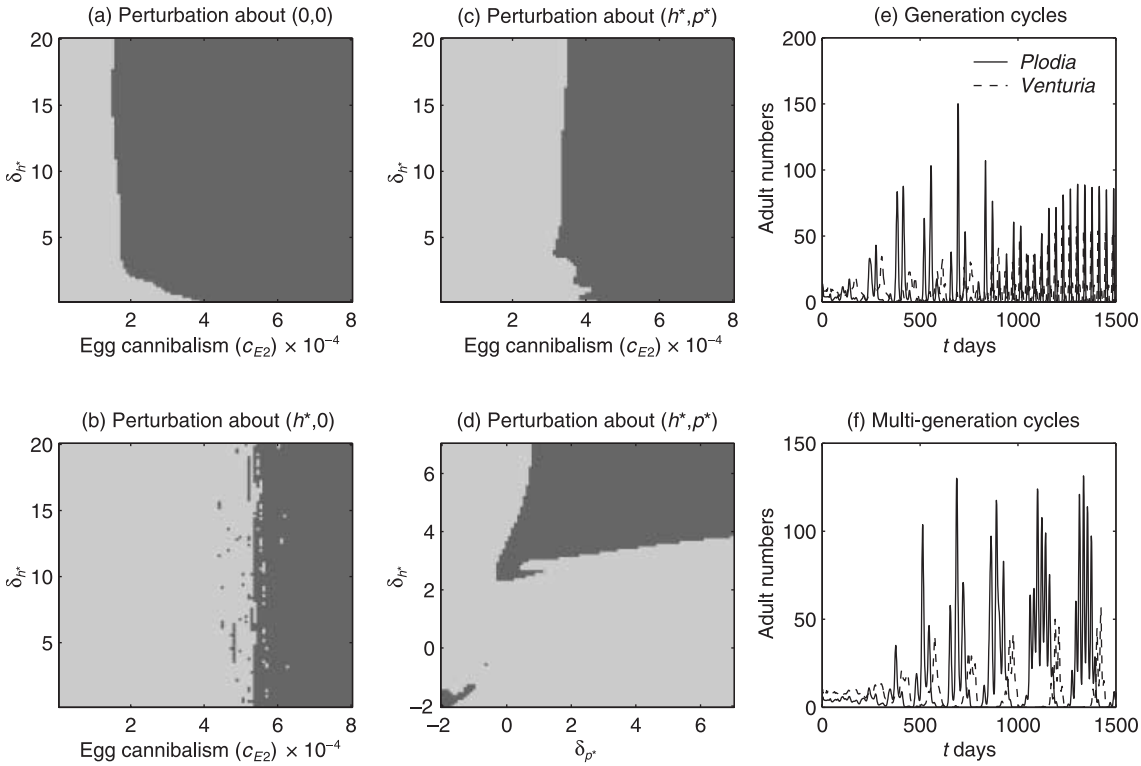

Fig. 9. Presence of multiple attractors in the egg cannibalism model. Each of the shaded diagrams (a–c) shows the long-term dynamical outcome of perturbations (δ_h) about a particular steady state as we vary the strength of egg cannibalism (c_{E2}). Light grey shading indicates multi-generation cycles, dark grey shading indicates generation cycles in both host and parasitoid dynamics. For the same value of the egg cannibalism parameter, the populations cycle with different periods depending on the initial conditions. In (d) different perturbations of adult host (δ_h) and adult parasitoid (δ_p) numbers about the coexistence equilibrium lead to different dynamical outcomes. The time series labelled (e) and (f) display examples of the different model outcomes from perturbations about the coexistence steady state. All the simulations for this figure are initialized at the particular equilibrium values and additional host adults (δ_h) and parasitoid adults (δ_p) are introduced to the system. For the zero equilibrium, we add two parasitoid adults after 15 days, which is when the first cohort of late instar host larvae are emerging. (The parasitoid has no chance of persisting if added immediately because we inoculate with host adults and the parasitoid adult life span is shorter than the host egg stage.) For the host-alone and host–parasitoid equilibrium, we introduce two parasitoid adults at the start because all host stages are present initially. In (d–f), the egg cannibalism parameter is fixed at $c_{E2} = 0{\cdot}00036$, and in (e) $\delta_h = 10$, and in (f) $\delta_h = 2$. The parasitism parameters are $a_1 = 0{\cdot}005$ and $a_2 = 0{\cdot}025$; all other parameter values are as given in the Appendix.

generation time does not necessarily imply that the parasitoid is entrained on the dynamics of its host. In some cases, the presence of the parasitoid is necessary for the host population to cycle with a period close to one generation. In the simplest scenario, where the host-alone model includes no density dependence and the population will grow without limit, it is obvious that the parasitoid interaction is driving the cycles in the host–parasitoid system (Godfray & Hassell 1989). When the host-alone model includes density dependence and the resulting dynamics are half-generation cycles or a stable equilibrium, then the parasitoid can still induce generation cycles for certain parameter values (see Summary of ALC + parasitoid developmental lag model and EC models presented in Table 1). This is especially the case when parasitoid development is delayed until the host has completed a substantial part of its development. In our host–parasitoid models, generation cycles therefore arise from both intraspecific competition and interaction with natural enemies (Knell 1998). It is also apparent that a subtle combination of larval competition and parasitism is sometimes key to producing generation cycles. At present, the models assume that once larvae are parasitized they escape competition and exert no competitive effects on healthy larvae. Current experimental investigation of competition between parasitized and healthy larvae offers the possibility of extending the model to consider the effects of larvae experiencing competition and parasitism simultaneously (Lane & Mills 2003).

Coexisting stable attractors, as seen in our model of egg cannibalism, have been reported in other stage-structured models (Hastings & Costantino 1987; Briggs *et al*. 1999), and recently in an experimental system (McCauley *et al*. 1999). Hastings & Costantino (1987) explored a single-species model for the flour beetle genus *Tribolium* and proved that for an intermediate range of the egg cannibalism parameters two locally stable solutions existed – an equilibrium solution and a cyclic solution with a period approximately equal to the host's development time. Briggs *et al*. (1999) showed that in a host–parasitoid model with delayed parasitoid recruitment,

Table 1. Summary table of the key features of each host–parasitoid model that we explore, along with a comparison between the different dynamical outcomes of the host-alone and host–parasitoid models. For each model formalism, we list the possible host-alone dynamics and then for each of these the corresponding host dynamics that we observe in the host–parasitoid model. (Host dynamics are also representative of the qualitative behaviour of the parasitoid in the host–parasitoid system). Looking at the models with uniform parasitism (ULC, ALC and EC), our results demonstrate that parameters specific to the host can alter the qualitative behaviour of the host–parasitoid system while having no effect on the qualitative behaviour of the host-alone system. We note, however, that generation cycles in the host-alone EC model always correspond to generation cycles in the equivalent host–parasitoid models, which is the closest situation to the data from the laboratory *Plodia–Venturia* system. SS = steady state, GCs = generation cycles, HGCs = half-generation cycles, MGCs multi-generation cycles. Generation refers to the host generation length

Model	Possible host-alone dynamics	Corresponding host-parasitoid dynamics
Uniform larval competition (ULC)	SS	SS MGCs
	'Long' GCs	MGCs SS 'Long' GCs
Asymmetric larval competition (ALC)	SS	MGCs
	HGCs	MGCs SS HGCs
ALC + differential parasitism	SS	MGCs
	HGCs	MGCs HGCs SS
ALC + parasitoid developmental lag	SS	SS
	HGCs	GCs
Egg cannibalism (and asymmetric comp) (EC)	HGCs	MGCs GCs SS HGCs
	SS	MGCs GCs
	GCs	GCs
EC + differential parasitism	HGCs	MGCs GCs
	SS	GCs
	GCs	GCs
EC + parasitoid developmental lag	HGCs	GCs
	SS	GCs
	GCs	GCs

delayed feedback cycles were possible in regions of parameter space where the equilibrium was locally stable. What is interesting in our model is that the two coexisting attractors are cycles of widely different periods, and that often these dynamics – generation cycles and long period consumer–resource cycles – are assumed to arise from different mechanisms rather than different initial conditions.

This property of the model has consequences for the timing of a parasitoid invasion. Because the host-alone system may exhibit transient dynamics for a substantial period of time, the point of introduction of the parasitoids into the system may be crucial to the dynamical outcome. Unless the host-alone model is at its asymptotic state, the invasion of the same number of parasitoids at different time points is equivalent to starting from different initial conditions and may, thus, lead to different outcomes in the regions of multiple attractors. This is an area of current investigation and it remains to be seen whether the stochasticity present in real systems changes these conclusions. Preliminary results suggest that generation cycles are more stable than the coexisting multi-generation cycles in the presence of demographic noise (H. J. Wearing *et al.*, unpublished data). Our analyses indicate that it is a combination of within host and host–parasitoid stage-structured interactions that give rise to multiple attractors. These results emphasize the potential importance of stage-structured interactions and why for some organisms they cannot be ignored when exploring multi-species communities (Polis & Strong 1996).

Finally, an area of current debate is whether the information gleaned from the dynamics of a single species is enough to determine its interactions within the broader ecological web, and whether simple models that focus on one or two species are really useful in providing insight into these complex dynamics. Attempts at disentangling trophic interactions from single-species time series date back to Royama (1977), who first made the connection between density dependence and the order of ecological time series. Techniques from time-series analysis (Box & Jenkins 1970; Cheng & Tong 1992) are used to find the number of significant density-dependent time lags that best describe single-species population data, and inferences are made about the type of predator–prey coupling (Royama 1992; Stenseth *et al*. 1997). For example, Bjornstad *et al*. (2001) contrasted the order or embedding dimension of a host-alone, host–parasitoid and host–pathogen system, and showed that despite qualitatively similar dynamics, specifically cycles of approximately the same period, there was an increase in system dimension between the host and host–parasitoid systems (although there was no increase in dimension between the host and host–pathogen systems). A slightly different, but not unrelated, approach infers the strength of predator–prey interactions from an estimate of cycle period (Royama 1992; Berryman 1999). Recently, Murdoch *et al*. (2002) have analysed a wide range of population time series to demonstrate that, under certain assumptions, generalist consumers can be distinguished from specialist consumers by looking only at the cycle period of the consumer scaled with the consumer generation length.

Using dimensionality as a measure of the strength of coupling usually requires some point of reference, i.e. we need to know the dimension of the host's dynamics in the absence of a natural enemy or, for purposes of comparison, in the presence of a different natural enemy. Calculating the cycle period does not require such a reference. However, our work highlights that in a highly age-structured system, different mechanisms can generate similar dynamics (host-generation cycles can be induced via intraspecific competition or interaction with a natural enemy), but the same mechanism can also give rise to qualitatively different dynamics (the same model and parameter values may lead to different periods dependent on our starting point). In such cases, the period of the data on its own would be inconclusive. It is clear for these systems that studying the dynamics of the host or natural enemy independently of one another could result in misleading conclusions about the type of interaction.

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Appendix

In this appendix we formulate the equations for the most complex host–parasitoid model discussed in the paper. The other models are subsequently obtained by setting certain parameter values to zero and/or equal to one another. Parameter values are listed in a table following the equations.

MODEL 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:

$$\frac{dH_E(t)}{dt} = \overbrace{R_E(t)}^{\text{recruitment}} - \overbrace{(c_{E2}H_{L2}(t)}^{\text{cannibalism by lge larvae}} + \overbrace{d_E)}^{\text{background death rate}} H_E(t) - \overbrace{M_E(t)}^{\text{maturation}} \tag{1a}$$

$$\frac{dH_{L1}(t)}{dt} = R_{L1}(t) - (\overbrace{P_{f1}(P_A(t))}^{\text{parasitism}} + \overbrace{c_{11}H_{L1}(t)}^{\text{competition from sml larvae}} + \overbrace{c_{12}H_{L2}(t)}^{\text{competition from lge larvae}} + d_{L1})H_{L1}(t) - M_{L1}(t) \tag{1b}$$

$$\frac{dH_{L2}(t)}{dt} = R_{L2}(t) - (P_{f2}(P_A(t)) + c_{21}H_{L1}(t) + c_{22}H_{L2}(t) + d_{L2})H_{L2}(t) - M_{L2}(t) \tag{1c}$$

$$\frac{dH_A(t)}{dt} = R_A(t) - d_AH_A(t) - M_A(t), \tag{1d}$$

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

$$R_E(t) = rH_A(t), \quad M_E(t) = R_E(t-\tau_E)\sigma_E S_{HE}(t), \tag{2}$$

$$R_{L1}(t) = M_E(t), \quad M_{L1}(t) = R_{L1}(t-\tau_{L1})\sigma_{L1}S_{HL1}(t), \tag{3}$$

$$R_{L2}(t) = M_{L1}(t), \quad M_{L2}(t) = R_{L2}(t-\tau_{L2})\sigma_{L2}S_{HL2}(t), \tag{4}$$

$$R_A(t) = M_{L2}(t-\tau_P)\sigma_P + I_{HA}(t), \quad M_A(t) = R_A(t-\tau_A)\sigma_A, \tag{5}$$

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

$$S_{HE}(t) = \exp\left(\int_{t-\tau_E}^{t} -c_{E2}H_{L2}(x)dx\right) \tag{6}$$

$$S_{HL1}(t) = \exp\left(\int_{t-\tau_{L1}}^{t} -[c_{11}H_{L1}(x) + c_{12}H_{L2}(x) + P_{f1}(P_A(x))]dx\right) \tag{7}$$

$$S_{HL2}(t) = \exp\left(\int_{t-\tau_{L2}}^{t} -[c_{21}H_{L1}(x) + c_{22}H_{L2}(x) + P_{f2}(P_A(x))]dx\right). \tag{8}$$

The probabilities are evaluated by differentiating equation 6–8 with respect to t and solving the resulting delay-differential equations with those for the other variables. For example, from equation 6 we obtain

$$\frac{dS_{HE}(t)}{dt} = -c_{E2}(H_{L2}(t) - H_{L2}(t-\tau_E))S_{HE}(t). \tag{9}$$

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

Parasitoid equations

For the parasitoid population, we only need to solve explicitly for adults, $P_A(t)$:

$$\frac{dP_A(t)}{dt} = R_{PA}(t) - d_{PA}P_A(t) - M_{PA}(t), \tag{10}$$

where

$$R_{PA}(t) = P_{f1}(P_A(t-\tau_{PL}))H_{L1}(t-\tau_{PL})\sigma_{PL} + P_{f2}(P_A(t-\tau_{PL}))H_{L2}(t-\tau_{PL})\sigma_{PL} + I_{PA}(t) \tag{11}$$

$$M_{PA}(t) = R_{PA}(t-\tau_{PA})\sigma_{PA}, \tag{12}$$

if there is no developmental lag in parasitism of the early larval class, otherwise

$$R_{PA}(t) = R_{L1}(t-\tau_{L1}-\tau_{PL})(1-S_{HL1P}(t-\tau_{PL}))\sigma_{PL} + P_{f2}(P_A(t-\tau_{PL}))H_{L2}(t-\tau_{PL})\sigma_{PL} + I_{PA}(t), \tag{13}$$

with $\sigma_i = \exp(-d_i\tau_i)$ and the probability of small host larvae escaping parasitism, $S_{HL1P}(t)$, given by

$$S_{HL1P}(t) = \exp\left(\int_{t-\tau_{L1}}^{t} -P_{f1}(P_A(x))dx\right). \tag{14}$$

The parasitism function is defined as

$$P_{fi}(P_A) = k\ln\left(1+\frac{a_iP_A}{k}\right), \qquad i = 1, 2. \tag{15}$$

The inoculation of parasitoid adults, $I_{PA}(t)$ is defined as a constant i_{PA} over the time interval ($i_{PA}start$, $i_{PA}start + 1$] and zero elsewhere.

MODEL PARAMETERS

Here we list the parameters used in the simulations of the three main models (Tables A1 and A2). Where indicated, the parameters have been derived from experiments. We only list parameter values for the simpler models – uniform larval competition model (ULCM) and asymmetric larval competition model (ALCM) – when they differ from those for the egg cannibalism model (ECM). Otherwise all parameters are the same as the egg cannibalism model.

Table A1. Host parameters. Sources of estimates: [a]data from Sait, Begon & Thompson (1994a); [b]data from Reed (1998)

Host parameter	Description	ECM value	ALCM value	ULCM value
τ_E	Duration (in days) of egg stage	4·3[a]		
τ_{L1}	Duration of early larval stage	10[b]		
τ_{L2}	Duration of late larval stage	15[b]		
τ_P	Duration of pupal stage	7[b]		
τ_A	Duration of adult stage	5·5[a]		
r	Daily adult fecundity (female eggs)	21[a]		Varied
d_E	Background mortality of eggs	0·017[a]		
d_{L1}	Background mortality of early larvae	0[a]		
d_{L2}	Background mortality of late larvae	0[a]		
d_p	Background mortality of pupae	0[a]		
d_A	Background mortality of adults	0·1		
c_{E2}	Egg cannibalism by late larvae	Varied	0	0
c_{11}	Mortality of H_{L1} from competition by H_{L1}	0·00004		Varied
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	Varied	1
ψ	Ratio of competitive sensitivities	10	Varied	1
i_{HA}	Inoculation of adults	10		

Table A2. Parasitoid parameters. Sources of estimates: c, data from Harvey *et al.* (1994)

Parasitoid parameter	Description	ECM value	ALCM value	ULCM value
τ_{PL}	Duration (in days) of juvenile stage	20[c]		
τ_{PA}	Duration of adult stage	2[c]		
d_{PL}	Background mortality of juveniles	0·1		
d_{PA}	Background mortality of adults	0·1		
a_1	Attack rate on early larvae	0·005		0·01
a_2	Attack rate on late larvae	a_1 (when fixed)		a_1
k	Interference parameter	1		
i_{PA}	Inoculation of adults	2		
$i_{PA}start$	Timing of inoculation of adults	200		

NUMERICAL SIMULATIONS AND SPECTRAL ANALYSIS

The delay-differential equations are solved using a Fortran subroutine by Hairer and Wanner based on a fourth order explicit Runge–Kutta method with quintic Hermite interpolation (Hairer, Norsett & Wanner 1993).

Spectral analysis is performed by taking the fast Fourier transform of 10 overlapping segments, each of length 512 days, from the last 2816 ($(10 + 1) \times 256$) days of the time series. The resulting periodograms (power = mean square amplitude) are then averaged together to reduce the spectral variance per data point. The period corresponding to the maximum peak of the power spectrum is calculated for use in the bifurcation diagrams. The model output used is a time series of length 5000 days, so the first 2000 days are always discarded to avoid transient dynamics.